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- 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.
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THE MIND-BODY CONNECTION





"A COMPREHENSIVE INTRODUCTION TO INTEGRATIVE NEUROLOGY"

DR. TREVOR BERRY DC, DACNB



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ALZHEIMER'S RATES



Alzheimer's is not just memory loss. Alzheimer's kills.

 1 in 3 seniors dies with Alzheimer's or another dementia. It kills more than breast cancer and prostate cancer combined. There are currently three times the number of new Alzheimer's disease cases compared to HIV/AIDS — that is **one new case** every four seconds.

The world is now facing an epidemic of dementia. By 2050, it is estimated that China will have a population of demented people equal to the current population of California (37 million). Almost 5.5 million Americans suffer from the ravages of Alzheimer's

disease. Among the top ten causes of death in the U.S., the dementias are the only ones that do not have a cure.

The greatest risk factor for Alzheimer's is age. At age 65, 1 in 20 people develop the disease. By age 75, 1 in 10 have Alzheimer's.

At age 85, 1 in every 2 people becomes demented.

PARKINSON'S DISEASE



Parkinson's disease is 50% more common than previously reported, researchers say

For the study, researchers examined Parkinson's incidence from 2012 healthcare data in North America. The data included cohorts of 6.7 million person-years of adults aged 45 and older and 9.3 million person-years of adults aged 65 and older.

The researchers found that 60,0000–95,000 people were diagnosed with PD in North America in 2012, significantly more than previous estimates of <u>40,000–60,000</u>

Trusted Source

Advanced age was strongly linked to an increased rate of PD, and **males were consistently more likely to develop the condition than females.**

Open Access

Published: 15 December 2022

Incidence of Parkinson disease in North America

 A. W. Willis, E. Roberts, J. C. Beck, B. Fiske, W. Ross, R. Savica, S. K. Van Den Eeden, C. M. Tanner & C. Marras on behalf of the Parkinson's Foundation P4 Group npj Parkinson's Disease

volume 8, Article number: 170 (2022) Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

APRIL 1, 2022



ERCHONIA

Half of older adults now die with a dementia diagnosis, up sharply from two decades ago

The study, published in *JAMA Health Forum* by a University of Michigan team, uses data from 3.5 million people over the age of 67 who died between 2004 and 2017.

...claims with dementia diagnosis rose to more than 47% in 2017.

Trends in US Medicare Decedents' Diagnosis of Dementia From 2004 to 2017

7

Matthew A. Davis, PhD, MPH^{1,2,3}; Chiang-Hua Chang, PhD^{1,4}; Sharon Simonton, PhD, MPH^{1,4}; <u>et al</u> Julie P. W. Bynum, MD, MPH^{3,4} Author Affiliations <u>Article Information</u> JAMA Health Forum. 2022;3(4):e220346. doi:10.1001/jamahealthforum.2022.0346



The Global Burden of Disease Study represents the first effort of its kind to forecast the future rates of Alzheimer's in 204 countries and territories, and the projections are dire. It estimates the number of adults over the age of 40 living with dementia worldwide will increase from 57 million to 153 million over the next three decades.

THE LANCET

ARTICLES<u>| VOLUME 7, ISSUE 2</u>, E105-E125, FEBRUARY 01, 2022 Estimation of the global prevalence of dementia in 2019 and forecasted prevalence in 2050: an analysis for the Global Burden of Disease Study 2019

GBD 2019 Dementia Forecasting Collaborators +

Open Access Published: January 06, 2022DOI:https://doi.org/10.1016/S2468-2667(21)00249-8 Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

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



In 2018, an estimated 9.1 million people aged over 60 are living with dementia in EU member states, up from 5.9 million in 2000. If the age-specific prevalence of dementia remains the same, ageing populations mean that this number will continue to grow substantially in the future.

The overall number of people living with dementia in EU countries is expected to rise by about 60% over the next two decades to reach 14.3 million in 2040, with the oldest people (those aged over 90) accounting for a growing share.

NEURODEGENERATIVE DISEASE



NEURODEGENERATIVE DISEASE COSTS EXCEED \$655 BILLION A YEAR IN MEDICAL EXPENSES AND ECONOMIC LOSSES (USA, 2020)

May 27, 2021 (WASHINGTON, D.C.) The *Partnership to Fight Chronic Disease (PFCD)* today released a *literary review and infographic data* on Alzheimer's disease and related dementias (ADRD), Parkinson's disease and motor neuron diseases, specifically amyotrophic lateral sclerosis (ALS) and spinal muscular atrophy (SMA). The data review examines specific disease burden, economic burden, individual costs, and caregiver burden for these neurodegenerative diseases. The annual cost for these conditions in the U.S. was \$655 billion in 2020, including direct medical and non-medical costs and indirect costs from lost productivity and uncompensated caregiving hours.

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DEMENTIA RATES IN AUSTRALIA



New report reveals the Australian burden of disease for brain disorders is almost twice the global figure

Neurological, mental health and substance use disorders currently account for over 20% of the burden of disease in Australia. In the future these brain disorders will have a greater cost to the Australian economy than heart disease, cancer, and respiratory disease combined. Mindgardens Neuroscience Network commissioned KPMG to review this data and our White Paper shows the burden of disease cost in 2017 was in excess of \$74 billion. We need to build new models of care to reduce this burden of disease for all Australians.

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14 March 2019

WHAT IS YOUR PURPOSE?

"THE GREATEST TRAGEDY IS NOT DEATH BUT LIFE WITHOUT PURPOSE"

CURRENT ALLOPATHIC MODEL FOR ALZHEIMER'S



FDA Grants Accelerated Approval for Alzheimer's Disease Treatment

January 06, 2023 Today, the U.S. Food and Drug Administration approved <u>Leqembi</u> (lecanemab-irmb) via the <u>Accelerated Approval pathway</u> for the treatment of Alzheimer's disease.

These results support the accelerated approval of Leqembi, which is based on the observed reduction of amyloid beta plaque, a marker of Alzheimer's disease.

Leqembi was approved using the <u>Accelerated Approval pathway</u>, under which the FDA may approve drugs for serious conditions where there is an unmet medical need and a drug is shown to have an effect on a surrogate endpoint that is reasonably likely to predict a clinical benefit to patients. The results of a Phase 3 randomized, controlled clinical trial to confirm the drug's clinical benefit have recently been reported and the agency anticipates receiving the data soon.

CURRENT ALLOPATHIC MODEL FOR ALZHEIMER'S: THE AMYLOID ONLY HYPOTHESIS



Seminal Alzheimer's study claiming memory-robbing disease was caused by build-up of protein in brain may have been MANIPULATED, damning investigation claims

- Images used to prove a protein is behind Alzheimer's may have been tampered
- Forensic imaging experts say they appear doctored to 'better fit a hypothesis'
- Seminal paper was used as starting point for billions of pounds of research

Experts fear the allegedly falsified results have misled research over the last 16 years, potentially wasting billions of pounds of funding.

A six-month investigation by Science, considered one of the world's most respected research journals, uncovered 'shockingly blatant' tampering of results in the seminal 2006 University of Minnesota study.

Published in rival journal Nature, the study became one of the most cited articles on Alzheimer's ever published.

Around £1.3billion (\$1.6billion) of funding for studies mentioning amyloids was spent by the US Government over the last year alone. It made up half of the country's total Alzheimer's research funding.

But images from the study, which involved injecting mice with the protein, appear to be doctored to 'better fit a hypothesis', according to Dre Elisabeth Bik, a forensic image consultant who was asked to review the date rry, DC, DACNB is prohibited.

without permission of Trevor Berry, DC, DACNB is prohib ALZHEIMER'S DRUGS



Manufacturers need to be more open about a dangerous Alzheimer's drug side effect

By Madhav ThambisettyNov. 28, 2023

brain swelling and bleeding that are together called amyloid-related imaging abnormalities, or ARIA, which have been found in all three drugs designated as "breakthrough treatments" by the Food and Drug Administration: lecanemab, aducanumab, and donanemab. I am especially worried about drugmakers' lack of transparency in fully reporting all details of clinical outcomes related to ARIA.

the FDA has determined that these drugs should carry a black box warning, the highest level of precaution reserved for medicines associated with major risks of harm to patients.

Each of the three new Alzheimer's drugs have also been linked with patient deaths in clinical trials

the maker of lecanemab has refused to share any clinical trial data with other researchers

Rev Neurol (Paris)

2022 Dec;178(10):1011-1030. doi: 10.1016/j.neurol.2022.06.012. Epub 2022 Sep 29. High-clearance anti-amyloid immunotherapies in Alzheimer's disease. Part 1: Meta-analysis and review of efficacy

and safety data, and medico-economical aspects N Villain 1, V Planche 2, R Levy 3

CURRENT ALLOPATHIC MODEL FOR ALZHEIMER'S



OTHER CONSIDERATIONS FOR LECANEMAB

-ONLY CLEARED FOR EARLY ONSET DEMENTIA, AB PLAQUE REDUCTION

- -MUST DO IV DOSE EVERY 2 WEEKS
- -13% OF THE STUDY PARTICIPANTS HAD BRAIN SWELLING
- -17% OF PARTICIPANTS HAD LIFE THREATENING BRAIN BLEEDS
- -MULTIPLE DEATHS WERE REPORTED DURING THE STUDY ESPECIALLY THOSE ON BLOOD THINNERS. BLOOD THINNERS MAY BE A CONTRAINDICATION FOR IT'S USE. OTHER COMMON SIDE EFFECTS INCLUDED NAUSEA, CHILLS, BLOOD PRESSURE FLUCTUATIONS AND FEVERS
- COST OF THE DRUG IS \$26,500/YEAR. HOW MUCH OUT OF POCKET DEPENDS ON YOUR INSURANCE COVERAGE. WILL INSURANCES EVEN COVER IT (THEY ARE WAITING TO SEE IT'S ACTUAL CLINICAL EFFECTS)
- SAME COMPANY (BIOGEN ALONG WITH JAPANESE COUNTERPART EISAI) AND SIMILAR DRUGplasson Added HEisterburioneof this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CASSAVA SIMUFILAM



Scientist behind Alzheimer's drug in late-stage trials is indicted on charges of research fraud

The charges are related to alleged fabrication of research images and data that Hoau-Yan Wang may have used to secure federal grants from the National Institutes of Health.

The Summary

- A neuroscientist whose work helped pave the way for an Alzheimer's drug candidate was indicted on charges of fraud.
- The charges are related to the alleged fabrication of research images and data that the scientist may have used to secure grants.
- The manipulation of research images is a <u>growing concern</u> in the scientific community. (REVIEW THE ORIGINS OF THE AMYLOID HYPOTHESIS)

The indictment charges Wang with one count of fraud against the United States, two counts of wire fraud and one count of false statements. It accuses *Wang of manipulating or adding to images of Western blots, a laboratory method that researchers use to identify proteins, in order to bolster evidence and help secure grants.*

The indictment also suggests that Wang may have lied to scientific journals to substantiate his research, which contributed to the early development of simufilam.

The drug is currently in a late-stage clinical trial, and some 735 patients had participated as of May 2024, according to a news release from Cassava last month.

ALZHEIMER'S MEDICATIONS



HEALTH AND SCIENCE

Eli Lilly Alzheimer's treatment solanezumab failed to slow disease progression

PUBLISHED WED, MAR 8 202311:34 PM ESTUPDATED THU, MAR 9 20231:50 PM EST

KEY POINTS

- Solanezumab's failure is a blow to efforts to treat Alzheimer's in people who are in the very early stage of the disease and have not yet shown clinical symptoms.
- Solanezumab did not clear or halt accumulation of brain plaque and did not slow cognitive decline in the participants who received the treatment.



Supreme Court overturns opioid settlement with Purdue Pharma that shielded Sacklers

After the Supreme Court struck down a controversial bankruptcy plan from Purdue Pharma, the maker of OxyContin, those who sued the drug company were left uncertain about when promised funds would be available to combat addiction and other damage from the ongoing drug epidemic. The ruling upended a carefully-crafted settlement worth roughly \$8 billion, and involving the Sackler family, which owns Purdue, and all the individuals, states and local governments that had sued over harms from the opioid epidemic.

Purdue Pharma was facing thousands of lawsuits for falsely marketing OxyContin as nonaddictive and fueling the opioid crisis. The company filed for bankruptcy in 2019.

Before that, the Sackler family, which owns Purdue, had moved about \$11 billion of profits into personal accounts. In his ruling, Gorsuch said members of the family had created a "milking program" designed to shelter opioid profits from their company's bankruptcy.

During the bankruptcy negotiations, the family offered to pay \$6 billion in exchange for immunity from future lawsuits.

A federal bankruptcy judge approved that deal in 2021, but Gorsuch ruled that it was an overreach.

OVER 500 EXPERIMENTAL DRUGS INTENDED TO TREAT OR CURE ALZHEIMER'S DISEASE HAVE FAILED IN THE PAST 30 YEARS MAINLY BECAUSE OF THE SHORTCOMINGS OF THE "AMYLOID HYPOTHESIS". THE FDA HAD ONLY SIGNED OFF ON 5 DRUGS TO MANAGE ALZHEIMER'S SYMPTOMS SINCE 2003.

HOWEVER, COMMONLY USED CLASSES OF DRUGS MAY INDUCE DEMENTIA LIKE SIDE EFFECTS AND OTHER NEUROLOGICAL: DISORDERSI of in part



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

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)."

2. <u>ANTICHOLINERGICS</u>. 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.

PHARMACEUTICALS AND DEMENTIA



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, the German dataset, and with of distribution of distributi



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*.

MEDICATION INDUCED DEMENTIA



Popular medication could increase

dementia risk by staggering 79 per cent

23

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 202 without permission of Trevor Berry, Do, DAC NB is prohibited.



These Common Insomnia and Anxiety Drugs May Increase Your Dementia Risk, Study Shows

Mario M. Dorostkar, neuropathologist, and co-author of the *Nature Neuroscience* study tells *Woman's World*, they

affect the structure of dendritic spines...the drug interferes with the dissolution and reformation (of neuronal dendritic activity). "A disturbance in this process leads to impaired memory and cognition," he explains.

• Published: 28 February 2022

Long-term **diazepam (VALIUM)** treatment enhances microglial spine engulfment and impairs cognitive performance via the mitochondrial 18 kDa translocator protein (TSPO)

 Yuan Shi, Mochen Cui, Katharina Ochs, Matthias Brendel, Felix L. Strübing, Nils Briel, Florian Eckenweber, Chengyu Zou, Richard B. Banati, Guo-Jun Liu, Ryan J. Middleton, Rainer Rupprecht, Uwe Rudolph, Hanns Ulrich Zeilhofer, Gerhard Rammes, Jochen Herms & Mario M. Dorostkar Nature Neuroscience
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INHALERS AND BRAIN DEGENERATION



Common steroids used for asthma, allergies linked to brain decline, study finds

By Sandee LaMotte, CNN Updated 6:31 PM ET, Tue August 30, 2022

volume



Conclusions Both systemic and inhaled glucocorticoid use are associated with decreased white matter integrity and limited changes in GMV. This association may contribute to the neuropsychiatric side effects of glucocorticoid medication, especially with chronic use.

White matter is the tissue that forms connections between brain cells and the rest of the nervous system. *Having less white matter can slow the brain's ability to process information, pay attention and remember. Lower levels of white matter have also been connected to psychiatric issues such as depression, anxiety and irritability.* The study followed 500,000 people from 2006-2010

Association between use of systemic and inhaled glucocorticoids and changes in brain volume and white matter

microstructure: a cross-sectional study using data from the UK Biobank Duplication, citation or distribution of this material in whole or in part http://orcid.org/0000-0002-000 with the log material in whole or in part
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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. 27

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

EEG EFFECTS OF COMMON MEDICATIONS/SUBSTANCES



	DELTA	ТНЕТА	ALPHA	BETA	ALPHA PF
BENZODIAZEPINES	+	+	-	+	
ALCOHOL			+	+	
GABAPENTIN	+	+			-
RISPERDAL	+	+	+	+	
ABILIFY			-		-
ADDERALL/RITALIN		-	+	+	
AMBIEN		-	-	+	
MELATONIN	-	-	+		
COCAINE	_	-	+	+	
CAFFEINE			-		
ZOLOFT	+	+		+	
MARIJUANA		-	+		
FENTANYL	+	+			
CORTISONE			+		
KETAMINE	-	+			



ARE THERE VIABLE SOLUTIONS?

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Boosting neuron formation can cure Alzheimer's disease

[Aug 21, 2022:

The study, to be published in the *Journal of Experimental Medicine* (*JEM*), shows that new neurons can incorporate into the neural circuits that store memories and restore their normal function, suggesting that boosting neuron production could be a viable strategy to treat AD patients.

"Our study is the first to show that impairments in hippocampal neurogenesis play a role in the memory deficits associated with AD by decreasing the availability of immature neurons for memory formation," Lazarov says. "Taken together, our results suggest that augmenting neurogenesis may be of therapeutic value in AD patients."

Skip Nav Destination Article IAugust 19 2022

Augmenting neurogenesis rescues memory impairments in Alzheimer's disease by restoring the memory-storing neurons

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NEUROGENESIS AND ALZHEIMER'S

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

PMCID: PMC6990771 PMID: <u>31719242</u>

RCHONIA

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

Mehmet Ilyas Cosacak,1 Prabesh Bhattarai,1 and Caghan Kizil, PhD1,2* 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.

However, this provocative idea requires further evidence and tools for investigation. Recently, single cell sequencing appeared as a revolutionary tool to understand cellular programs in unprecedented resolution and it will undoubtedly facilitate comprehensive investigation of different cell types in Alzheimer's disease. In this mini-review, we will touch upon recent studies that use single sells equencing for investigating cellular mesons and some consideration pertaining to the utilization of sieu call regeneration of GAADABeimer's disease research.

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)<u>1</u>. Moreover, this structure hosts one of the most unique phenomena of the adult mammalian brain, namely, the addition of new neurons throughout life<u>2</u>.

This process, called adult hippocampal neurogenesis (AHN), confers an unparalleled degree of plasticity to the entire hippocampal circuitry_{3.4}. 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.

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



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 SXN-1 and GAP43 genes. It was

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

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!

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 THESE ARE JUST A FEW OF THE RESEARCHED MECHANISMS OF ACTION OF LOW LEVEL LASER IN OVER 16,000 PUBLISHED PAPERS! Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

"If a drug company found a drug that could do all of that, it would immediately be hailed as a miracle drug—the most dramatically beneficial drug ever created.

If one supplement could do all of that, every American would be taking it every day. The only reason why healthcare providers are not performing Laser therapy on EVERY patient is the lack of knowledge of the far reaching effects of this technology. The case could be made that Low Level Laser is the single most important therapy a practitioner can add to their services."

-Dr. Trevor Berry DC, DACNB



LOW LEVEL LASER THERAPY

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THE NEUROLOGY JEDI LIGHTSABER FOR ANY UNIT OF THE STORE FOR ANY UNIT OF THE STORE OF THE PRACE THE TONER!

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WARNING





LED vs TRUE LASER



FOR A DEEPER DIVE INTO LED VS TRUE LOW LEVEL LASER THERAPY PLEASE CHECK OUT:

LASERS VS RED LIGHT OR LEDs: IS THERE ANY DIFFERENCE? LASER HEALTH REVOLUTIONS WITH DR. GAIR

SPOTIFY, DECEMBER 7TH 2023

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E You

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

ShatGPT

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 or different applications. Safety considerations are crucial as well, as excessive doses or inappropriate application methods could have adverse effects.

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BIPHASIC DOSE AND 10 J/CM2



Therapeutic technique and dosage of LLLT

The minimum therapeutic dose for a bio-stimulatory effect for red and infrared

laser is 0.01 J/cm2 while for ultraviolet, blue, green laser it is 0.001 J/cm2.

LLLT has a noticeable biphasic dose response. The effective stimulation dose is 1 J/cm2 on the target tissue. Doses greater than 10 J/cm2 produces inhibitory effects. The

inhibitory effects are used in conditions requiring inhibition and suppression $[\underline{2}]$.

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>

PMCID: PMC7428000 PMID: <u>32844112</u>

Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19

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https://www.kmlabs.com/en/ wavelength-to-photon-energycalculator

STAY BELOW 10 J/CM2

STAY IN BETWEEN 400 AND 850 NM WAVELENGTHS BELOW 400 CAN CAUSE IONIZING RADIATION AND MUTAGENIC CONCERNS (IE. UV LIGHT) ABOVE 850 CAN ALTER VITAMIN D RECEPTOR SHAPE, DEGRADE THE COLLAGEN MATRIX, AND PRODUCE GLUTAMATE EXCITOXICITY DO NOT MISTAKE HIGHER ENERGY FOR HIGHER DOSE SHORTHER WAVELENGTHS GET BETTER RESULTS NOT BECAUSE THEY ARE HIGHER POWER BUT RATHER, HIGHER ENERGY

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SUMMARY POINTS OF LIGHT THERAPY





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

2.ABOVE 10 EV IS IONIZING (MAX OF ERCHONIA VIOLET IS 3.04)

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

4.FDA CLEARANCES (ESPECIALLY FOR SAFETY)

5.PHOTOCHEMICAL VS. PHOTOTHERMAL

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FX SCANNING LASERS





WHY LOW LEVEL LASER UNDER 10 J/CM2 TO THE HEAD IS CRITICAL...





Like ל'ח

Low Level Laser Therapy (LLLT) ••• and Photobiomodulation (PBM) Discussions

Hedy Gervais · 22m ·

My daughter had mls laser therapy for her TMJ. The doctor had it on the highest setting and kept it on the joints near her eyes for 2 plus minutes on each side. Within a half hour, she was in terrible pain with tinnitus and eye pain. Going on day 2 and she is still in a lot of pain. Will this simmer down or has permanent damage been done? Thank you for any feedback.

Comment

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2 comments

Send

х

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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:



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.

- 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 Lin relief and injury treatment.

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VE 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.

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Action or distribution of this material in whole or in part MILL ONLY USE ERCHONIA PRODUCTS TO TREAT AND PROTECT THE BRAINS OF MY LOVED ONES





EARLY ONSET DEMENTIA



Major Study Identifies 15 Factors Linked to Early Dementia Risk

The research team analyzed data collected on 356,052 people aged under 65 in the UK. Low socioeconomic status, social isolation, hearing impairment, stroke, diabetes, heart disease, and depression were all associated with a higher risk of YOD.

Vitamin D deficiency and high levels of the C-reactive protein (produced by the liver in response to inflammation) also meant a higher risk, as did having two of the ApoE4 ε4 gene variants (a genetic scenario already linked to Alzheimer's disease).

The researchers describe the relationship between alcohol and YOD as "complex". While alcohol abuse and abstaining from alcohol led to an increased risk, moderate drinking correlated with a reduced risk.

Higher levels of formal education and lower physical frailty (measured through higher handgrip strength) were also associated with a lower YOD risk. "In addition to physical factors, mental health also plays an important role, including avoiding chronic stress, loneliness and depression."

BE RESPONSIBLE WITH ETOH AND HEALTH. EX. ALL CANCERS ARE AT GREATER RISK

Risk Factors for Young-Onset Dementia in the UK Biobank

Stevie Hendriks, PhD¹; Janice M. Ranson, PhD²; Kirsten Peetoom, PhD¹; <u>et al</u> Ilianna Lourida, PhD²; Xin You Tai, PhD^{2,4}; Marjolein de Vugt, PhD¹; David J. Llewellyn, PhD^{2,4}; Sebastian Köhler, PhD¹ Author Affiliations

JAMA Neurol. Published online December. 260 2023 gaditation and instruction of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

AGING



Wonder enzyme may hold the key to longer, healthier lives

In a surprise finding, researchers at the University of Virginia (UVA) found that the role of alcohol dehydrogenase (ADH-1) in detoxifying the body of glycerol and glyceraldehyde – the harmful by-products of fat that build up over time – may provide the best approach to not just extending life but aging more healthily.

KEY CONCEPTS:

-THIS MECHANISM WAS A COMMON DENOMINATOR IN WELL DESCRIBED ANTI-AGING MECHANISMS (AUTOPHAGY, MITOPHAGY, FASTING/CALORIC RESTRICTION, EXERCISE/HIIT, RAPAMYCIN ETC)

- PEARS AND ORANGES ARE POWERFUL ADH-1 STIMULATORS
- CONSIDER MONOGLYCERIDES FOR OMEGA-3 SUPPORT
- STANDARD AMERICAN DIET AND EXCESS ALCOHOL CONSUMPTION

LIVER PROTOCOL WITH ERCHONIA LOW LEVEL LASER THERAPY

Increased alcohol dehydrogenase 1 activity promotes longevity

Abbas Ghaddar 5 Published:February 17, 2023. CURRENT BIOLOGY

ALZHEIMER'S ETIOLOGIES



Breakthrough Alzheimer's Discovery Reveals Five Distinct Variants

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The researchers were able to identify five distinct biological subtypes of the disease, distinguished by variations such as hyperplasticity, immune activation, RNA dysregulation, choroid plexus dysfunction, and blood-bain barrier impairment. Each variation was characterized by specific alterations in clusters of proteins related to inflammation, nerve cell growth, and other biological processes.

Hyperplasticity seems to involve an overactive cellular growth response, leading to amyloid and tau protein buildup.

In innate immune activation, the immune system goes into overdrive, excessively attacking healthy brain tissue.

RNA dysregulation involves changes in the transport of proteins along axons that allow nerve cells to function correctly.

Choroid plexus dysfunction impacts the ventricular system of the brain, which contributes to cerebrospinal fluid production and transfer of nutrients to the brain.

Blood-brain barrier impairment weakens the barrier protecting the brain, allowing damaging molecules to infiltrate. Unlike hyperplasticity, this subtype features slow nerve cell growth and low amyloid production.

- Open access
- Published: 09 January 2024

YOU MUST ADDRESS MULTIPLE UNDERLYING CAUSES NOT JUST AMYLOID BURDEN!

Cerebrospinal fluid proteomics in patients with Alzheimer's disease reveals five molecular

subtypes with distinct genetic risk profiles Duplication, citation or distribution and this material in whole or in part

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rial in AGING, September 2014, Vol 6 N 9

NEW YORK TIMES BESTSELLER "A MONUMENTAL WORK."

Review 5

RCHONIA

Reversal of cognitive decline: A novel therapeutic program

Dale E. Bredesen^{1, 2}

¹ Mary S. Easton Center for Alzheimer's Disease Research, Departn Angeles, CA 90095;

² Buck Institute for Research on Aging, Novato, CA 94945.

Key words: Alzheimer's, dementia, mild cognitive impairment, neurobehc neurodegeneration, systems biology Received: 9/15/14; Accepted: 9/26/14; Published: 9/27/14 Correspondence to: Dale E. Bredesen, MD; E-mail: <u>dbredesen@mednet</u>

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Abstract: This report describes a novel, comprehensive, and persounderlying pathogenesis of Alzheimer's disease, and which involves enhancement for neurodegeneration (MEND). The first 10 patients w memory loss associated with Alzheimer's disease (AD), amnestic mild c impairment (SCI). Nine of the 10 displayed subjective or objective imp with the one failure being a patient with very late stage AD. Six of th struggling with their jobs at the time of presentation, and all were improved performance. Improvements have been sustained, and at one-half years from initial treatment, with sustained and marked imprextensive trial of this therapeutic program is warranted. The resul cognitive decline may be driven in large part by metabolic processes. in AD to date, the results raise the possibility that such a therapeutic that would fail as monotherapeutics may succeed as key components on

The First Program to Prevent and Reverse Cognitive Decline

DALE E. BREDESEN, MD

Reverse Alzheimer's With The Bredesen Protocol - Recode

BREDESEN HAS OVER 100 CASE STUDIES OF ALZHEIMER'S REVERSAL

IT HAS BEEN SHOWN IN OVER 250 CASES FOR REVERSAL OF COGNITIVE DECLINE IN NEUROGENERATION

THIS INCLUDES IMPROVEMENT IN PATIENTS WITH AS MUCH AS 85% HIPPOCAMPUS ATROPHY

This protocol used to be called the MEND protocol, but now is called ReCODE.

The overall goal of this protocol is to fix the following (which in turn reverses cognitive decline and helps with Alzheimer's and Dementia):

- Insulin resistance
- Inflammation/infections
- Hormone, nutrient, and trophic factor optimization
- Toxins (chemical, biological, and physical)
- Restoration and protection of lost (or dysfunctional) synapses

Protocols like this may never reach global recognition or even FDA consideration for further evaluation. The problems with most FDA-based studies is that they only look at one aspect of the disease - X causes Y. Unfortunately, AD is complex and there are many root causes to it.

The ReCODE protocol looks at 45 factors (metabolic markers) that have to be simultaneously optimized.

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AMYLOID: FRIEND OR FOE?



This positive feedback loop creates a clastic (destructive) effect on the synapses, instead of a blastic (protective) response.

Amyloid Precursor Protein

Amyloid precursor protein (APP) is **naturally occurring** in the brain and depending on *how it is cut* (either by netrin-1 or other molecules) it can either turn into:

- 1. Something healthy for the brain (such as sAPP α and α CTF) OR
- 2. Something toxic such as **amyloid-beta** (as well as Jcasp and C31). <u>R</u>

If APP is cut to produce amyloid-beta, then amyloid-beta can continue to cut APP into more amyloid-beta plaques.

This creates a positive feedback loop, thus creating more and more amyloidbeta plaques in the brain, instead of creating healthy molecules for the brain.



RECODE



Amyloid-Beta Plaque

The dogma behind amyloid plaque (the sticky plaque that builds up in the brain of Alzheimer's patients) as the main evil in Alzheimer's Disease (AD) is actually incorrect.

In fact, it may be protective:

- Acts as an anti-microbial
- Binds to toxins (like heavy metals)
- Protects against inflammation

If you have been shot, would you just patch up the wound and not remove the bullet?

No, you would treat the underlying problems (remove the bullet) and replace the blood loss.

The Bredesen Protocol first targets the underlying problem.



3 Types Of Alzheimer's Disease

There are 3 types of Alzheimer's disease described in the ReCODE protocol. You can develop one of these or a combination:

1. Inflammation

Anything that causes inflammation to the brain (low chronic inflammation can do this as well) can contribute to Alzheimer's Disease (AD):

- AGEs
- <u>ApoE4</u> (and <u>ApoE3</u>)
- Diet High in Lectins
- Imbalances in fatty acids (omegas)
- Infections
- Insulin Resistance
- Leaky Gut or Leaky Blood Brain Barrier
- Neuroinflammation
- <u>Toxins</u> (incl metals)



RECODE



3 Types Of Alzheimer's Disease

There are 3 types of Alzheimer's disease described in the ReCODE protocol. You can develop one of these or a combination:

1.5 Glycotoxic

Glycotoxicity comes form an imbalance of glucose/insulin usage in the brain.

The pancreas produces Insulin-Degrading Enzyme (IDE, the enzyme that breaks down insulin) to break down amyloid beta.

If IDE is used up by a diet too high in sugar (like someone with insulin resistance), then there is none left to break down amyloid beta.

This type of AD is called 1.5 because it is a combination of AD 1, inflammation and AD 2, trophic loss.

For example, having high amounts of glucose in the blood creates inflammation, and having improper usage of insulin, degrades insulin's ability to act as a neurotrophic (brain growth) promoter.







3 Types Of Alzheimer's Disease

There are 3 types of Alzheimer's disease described in the ReCODE protocol. You can develop one of these or a combination:

2. Metabolic/Trophins Loss

This type of AD is usually caused by imbalances in the endocrine system (hormones) and nutrient depletion, as well as neurotrophic loss (brain breaking down faster than it can regrow).

This includes:

- <u>ApoE4</u>
- Hormone Imbalances (Vitamin D, Sex and Neuro Steroids, Thyroid)
- Insulin Resistance
- Methylation Problems
- Mitochondrial Damage
- Neurotrophic Loss (atrophy in brain)
- Nutrient Depletion

RECODE

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3 Types Of Alzheimer's Disease

There are 3 types of Alzheimer's disease described in the ReCODE protocol. You can develop one of these or a combination:

3. Toxins

The toxin/infectious type of AD is more environmental and can be caused by:

- APOE4 (more common)
- <u>Biotoxins</u>
- Heavy Metals (including amalgams)
- Hormonal Imbalances
- HPA-Axis Imbalances
- Infections (such as mold, Lyme, <u>HSV</u>, active EBV, oral/nasal/gut <u>dysbiosis</u>)
- Low Zinc/high copper ratio
- Psychiatric disorders (correlation)
- Toxins (including haptens, pesticides, instance, instruction of the system of the

ALZHEIMER'S CAUSES



Dr. Bredesen articulates six different categories of the root causes of Alzheimer's. Most patients have more than one category of root cause and there are dozens of biomarkers that can be tested. These categories include:

- **Type 1 Inflammation**: there are elevated markers of chronic inflammation such as tumor necrosis factor alpha and cardiac reactive protein
- **Type 1.5 Glycotoxic**: there is evidence of insulin resistance or high glucose
- **Type 2** Atrophic: there is evidence of hormone deficiencies such as sex, adrenal, nutrient deficiencies such as folate, B12 or insufficient oxygen at night from sleep apnea.
- Type 3 Toxic: there is evidence of higher levels of chronic toxins including mold, indoor / outdoor air pollutants, chemicals, metals and/or chronic infections including herpesviruses and tick borne infections
- Type 4 Vascular: there is evidence of strokes, vascular disease or mini strokes at the capillary level caused by hypercoagulation
- **Type 5 Traumatic Brain Injury**: there is evidence of prior traumatic brain injury often from previously unappreciated major or mini-concussions which result in wounds that do not heal we

	RECODE	ERCHONIA Community Education
Testing/Biomarkers	Complement C4a	• Glucose (fasting)
Blood tests (Optimal Range):	○ < 2830 ng/ml	• 70-90 mg/dL
• Albumin/Globulin Ratio (A:G R	atio) • Copper	Glutathione
○ ≥ 1.8	• 90-110 mcg/dL	• 5-5.5 micromolar
• >4.5 (albumin)	Copper:Zinc Ratio (also look at	• HbA1C
	ceruloplasmin \leq 30)	○ ≤5.6%
• <u>Alpha-MSH</u>	0.8-1.2	
• 35–81 pg/ml		• HDL
	Cortisol (morning)	· >50
	• 10-18 mcg/dL	
• <u>Arsenic</u>		• HLA-DR/DQ
• <7 mcg/L	• DHEA	• negative
	 350-430 (women) mcg/dL 	
<u>Cadmium</u>		• Hs-CRP
• <2.5 mcg/L	• 400-500 (men) mcg/dL	o ≤0.9ng/dL
Calcium	• Estradiol (Estrogen)	• II-6
• 8.5-10.5 mg/dl	• 50-250 pg/ml	o ≤3pg/ml
Cholesterol	• Folate	• Insulin (fasting)
• 150	uplication, citation or distributio的 of this material in whole or in part without permission of The of Berly, DC, DACNB is prohibited.	o ≤4.5 microIU/ml

Testing/Biomarkers

- LDL-p
 - o 700-1000
- Lead
 - <2 mcg/dL
- Leptin
 - 0.5-13.8 ng/mL (male)
 - 1.1-27.5 ng/mL (female)
- <u>Mercury</u>
 - o <5 mcg/L</p>
- MMP9
 - 85-332 ng/mL
- Omega 6:3 Ratio
 - 0.5-3.0

Osmolality

• 280-300 mosmol

RECODE

- <60 U/l
- Pregnenolone
 50-100 ng/dL
- 1-20 ng/ml
- Potassium • 4.5-5.5 mEq/L
- RBC Magnesium • 5.2-6.5 mg/dL
- RBC Thiamine Pyrophosphate

 100-150 ng/ml
 - 63

- sdLDL • <20 mg/dL
- Selenium

 110-150 ng/ml
- **T3** o 3.2-4.2 pg/ml (free)
 - o <20 ng/dL(reverse)</p>
- **T4** • 1.3-1.8 ng/dL (free)
- TSH • <2 microIU/ml
- Testosterone • 500-1000 ng/dL (total)
 - 6.5-15 ng/dL (free)

RECODE



Testing/Biomarkers

- TGF-β1
 - < 2380 pg/ml
- TNF-alpha
 - o ≤6pg/ml
- Triglycerides
 - ° <150
- Vasopressin
 - 1.0-13.3 pg/ml
- VEGF
 - 31-86 pg/mL
- VIP
 - 23-63 pg/mL
- Vitamin B6
 - 60-100 mcg/L

- Vitamin B12 (MMA test can complement, but isn't a replacement) o 500-1500 pg/ml
- Vitamin C

 1.3-2.5 mg/dL
- Vitamin D
 50-80 ng/ml
- Vitamin E (as Alpha-Tocopherol)

 12–20 mcg/ml
- Zinc

 90-110 mcg/mL

Suggested testing for leaky gut, leaky brain, and food sensitivities:

VIBRANT

WHEAT ZOOMER OR INTESTINAL PERMEABILITY PANEL

NEURAL ZOOMER PLUS

FOOD SENSITIVITIES 1 & 2

- <u>Cyrex Array 2</u> leaky gut
- Cyrex Array 3/4 food sensitivities and gluten intolerance
- <u>Cyrex Array 5</u> autoantibodies

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Testing/Biomarkers

Infections can travel to the brain (via a leaky brain) through the nose, vagus nerve, or eye such as:

- Aspergillus
- CIRS
- Gingivitis
- Lyme (Borrelia)
- HSV
- Syphilis (neurosyphhilus)

This can also cause meningitis.

VIBRANT

NEURAL ZOOMER PLUS

MYCOTOXIN OR LYME PANELS

CIRS PANEL

For **mitochondrial** function testing, use an <u>organic acids test</u> or look at <u>8-oxo-dg</u>. VIBRANT ORGANIC ACIDS TEST

A urine culture should be free of mycotoxins. VIBRANT MYCOTOXINS TEST

All microbiomes shouldn't have dysbiosis or infections. VIBRANT GUT ZOOMER 3.0

Imaging can be done with:

- **PET** (FDG-PET, Amyloid PET, or Tau PET)
- MRI with volumetrics (Neuroreader or NeuroQuant).

Body mass index (BMI) should be 18–25; waistline < 35 inches (women) or < 40 inches (men).

Here are some cognitive tests:

- MMSE (Mini-Mental State Examination)
- MoCA (Montreal Cognitive Assessment) A normal MoCA score is 26 to 30
- SAGE (Self-Administered Gerocognitive Examination)
 65
- RECODE



Genetics

Watch inflammation source: The major epigenetic tripwire!

ApoE4 (epsilon 4) is the most common genetic variable for predicting Alzheimer's.

What does ApoE4 do?

- · Reduces the clearance of amyloid-beta plaques
- Regulates over 1,700 different genes (1/20 of human genome)
- Shuts down the gene that makes SirT1, which helps with gene regulation (<u>resveratrol</u> would help this)
- Activates NF-xB, the Lynch pin of inflammation (Turmeric and Omega 3's would help this)

<u>ApoE4</u> (14% of the population) is the worst, followed by <u>ApoE3</u> (78%), then <u>ApoE2</u> (8%).

It's also a good idea to check any mutations or polymorphisms in APP, PS1, PS2, Construction of Trever Beny, DC, DACNE is prohibited.



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RECODE



ERCHONIA

Treatment

Treatment is different for everyone, but simply goes like this:

- 1. Fixing the **underlying cause** (infections, toxin exposure, chronic inflammation)
- 2. Changing lifestyle to increase neurotrophic factors and proper autophagy
- 3. Using <u>diet</u> and treatments to **restore biomes** and **insulin sensitivity** in the brain/body
- Optimizing hormones and other biomarkers using bioidentical hormones, supplements, and herbs

Infections

Treat MARCoNs if positive.

- Inactivate/excrete pathogens using:
- IV glutathione
- Intranasal VIP
- Vasoactive Intestinal Peptide
- Helps optimize other hormones
- Cruciferous foods (more listed in <u>diet</u>)

Here are some other useful tools to help remove infections or toxins:

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RECODE

- <u>Activated Charcoal</u>
- <u>Alpha lipoic acid</u>
- <u>Chitosan</u>
 - <u>Chlorella</u>

- Cholestyramine
- <u>Guggul</u>
- <u>Manganese</u>
- <u>NRF2 Activation</u>
- <u>Restore4Life</u>
- <u>Sauna</u>
- <u>Vitamin B6</u>
- Vitamin C
- Welchol
- Zinc picolinate

DR. BERRY CONSIDERATIONS: ARGENTYN SILVER VIRAGRAPHIS/ANDROGRAPHIS BIOCIDIN/LAURICIDIN-MONOLAURIN CELL CORE PRODUCTS ERCHONIA LLLT

Lifestyle

These are lifestyle requirements for the ReCODE protocol that help reverse AD:

Sleep:

- <u>8 hours of sleep</u>/night (also going to bed before midnight) SEE NEW SLEEP STUDIES
- No blue light at night
- No <u>EMFs</u> at night

Brain Stimulation:

- Do brain training games (SEE DR. BERRY APP LIST)
- Exercise (EWOT, HIIT, COMPLEX MOVEMENTS)
- Increase neurotrophic factors (<u>BDNF</u> and <u>NGF</u>, but I recommend <u>CNTF</u>, <u>GDNF</u>, <u>CDNF</u> and <u>MANF</u> as well)

FAST, HIIT, BACOPA, LOW LEVEL LASER THERAPY

Psychological:

 Keep stress low (for reduced atrophy) SEE ADRENAL SECTION

Oral Hygiene:

- Brush and floss daily
- <u>Coconut pulling</u>
- (ERCHONIA LLLT)

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Diet (Ketoflex 12/3)

The <u>ReCODE diet</u>, called "<u>Ketoflex 12/3</u>", consists of being in ketosis, eating high amounts of fiber, eating within a 12 hour window, and stop eating at least 3 hours before bed.

The goals:

- Increases ketone bodies (acetoacetate, beta-hydroxybutyrate, and acetone) and BDNF
- <u>MCT oil (caprylic acid</u> is the strongest form) is a must for <u>ApoE4</u> until insulin sensitivity is restored, then must switch to MUFAs (like avocado) and PUFAs predominantly (such as <u>olive oil</u>)
- Lots of uncooked veggies
- Fasting 12 hours/day
- Stop eating 3 hours before bed
- Increase insulin sensitivity
- RECODE

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Diet (Ketoflex 12/3) SUGGESTED FOODS LIST:

Eat frequently:

- Avocados
- Artichokes
- Beets
- Cilantro
- Cruciferous vegetables (such as cauliflower, broccoli/broccoli sprouts, various types of cabbage, kale, radishes, Brussels sprouts, turnips, watercress, kohlrabi, rutabaga, arugula, horseradish, maca, rapini, daikon, wasabi, and bok choy)
- Dandelions
- Garlic
- Ginger
- Grapefruit

- Jicama
 - Kimchi
 - Leafy greens (such as kale spinach, and lettuce)
 - Leeks
 - Lemons
 - Mushrooms
 - <u>Olive oil</u>
 - Onions
 - Pasture raised eggs
 - Resistant starches (such as sweet potatoes, rutabagas, parsnips, and green bananas)
 - Saurkraut

- Tea (oolong, black, and green)
- Wild-caught fish (SMASH fish such as salmon, mackerel, anchovies, sardines, and herring)

Eat less frequently:

- Coffee (such as super coffee)
- Grass-fed beef
- Legumes (such as peas and beans)
- Nightshades (such as eggplant, peppers, and tomatoes)
- Nontropical fruits (low glycemic, such as berries)
- Pasture raised chicken
- Starchy veggies (such as corn, peas, squash, but sweet potatoes are an exception)
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Diet (Ketoflex 12/3):

Avoid:

- Dairy (occasional cheese or plain yogurt is okay, I recommend **A2-based dairy**)
- Fruits (high glycemic ones especially)
- Gluten
- Grains
- High **mercury** fish (such as tuna, shark, and swordfish)
- Processed foods
- Sugar and simple carbs (including breads, wheats, rice, cookies, cakes, candies, sodas, etc)

Some important notes about the diet:

- Avoid overheating foods (as it creates AGEs)
- Fish is good, but don't do too much meat.
- Remove all inflammatory lectins. <u>R</u>
- If you do eat fruits, make sure they are higher in fiber and not as juice.
- Include lots good fats in your diet (such as avocados, <u>olive oil</u>, <u>MCT oils</u> like <u>caprylic acid</u>, and if non-lectin sensitive then nuts and seeds oils are okay)
- Use <u>digestive enzymes</u>
- 71

RECODE



MicroBiome (RUN GUT ZOOMER 3.0)

Include probiotics and prebiotics:

- <u>B. lactis (fermented dairy)</u>
- <u>B. longum</u> (fermented veggies and dairy)
- <u>L. acidophillus (fermented dairy)</u>
- <u>*L. brevis*</u> (sauerkraut and pickles)
- <u>L. plantarum (kimchi, sauerkraut and fermented veggies)</u>
- Probiomax
- <u>S. boulardii</u>

If you have any infections with **biofilms**, you must take care of those as well (may use **Bactroban/Mupirocin**, **SinuClenz**, or **Xlear**).

For the nasal microbiome:

- <u>Kimchi juice</u> + <u>nasal swab</u>
- <u>Restore4Life</u>

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Insulin Resistance

Here are some supplements recommended for decreasing insulin resistance:

- <u>Alpha lipoic acid</u>
- Berberine
- <u>Chromium picolinate</u>
- <u>Cinnamon</u>
- Magnesium Glycinate
- <u>Magnesium Threonate</u>
- Metformin (drug)
- Zinc picolinate
Supplements Anthon there is a second second

RECODE



Supplements on the ReCODE program that help with cognition and inflammation:

- <u>ALCAR</u>
- <u>Citicoline</u>
- <u>Coffee fruit extract</u>
- DHA/EPA (fish oil or krill oil)
- <u>Nicotinamide riboside</u> (combines well with <u>resveratrol</u>)
- **Pantothenic acid** (use **<u>B6/B12/folate</u>** if homocysteine ≥ 6)
- POO
 pyrroloquinoline quinone supports CoQ10
- <u>Resveratrol</u>
- <u>Ubiquinol</u>
- <u>Vitamin B1</u>
- <u>Vitamin C</u>
- <u>Vitamin D</u>
- <u>Vitamin E</u>
- <u>Vitamin K2</u>

MAGTEIN (Mg L Threonate)

- Herbs on the ReCODE program that help with cognition and inflammation:
 - <u>Ashwagandha</u>
- Bacopa
- <u>Gotu Kola</u>
- <u>Guduchi</u>
- <u>Guggul</u> (or <u>activated charcoal</u>)
- Lion's Mane
- Rhodiola
- <u>Skullcap</u>
- Triphala (Amalaki + Haritaki + Bibhitaki)

Also, pro-resolving mediators (like **SPM Active**), such as **resolvins**, **protectins**, and **maresins** will also help against inflammation.

RECODE

73

Mechanism Of Action

Here are all the functions that the ReCODE protocol aims to accomplish:

- Increase α-cleavage
- Increase ADNP
- Increase autophagy
- Improve axoplasmic transport
- Increase <u>BDNF</u>
- Increase cAMP
- Increase GABA
- Increase glutathione
- Increase IDE
- Increase insulin sensitivity
- Improve LTP

- Increase <u>NGF</u>
- Increase microglial clearance of $A\beta$
- Increase netrin-1
- Increase neprilysin
- Increase PPAR-γ
- Increase phagocytosis index
- Increase PP2A
- Increase resolvins
- Increase SirT1
- Increase synaptoblastic signaling
- Increase telomere length
- Improve vascularization
- Increase VIP

ecommunity Education

ERCHONIA

- Optimize all **metals**
- Optimize <u>cholinergic</u> neurotransmission
- Optimize cortisol
- Optimize detoxification
- Optimize DHEA
- Optimize E2:P (estradiol to progesterone) ratio
- Optimize estradiol
- Optimize free T3
- Optimize free T4
- Optimize insulin secretion and signaling

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Mechanism Of Action

- Optimize mitochondrial function and biogenesis
- Optimize pregnenolone
- Optimize progesterone
- Optimize stem-cell-mediated brain repair
- Optimize synaptic components
- Optimize testosterone
- Optimize **TSH**
- Reduce amyloid-beta oligomerization
- Reduce APPβ-cleavage
- Reduce caspase-6 cleavage
- Reduce caspase-3 cleavage

- Reduce γ-cleavage
- Reduce glial scarring
- Reduce homocysteine
- Reduce inflammation
- Reduce **mTOR** activation
- Reduce NF-*×*B
- Reduce phospho-tau
- Reduce oxidative damage and optimize ROS
- Reduce synaptoclastic signaling

ONE OTHER CONSIDERATION THAT HAS BEEN UTILIZED BY HIS TEAM...

LOW LEVEL LASER THERAPY AND DEMENTIA



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

PMCID: PMC7956031 PMID: <u>33995964</u>

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 $(A\beta)$ 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/CM9.000000000001301

PMCID: PMC7990011 PMID: <u>33507006</u>

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 of Orisk ND and this material in whole or in part

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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 moderateintensity 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: <u>10.2147/IJGM.S280559</u>

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,

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. 78

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. 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 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 AB-induced cell apoptosis through the Akt/GSK3B/B-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.

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.

FRCHONIA

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)

OPTIMAL WAVELENGTHS, ENERGY AND FREQUENCIES



The Universe Doesn't Speak English.



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LASER <u>ENERGY</u> FUELS THE MITOCHONDRIA

WHAT IS THE IMPORTANCE OF MITOCHONDRIA?

The human body is fueled by trillions of microscopic powerhouses called mitochondria. Present in almost every cell in our bodies, these tiny but mighty, energy-producing structures generate the energy we need to survive

Mitochondria produce 90% of the energy (ATP) our body needs to function and play a pivotal role in cell life and cell death.

HOW IMPORTANT ARE THE MITOCHONDRIA?



SEPTEMBER 20, 2023

Exploring the mind-mitochondria connection

Picard winces a little whenever the book calls mitochondria the "powerhouses of the cell"

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."

Open Access
 Published: 10 August 2023

Brain mitochondrial diversity than dent work for gan bady or preduction and male mice



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/10.2389/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*†

ALZHEIMER'S CAUSES



KU Doctor Wins Big Grant to Pursue New Alzheimer's Theory AUG 31,2022

A local physician who has spent decades pursuing an alternative theory for the cause of Alzheimer's has gotten crucial affirmation — a chunk of the \$4 million Oskar Fischer Prize.

Swerdlow, who was awarded \$300,000, is hoping to move Alzheimer's research in a direction he's been studying since the 1980s. He has long posited that mitochondria are at the heart, and cause, of Alzheimer's disease. Mitochondria are membrane-bound parts of cells that generate most of the chemical energy needed to power cells' biochemical reactions.

WHAT PRODUCES ou Per Iso At I Por Pry DE DECNB is prohibited. MITOCHONDRIA?





LOCATION OF ELECTRON TRANSPORT

EACH COMPLEX (1-4) OF THE ELECTRON TRANSPORT CHAIN PLAYS A CRUCIAL ROLE IN PRODUCTION OF ATP. IF ONE OR MORE OF THE COMPLEXES ARE INHIBITED, ATP WILL BE LIMITED.



WHAT DISEASES ARE AFFECTED BY THE MITOCHONDRIA?



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MITOCHONDRIA



ERCHONIA

THERE MUST BE INTEGRITY OF ALL COMPLEXES FOR MITOCHONDRIAL FUNCTION



WHICH COMPLEXES ARE NEEDED FOR MITOCHONDRIAL COUPLING...



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Mol Nutr Food Res

2008 Jul;52(7):780-8. doi: 10.1002/mnfr.200700075.

Medication-induced mitochondrial damage and disease

John Neustadt 1, Steve R Pieczenik

Table 1. Signs, symptoms and diseases associated with mitochondrial dysfunction [29]

Organ system	Possible symptom or disease
Muscles	Hypotonia, weakness, cramping, muscle pain, ptosis, opthalmoplegia
Brain	Developmental delay, mental retardation, autism, dementia, seizures, neuropsychiatric disturbances, atypical cerebral palsy, atypical migraines, stroke, and stroke-like events
Nerves	Neuropathic pain and weakness (which may be intermittent), acute and chronic inflammatory demyelinating polyneuropathy, absent deep tendon reflexes, neuropathic gastrointestinal problems (gastroesophageal reflux, constipation, bowel pseudo-obstruction), fainting, absent or excessive sweating, aberrant temperature regulation
Kidneys	Proximal renal tubular dysfunction (Fanconi syndrome); possible loss of protein (amino acids), magnesium, phosphorus, calcium, and other electrolytes
Heart	Cardiac conduction defects (heart blocks), cardiomyopathy
Liver	Hypoglycemia, gluconeogenic defects, nonalcoholic liver failure
Eyes	Optic neuropathy and retinitis pigmentosa
Ears	Sensorineural hearing loss, aminoglycoside sensitivity
Pancreas	Diabetes and exocrine pancreatic failure
Systemic	Failure to gain weight, short stature, fatigue, respiratory problems including intermittent air hunger.

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MITOCHONDRIAL CONDITIONS



Mol Nutr Food Res

Medication-induced mitochondrial damage and disease

John Neustadt 1, Steve R Pieczenik

2008 Jul;52(7):780-8. doi: 10.1002/mnfr.200700075.

Table 5. Medications documented to induce mitochondrial damage [10, 35, 63-90]

Drug class	Drugs				
Alcoholism medications	Disulfiram (Antabuse®)				
Analgesic (for pain) and anti-inflammatory	Aspirin, acètaminophen (Tylenol), diclofenac (Voltaren®, Voltarol®, Diclon®, Dicloflex® Difen and Cataflam®), fenoprofen (Nalfon®), indomethacin (Indocin®, Indocid®, Indochron F-R® Indocins-SR®), Nanroxen (Aleve® Nanroxen®)				
Anesthetics	Bupivacaine, lidocaine, propofol				
Angina medications	Perhexiline, amiodarone (Cordarone®), Diethylaminoethoxyhexesterol (DEAEH)				
Antiarrhythmic (regulates heartbeat)	Amiodarone (Cordarone)				
Antibiotics	Tetracycline, antimycin A				
Antidepressants	Amitriptyline (Lentizol), amoxapine (Asendis), citalopram (Cipramil), fluoxetine (Prozac, Symbyax, Sarafem, Fontex, Foxetin, Ladose, Fluctin, Prodep, Fludac, Oxetin, Seronil, Lovan)				
Antipsychotics	Chlorpromazine, fluphenazine, haloperidol, risperidone, quetiapine, clozapine, olanza- pine				
Anxiety medications	, Alprazolam (Xanax®), diazepam (valium, diastat)				
Barbiturates	Amobarbital (Amytal®), aprobarbital, butabarbital, butalbital (Fiorinal®, hexobarbital (Sombulex®), methylphenobarbital (Mebaral®), pentobarbital (Membutal®), phenobarbital (Luminal®), primidone, propofol, secobarbital (Seconal®), Talbutal®), thiobarbital				
Cholesterol medications	Statins – atorvastatin (Lipitor®, Torvast®), fluvastatin (Lescol®), Iovastatin (Mevacor®, Altocor®), pitavastatin (Livalo®, Pitava®), pravastatin (Pravachol®, Selektine®, Lipostat®), rosuvastatin (Crestor®), simvastatin (Zocor®, Lipex®) bile acids – cholestyramine (Ques- tran®), clofibrate (Atromid-S®), ciprofibrate (Modalim®), colestipol (Colestid®), colesevelam (Welchol®)				
Cancer (chemotherapy) medications	Mitomycin C, profiromycin, adriamycin (also called doxorubicin and hydroxydaunorubicin and included in the following chemotherapeutic regimens – ABVD, CHOP, and FAC)				
Dementia	Tacrine (Cognex®), Galantamine (Reminyl®)				
Diabetes medications	Metformin (Fortamet [®] , Glucophage [®] , Glucophage XR, Riomet ¹), troglitazone, rosiglita- zone, buformin				
HIV/AIDS medications	AtriplaÔ, Combivir®, Emtriva®, Epivir® (abacavir sulfate), EpzicomÔ, Hivid® (ddC, zalcita- bine), Retrovir® (AZT, ZDV, zidovudine), Trizivir®, Truvada®, Videx® (ddl, didanosine), Videx® EC, Viread®, Zerit® (d4T, stavudine), Ziagen®, Racivir®				
Epilepsy/Seizure medications	Valproic acid (Depacon®, Depakene®, Depakene syrup, Depakote®, depakote ER, depakote sprinkle, divalproex sodium)				
Mood stabilizers	Lithium				
Parkinson's disease medications	Tolcapone (Tasmar [®] , Entacapone (COMTan [®] , also in the combination drug Stalevo [®])				

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Lin et al. BMC Bioinformatics (2021) 22:369 https://doi.org/10.1186/s12859-021-04285-3 **BMC Bioinformatics**

Open Access

RESEARCH

MitoTox: a comprehensive mitochondrial toxicity database

Yu-Te Lin¹, Ko-Hong Lin², Chi-Jung Huang¹ and An-Chi Wei^{1,2*}

From The 19th Asia Pacific Bioinformatics Conference (APBC 2021) Tainan, Taiwan. 3-5 February 2021

*Correspondence: acweiß6@intuedutw / 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 mitachondrial toxicity-related drugs in MitaTox



ERCHONIA

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	Hepatotox- icity	16–34	>99%	1.9768 mol/ kg
	Rosiglita- zone	Inhibition of ETC	Approved, investiga- tional	Cardiotox- icity	3–4	99.8%	2.4515 mol/ kg
	Pioglitazone	Inhibition of ETC	Approved, investiga- tional	Cardiotox- icity	3–7/16–24	>99%	2.0115 mol/ kg
	Ciglitazone	Inhibition of ETC	Discontin- ued	NA	NA	NA	NA
	Darglitazone	Inhibition of ETC	Discontin- ued	NA	NA	NA	NA
	Muraglitazar	Inhibition of ETC	Discontin- ued	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	Cardiotox- icity	20–48	74–76%	21.8 mg/kg
	Cisplatin	Inhibition of Complex I	Approved	Nephrotox- icity	0.3–0.7	>90%	2.7612 mol/ kg
Hyperlipi- demia	Fenofibrate	Inhibition of complex I	Approved	Hepatotox- icity	20	~ 99%	>2000 mg/ kg
	Clofibrate	Inhibition of complex I	Approved	Hepatotox- icity	18–22	95–97%	940 mg/kg



l	Psycho- tropic	Valproic acid	Inhibition of TCA cycle	Approved	Coma and res- piratory depres- sion	9–16	90%	670 mg/kg
l		Clozapine	Inhibition of the ETC	Approved	Metabolic syndrome	8	97%	3.0838 mol/ kg
L		Fluoxetine	Uncoupler	Approved	CNS, GI effects	1–3 days	94.5%	2.6048 mol/ kg
l		Nefazodone	Inhibition of Complex I and complex IV	Withdrawn	Hepatotox- icity	2–4	>99%	2.9067 mol/ kg
L	Analgesic	Acetami- nophen	Oxidative stress	Approved	Hepatotox- icity	1–4	25%	1944 mg/kg
t		Aspirin	mPTP potentia- tion	Approved	Cardiotoxic- ity, Gl effects	0.25	99.5%	920– 1480 mg/ kg
l		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
L		Chloram- phenicol	Depletion of iron	Approved	Aplastic anemia	1.5–3.5	50-60%	2500 mg/kg
	Antiviral	Zidovudine	mtDNA replica- tion (inhibit mtDNA	Approved	Myopathy	0.5–2.9	30–38%	NA
			polymerase-y)	95				

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 ^T, Camilo Lopez-Alarcón, Margarita E Aliaga, Hernán Spelsky 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 (IKSAID). A major objective of the present study was to investigate whether in vitro the IKSAID, applini, Indomethadin, dicidenac, piroxicam and ibuporfen, which feature different chemical structures, are able to inhibit mitochondrial complex I. All IKSAID swere effective inhibitors when added both, directly to mitochondria isolated from rat duodenum epithelium (50 µÅ) or to Caco-2 cells (250 µÅ). In the former system, complex I. inhibitos were endercive material and susceptible to complexition and reversion by the addition of coenzme Q (22.5-S20 µÅ). Based on reports suggesting a potential gastro-protective activity of quercelin, the ability of this flavonoid to protect isolated mitochondria against IKSAID-induced complex I inhibition was esaluated. Low micromolar concentrations of quercerin (1-20 µÅ) protected against such inhibitorin, in a concentration dependent maner. In the case of aspin, quercetin (51-0) µÅ) can behave as a 'coenzme Q-mimetic' indicule, allowing a normal lectron flow along the whole electron transporting chain (complexes 1, III and IV). The exposed findings reveal that complex i inhibiton is a common deleterious effect of IKSAIDs at the mutochondrial level, and that such effect is, for all tested agents, susceptible to be prevented by guercelin. Data provided here supports the contention that the protective action of quercetin esides on its, here for first time-shown, ability to behave as a coenzine Q-like molecule.

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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.

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MITOCHONDRIAL DAMAGE



Save Email

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

Pub Med.gov

> 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 Goncalves, 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-acetyloysteine (NAC) plus deferoxamine (DFX) or taurine. These results showed that acetaminophen administration inhibited the activities or of orapites I and IV in cerebra showed mata accaming the animation minimized in extinct of complexes I and IV in cerebra 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 actaminophen 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



Bioscience Reports (2021) 41 BSR20211520 https://doi.org/10.1042/BSR20211320

Review Article

Beyond base excision repair: an evolving picture of mitochondrial DNA repair

Kathrin Allkanjari¹ and O Robert A. Baldock²

¹Formerly: Solent University Southampton, East Park Terrace, Hall, Swindon Road, Cheltenham GL50 4AZ, UK ce: R.A. Baklock

Unlike other organelles, mitochondria contain their own DNA (mtDNA) and must either be tolerated or corrected to preserve mitochondrial function.

portantly, replication of damaged mtDNA can d to stalling and introduction of mutations on netic loss.

rsfunctional mitochondria survive longer than ealthy mitochondria. In this way, some types of sfunctional mitochondria build up and entually become more common than healthy

Mitochoodria are highly specialised organalise required for key calular processes including ATP production through calular respiration and controlling call death via apoptosis. Unlike other organelise, mitochondria contain their own DNA genome which encodes both protein and FNA required for calular respiration. Each call may contain hundreds to thousands of copies of the mitochondrial dynamic respiration. Each call may contain hundreds to thousands of the mitochondrial genome, which is essential for normal callar galangiang and dis-ease. Furthermore, mtDNA lacions can arise from both endogenous or exceptous sources and must alther be tosteated or corrected to preasore mitochondrial function. Importantly, replication of damaged mitDNA can had to statling and introduction of malations or genetic rely on nuclear-encoded DNA requir proteins that are translocated with thore hordinal bother and proteins, it remains to be established which DNA (mappi modernism) replication of damaged and the regular dynamics of the proteins being found in the mi-tochondrial proteoms, it remains to be established which DNA (mappi modernism) are functional in mammalian mitochondria. Here, we summarise the existing and merging research, dioragis deast Excision Repair (EER). Hornologous Recombination (HB) may Microhomology- midduted CII during MMC), chronic plane HDNA damage can be repaired using Base Excision Repair (EER). Hornologous Recombination (HB) mito-mation diarding the books postfiwang used to to lotentor, repair microhamism & or replication is discussed, importantly, characteriain pon-canonical functions of key protein and undersiding the books postfiwang used to to lotentor, repair or bypase DNA damage will be fundamential h fully understanding the causes of mitochondrial genome mutation and mitochondrial dysfunction.

Entroduction Mitochoadra are highly specialised and dynamic organelles required for fundamental cellular pro-cresses including ATP generation via oxidative phosphorylation during callular repiration and the con-sinchoadra constant better own 1654 bereader DNA processome (often arfered to a mainchoadra) report of the standard of the standard DNA processome (often arfered to a mainchoadra) report of the standard distribution of the standard DNA and a Prihosonal RNAs enable protein synthesis of these proteins within the mitochoadra [15]. Compartmentalised in the mitochoadra intert, each cell is estimated to contain handreds to thousands of copies of the mitochoadra intert, each cell is estimated in the distribution of structures marked predominally by association with mitochoadrail transcription factor A (TFMA) and several other IDMA association with mitochoadrail transcription factor A (TFMA) and several other IDMA association with mitochoadrail transcription factor A (TFMA) and several other IDMA association with mitochoadrail transcription factor A (TFMA) and several other IDMA association with mitochoadrail and the standard in compared on (a).

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MITOCHONDRIAL DAMAGE



Mitochondrial disorders comprise a molecular and clinically diverse group of diseases that are associated with mitochondrial dysfunction leading to multiorgan disease.

However, the therapeutic approaches for mitochondrial disorders have lagged behind with <u>treatment options limited mainly to</u> tom specific therapies and supportive



王光

Review article

Primary mitochoodrait disorders represent an expanding group of clinatally heterogeneous diseases that are associated with mitochoo-land dynatic on the ounderping matchino in mitochoodraila IDNA imDNA is on unclear encoded mitochoodrail groots, miDNA code for 13 mitoRA and the second second second second second second matchino and the second second second second second second matchino and the second second second second second second matchino and the second s uncoln (1,2,) As introduction are are ough OXPHOS complexes, energy involvement usually seen in mito-also carry other biological function steroid synthesis, and apoptosis a e pathophysiology in various mitor

of deep, high-throughout, in-parallel DNA sequencing

-usreponung autor at: Department of Molecular and Human Genetics, Baylor College of Medicine and Texas Children's Hospital, Mark Wallace Tow S60,9701 Texas, 77030, USA Street, Hoston, Texas, 77030, USA
 - Rend adverse: Engingementor, 67, Sequita.

THE MITOCHONDRIAL SOLUTION...



ondrial on-m, 244 well-chara-"ochondrial disr "ology.

iore, a rec

ers [5] that could lay the

chanisms of a growing list of mitochor ology of inborn errors of metabolism, ers are listed under the category of mit s matabolism (http://www.lembare.org

nosology of Inborn errors or m orders are listed under the cate ergy metabolism (http://www.l on 4/18/2020) [4]. Furtherm epidemiological study has pro

history of mitoche

ious mitochondrial mostly limited to sy including treatment use of vitamins, cof Recently, the nu mostly limited to symptom specifi including treatment of catabolic se-use of vitamins, cofactors, and an Recently, the number of preci-drial disorders has been Increasin more specific and effective trea disorders. In this review, various i

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 ${\sim}3~{
m eV}$ 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



GREEN LASER COMPLEX 3



COMPLEX 3 CYTOCHROME BC 1 COMPLEX

THE MITOCHONDRIAL SOLUTION...





> 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/i responsible of the mitochondria-red light interaction, isolated rat liver mitochondria were insalated with a Helium-Neon laser (energy doce 2 Joules/cm2, light power 10 mit) and measurement made of the activity of cytochrome c oxidaxe. A low, but statistically significant increase in the oxygen uptake was found, as paloargaphicity, measured, in the presence of robene and antimycin, a vitth accordate and TMPD used as substate pair. Measurements were also made both of the election transfer and of proton pumping activity, as a result of a major stimulation in the proton pumping activity, about 55% increase of ~-H-ke- ratio was found in inclaited mitochondria.

Related information

PubChem Compound (MeSH Keyword)

RED LASER COMPLEX 4

COMPLEX 4 <u>CYTOCHROME C OXIDASE</u>

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WHY LOW LEVEL LASER THERAPY AND NOT OTHER LIGHT DEVICES





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

OPTIMAL WAVELENGTHS



VIOLET EXCITES 3.06 EV (3 LEVELS) **GREEN EXCITES 2 LEVELS RED EXCITES 1 LEVEL** THIS DONATES ELECTRONS TO

THE ELECTRON TRANSPORT CHAIN



Exciting **Electrons**

• For a photon to be absorbed, the energy (Ev) of the photon must match the difference between the energy (eV) between the obits of electrons in order to move to higher state.

• Energy Levels of all atoms:

To allow electrons to move to higher orbit the wavelength must be between 380nm to 683nm

AVOID TOO SHORT EX. XRAYS OR LONGER ASSOCIATED WITH HIGHER POWER

WITH ALL OF THE PHENOMENAL RESULTS RESEARCH HAS SHOWN WITH RED LASER IF UTILIZED AT LOW DOSE, WHAT ARE WE SEEING WHAT DO WE SEE WITH HIGHER ENERGY SHORTER WAVELENGTHS?

OPTIMAL WAVELENGTHS



ÉRCHONIA



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THE MITOCHONDRIAL SOLUTION...



ARE



INTRODUCING THE NEW GVL!





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EV VS POWER



IN SUMMARY, IT DOESN'T MATTER HOW MUCH POWER YOU CRANK UP WITH A LASER (WATTS). ENERGY (ELECTRON VOLT) IS NOT DEPENDENT ON POWER, IT IS DEPENDENT ON WAVELENGTH (NANOMETERS)! THE SHORTER THE WAVELENGTH, THE MORE ENERGY AND THE MORE YOU CAN EXCITE THE ELECTRONS TO DONATE ENERGY (RED 1 ORBIT, GREEN 2 ORBITS, VIOLET 3 ORBITS)

MAKING THE NEW GVL THE HIGHEST ENERGY DEVICE ON THE MARKET

BY ADDING MORE POWER TO LONGER WAVELENGTHS YOU ARE MERELY HEATING THE WATER MOLECULES PRIMARILY AT THE SURFACE LAYERS CAUSING HEAT.

THIS IS WHY THESE DEVICES ONLY HAVE FDA 510K MARKET CLEARANCE AS A MEDICAL HEAT LAMP

VS

PHOTOCHEMICAL REACTIONS PROVIDED BY ERCHONIA'S VISIBLE WAVELENGTHS AND THESE WAVELENGTHS ARE NOT POWER

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THE MITOCHONDRIAL SOLUTION ...



TREATING THE ELECTRON TRANSPORT CHAIN WITH ERCHONIA LASER



GOOD: RED DIODES BETTER: RED PLUS VIOLET BEST: RED. VIOLET AND GREEN

© 2022 Erchonia Corporation

WHICH LASER SHOULD I GET...









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

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LASER PROFIT CENTER IDEA



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





FX635

CLINICAL CONSIDERATIONS...



FX405

© 2022 Erchonia Corporation

RENTAL

Fixing the ETC.

- 1. The GVL is used in office to upregulate the initial ETC complex's (1-3).
- 2. Then the doctor rents out the XLR8 (more affordable device) so the patient can upregulate complex 4 at home.



BASE STATION RENTAL PROGRAM IS A MUST FOR CLINICIANS! ASK YOUR LOCAL REP FOR THE FORMS AND WEBINARS

CLINICAL CONSIDERATIONS...



BASESTATION

This allows Doctors to be Doctors and diagnose based on the condition and the possible underlaying health issues.

Each complex (1-4) of the electron transport chain plays a crucial role in production of ATP. if one or more of the complexes are inhibited, atp will be limited.



Example 1: NSAIDS are linked to inhibition of Complex 1. if the doctor is aware the patient commonly consumes NSAIDS, they should use the EVRL or GVL prior to the XLR8

<section-header><section-header><section-header><text><text><text>

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Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit vascular smooth muscle proliferation via differential effects on the cell cycle.

Example 2: If the patient is young athlete that is injured. Then red laser only would be suitable.

Example 3: If doctor is treating infection, the EVRL would be best due to the antimicrobial properties of the violet and the immune boasting properties of the red laser.

THE IMPORTANCE OF MITOCHONDRIA



2017 Oct;95(10):2025-2029. doi: 10.1002/jnr.24042. Epub 2017 Mar 16.

Mitochondrial biogenesis in neurodegeneration

P Andy Li 1, Xiaolin Hou 1 2, Shaocai Hao 1 3

Abstract

Mitochondria play a key role in energy production, calcium homeostasis, cell survival, and death. Adverse stimulations including neurodegenerative diseases may result in mitochondrial dynamic imbalance, free radical production, calcium accumulation, intrinsic

cell death pathway activation and eventually cell death. Therefore, preserving or promoting mitochondrial function is a potential therapeutic target for the treatment of neurodegenerative disorders. Mitochondrial biogenesis is a process by which new mitochondria are produced from existing mitochondria. This biogenesis process is regulated by Peroxisome proliferator-activated receptor-gamma (PPARy) coactivator-1alpha (PGC-1a). Once being activated by either phosphorylation or de-acetylation, PGC-1a activates nuclear respiratory factor 1 and 2 (NRF1 and NRF2), and subsequently mitochondrial transcription factor A (Tfam). The activation of this PGC-1a - NRF -Tfam pathway leads to synthesis of mitochondrial DNA and proteins and generation of new mitochondria.

Stem Cells Dev

2017 May 15;26(10):762-775. doi: 10.1089/scd.2016.0332. Epub 2017 Mar 27.

Low-Level Laser Effect on Proliferation, Migration, and Antiapoptosis of

Mesenchymal Stem Cells

The mechanism of the effects included proliferation rate increase mediated by increased S phase proportion; **mitochondrial biogenesis** and function alteration mediated by fusion (Mfn1, Mfn2, and Opa-1) and fission (Fis1, Drp-1, and MTP18)-related proteins, NRF1, TFAM, PGC-1a, and upregulated intracellular ROS and NO concentration; migration acceleration through the ERK1/2 and FAK pathway and upregulation of growth factors such as HGF and PDGF; and resistance to apoptosis with increased Bcl-2 and decreased Bax, or through tunneling nanotube formation between LLL-treated MSCs and 5-fluorouracil-induced apoptotic MSCs. These observations suggested that LLLs enhanced stem cell survival and therapetication clicit. Which costd appear to be an information between the application of MSCs. without permission of Trevor Berry, DC, DACNB is prohibited.

Mol Neurobiol



2020 Jul;57(7):2959-2980. doi: 10.1007/s12035-020-01926-1. Epub 2020 May 22.

The Role of Mitochondria in Neurodegenerative Diseases: the Lesson from Alzheimer's Disease and Parkinson's Disease

Giacomo Monzio Compagnoni 1 2 3, Alessio Di Fonzo 4, Stefania Corti 4 5, Giacomo P Comi 4 5, Nereo Bresolin 4 5, Eliezer Masliah 6

Abstract

Although the pathogenesis of neurodegenerative diseases is still widely unclear, various mechanisms have been proposed and several pieces of evidence are supportive for an important role of mitochondrial dysfunction. The present review provides a comprehensive and up-todate overview about the role of mitochondria in the two most common neurodegenerative disorders: Alzheimer's disease (AD) and

Parkinson's disease (PD). Mitochondrial involvement in **AD** is supported by clinical features like reduced glucose and oxygen brain metabolism and by numerous microscopic and molecular findings, including altered mitochondrial morphology,

impaired respiratory chain function, and altered mitochondrial DNA. Furthermore,

amyloid pathology and mitochondrial dysfunction seem to be bi-directionally correlated. Mitochondria have

an even more remarkable role in PD. Several hints show that respiratory chain activity, in particular complex I, is impaired

in the disease. Mitochondrial DNA alterations, involving deletions, point mutations, depletion, and altered maintenance, have been described. Mutations in genes directly implicated in mitochondrial functioning (like Parkin and PINK1) are responsible for rare genetic forms of the disease. A close connection between alpha-synuclein accumulation and mitochondrial dysfunction has been observed. However, the presented data strongly support the hypothesis that whatever the initial cause of neurodegeneration is, mitochondrial impairment has a critical role in maintaling and fostering the neurodegenerative process.



MITOCHONDRIA AND NEURODEGENERATION (STUDY WAS LIMITED TO COMPLEXES 1&4)



0

In summary, the strongest support for a role for complex I and/or IV deficits, is in the pathophysiology of PD and AD.

MDD	Small number of studies with small sample sizes Region-specific heterogeneity Moderate effects in complex I, small effects in complex IV
BPD	Small sample sizes Region-specific heterogeneity Moderate effects in complex I, small effects in complex IV
SZ	Small sample sizes Region-specific heterogeneity Moderate effects in complex I and IV
AD	Large number of studies with large sample sizes Tissue-specific heterogeneity (low in blood, high in brain) Strong effects in complex I and IV
PD	Large number of studies No heterogeneity Strong effects in complex I and IV
AGING	Most robust effects No heterogeneity Strongest effects in complex I and IV

Neuropsychopharmacology

REVIEW ARTICLE

Multivariate meta-analyses of mitochondrial complex I and IV in major depressive disorder, bipolar disorder, schizophrenia, Alzheimer disease, and Parkinson disease

Holper¹, D Ben-Shachar² and JJ Mann¹

Complex (NADH delydiograms, NDI) and complex IV (sytochrome c ondexe, COI) of the inhibitorial deciron transport have been implexed in the pathophysical org of major polytatic disorders, such as Anheimer disorder IMOD), by disorder (RD), and schizophrenia (SD), as well as in neurodepenerative disorders, such as Anheimer disorae (ND) and Pakin disorae (PD). We conducted meta-analysics comparing complexe 1 and V in each Midore MOD, BD, SZ, AD, and PD, as mornina Jaging. The electronic distabases: Fuhimed, EMBAGE, CENTRAL, and Coogle Scholar, were searched for studies publish between 1980 and LSD (O2499 screense) studies (SZ) as factor were displied for these analyses. Complex La MV here as a in peripheral blood, muscle biogon, or postmortem baria at the level of enzyme activity or subunits. Sparate meta-analyses and analysis and the studies that studies that for studies for studies that shorts. er studies, MDD and BD, udies, MDD and BD, revealed moderat I cortex, cerebellum and striatum, whi ve disorders, AD and PD, showed stror tex, cerebellum, and substantia nigra. nce is less robust for MDD, BD, or SZ.

macology (2019) 44:837-849; https://doi.org/10.1038/s41386-018-0090-0

nction is implicated in the pathophysiology biodres, such as major depresive disorder biologie (BD) [2] and kindprenia [G2 G12] society (BD) [2] and [2] and [2] and [2] and [2] society (BD) [2] and [2] and [2] and [2] and [2] society (BD) [2] and [2] and [2] and [2] and [2] and [2] society (BD) [2] and [2] and [2] and [2] and [2] and [2] society (BD) [2] and [2] and [2] and [2] and [2] and [2] and [2] society (BD) [2] and [Mitochondrial dysfunction is implicated in the path major psychiatric disorders; such as major depri (MDD) [1], bipolar disorder (BD) [2] and schizophre well as neurodegeneative disorders, such as Alzi (AD) [4] and Parkinson disease (PD) [4], Mi Intracellular organelles that produce adenosine (ATP), the main source of cellular energy. Inpaired function results in decreased ATP production, imp

of Molecular imaging and Neuropathology, Columbia University and New York State Psychiatric Institute, New York, NY, USA and "Lat stor of hypothesity, Remban Health Care Compass, Repapator Faculty of Medicine, Technion IIT, Hafe, brand demone, 1 Holger Industrial-Institution -

Received: 7 March 2018 Revised: 26 April 2018 Accepted: 7 May 2018 Published online: 16 May 2018

THIS STUDY ONLY ANALYSED COMPLEXES 1 AND 4

NEURODEGENERATION AND WAVELENGTHS



Ischemia/reperfusion injury, Parkinson's disease, Alzheimer's disease, neurodegenerative diseases, and other age-related degenerative changes.

Two mitochondrial electron-transfer complexes are major sources of ROS: complex I an ex III. Oxidative damage to either of these complexes would be expected to inhibit electron transport



MITOCHONDRIA AND NEURODEGENERATION





NEURODEGENERATIVE DISEASES

12.1



MITOCHONDRIA AND NEURODEGENERATION

SUMMARY OF MITOCHONDRIAL ETC COMPLEXES, COMMON NEURODEGENERATIVE DISEASES AND ERCHONIA WAVELENGTHS:

ALPHA SYNUCLEIN DISEASES INCLUDING PARKINSON'S, LEWY BODY DEMENTIA AND MULTIPLE SYSTEM ATROPHY (MSA): ETC COMPLEX 1 = VIOLET

TAU HYPERPHOSPHORYLATION DISEASES INCLUDING ALZHEIMER'S, FRONTOTEMPORAL DEMENTIA, PICK DISEASE, PROGRESSIVE SUPRANUCLEAR PALSY (PSP) AND CORTICOBASAL ATROPHY ETC COMPLEX 1 = VIOLET

> MHTT IN HUNTINGTON'S CHOREA ETC COMPLEX 2 = VIOLET



mSOD1 IN AMYOTROPHIC LATERAL SCLEROSIS (ALS) AND ALZHEIMER'S ETC COMPLEX 3 = GREEN

ALZHEIMER'S DISEASE: ETC'S 1,3 & 4 (VIOLET-GREEN-RED) STACK ALL 3 ERCHONIA WAVELENGTHS

PARKINSON'S DISEASE: ETC'S 1 & 4 (VIOLET-RED) FX 405 OR EVRL

HUNTINGTON'S DISEASE: ETC'S 2, 3 & 4 (VIOLET-GREEN-RED) STACK ALL 3 ERCHONIA WAVELENGTHS

ALS: ALL 4 COMPLEXES SO USE ALL 3 ERCHONIA DIODES AGING: ETC'S 1,2 & 4 (VIOLET-RED) FX 405 OR EVRL

DEFAULT TO MASTER BRANN CREED OF OUR DEFORM TACHING ON DISING 1-10-40-60 without permission of Trevor Berry, DC, DACNB is prohibited.

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"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 2023 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

MASTER TREATMENT ALGORITHM

1. MASTER BRAIN PROTOCOL FOR EACH DEVICE



ERCHONIA

- 2. BRAIN BALANCE BASED ON BEDSIDE NEURO EXAM OR OTHER OBJECTIVE MEASURES
- 3. "DOWN STREAM" CLEARANCE (CONSIDER ALWAYS DOING THIS TO GET BRAIN LASER EXPOSURE)
- 4. STACKING MODALITIES WITH LOCAL LASER EXPOSURE AND CO-ACTIVATION
- 5. "SET IT AND FORGET IT" (MAY BE ALL THAT IS NECESSARY)
- 6. FUNCTIONAL MEDICINE, INTEGRATIVE NEUROLOGY AND OTHER SYSTEMIC CONSIDERATIONS in whole or in part

PROTOCOL: YOUR DEFAULT PROTOCOL FOR ANY BRAIN APPLICATIONS



1. MOST COMMONLY USED FREQUENCIES (IN ORDER): 40 - 10 - 1 - 60 EX. OPTIMIZE: 10-40-10-40 MASTER BRAIN: 1-10-40-60 REST-DIGEST: 1-10-1-10 VAGUS/PEAK ALPHA: 10-10-10-10



2. BASE PREVENTION AND TREATMENT BRAIN PROTOCOLS:

FX 405 6 MINUTES WITH 1-40 TO PREFRONTAL CORTEX, 10-40 APICAL/PARIETAL, 10-40 BRAINSTEM CEREBELLUM IT'S OK TO TURN IT FROM SAGITTAL TO CORONAL PLANE AT 1/2 WAY. IF POSITIONED USE THE 1-10 TO THE RIGHT HEMISPHERE

FX 635 EXACT SAME SET UP AND FREQUENCIES AS FX 405 BUT DO A 10 MINUTE PROTOCOL (AGAIN, IT'S OK TO TURN THE DIODES FROM SAGITTAL TO CORONAL AT THE HALF WAY 5 MINUTE MARK)

FINISH EITHER FX DEVICE WITH THE "GUT SHOT". SCM/NECK, LUNG/UPPER GI, LOWER GI MINUM 4 MIN UP TO 10 MIN

HAND HELDS (USE ANY OF THE ABOVE COMMONLY USED FREQUENCIES):

GVL 2 MINUTES BILATERAL PREFRONTAL CORTEX, 2 MINUTES APICAL/PARIETAL, 2 MINUTES BRAINSTEM/CEREBELLUM

EVRL DO THE SAME POSITIONS AND GVL BUT 3 MINUTES PER REGION

XLR8 DO THE SAME POSITIONS AS OTHER HAND HELDS BUT DO 4 MINUTES PER REGION

FINISH ALL HAND HELDS WITH MINIMUM 2 MIN + 2 MIN VAGAL PROTOCOL (SCM/NECK AND OVER GUT) CAN DO UP TO 10 MINUTES (IE. WAND THE DEVICE OR 5 & 5 MIN NECK AND GUT)

NOTE: THESE ARE BASIC MINIMUM TREATMENT TIMES. YOU CAN SAFELY GO MUCH LONGER WITH ERCHONIA PRODUCTS BUT YOU MAY NOT NEED TO DUE TO THEIR WAVELENGTH ENERGY

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



ALPHA FREQUENCIES



PEAK ALPHA FREQUENCY

- -THE RESONANCE BETWEEN THE THALAMUS AND CORTEX
- -STATE OF RELAXATION, PEACEFULNESS AND ALERTNESS
- -THE OPTIMAL METAL 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 AWAK 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

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SOME OF DR. BERRY'S FAVORITE FREQUENCY SETTINGS



MASTER BRAIN 1-10-40-60 (70% OF MY PATIENTS, INCLUDING UPSTREAM/DOWNSTREAM PAIN)

VAGUS 10-10-10-10 BRAIN OPTIMIZATION 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 REPUGHA'S LASER A-Z PROTOCOL BOOK!

COMMON PATHOGEN AND IMMUNE FREQUENCIES

Community 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

SOLFEGGIO FREQUENCIES



What are Solfeggio Frequencies?

ALSO READ ABOUT ROYAL RIFE

The Solfeggio Frequencies are a series of 6 electromagnetic musical tones that the Gregorian Monks were said to use when they chanted in meditation. Rediscovered in 1974 by Dr. Joseph Puleo, the Solfeggio Frequencies are said to deeply penetrate the conscious and subconscious mind, stimulating inner healing. Dr. Puleo was intuitively led to rediscover these healing frequencies in the Book of Numbers (a book in the Hebrew Bible), using a numerological technique to decipher the six repeating codes he found. The result was the rediscovery of the Solfeggio Frequencies.

Physicist, inventor, and electrical engineer Nikola Tesla once said, "If you only knew the magnificence of the 3, 6 and 9, then you would hold a key to the universe." Interestingly, these three numbers form the root vibration of the six Solfeggio Frequencies.

- UT 396 Hz transforming grief into joy and guilt into for giveness
- RE 417 Hz clears negativity and removes subconscious blockages
- MI 528 Hz stimulates love, restores equilibrium, repairs DNA
- FA 639 Hz strengthens relationships, family, and community unity
- SOL 741 Hz physically cleanses the body from all types of toxins
- LA 852 Hz a waite anscitation of the proving the



THE MASTER PROTOCOL NEUROMUSCULOSKELETAL



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" RCHONIA 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)



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AYA



BREAK

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AFTER HOURS REQUESTS...





HAND DR. BERRY A WRITTEN REQUEST FOR ANY FUNCTIONAL MEDICINE TOPIC(S) YOU WANT TO LEARN MORE ABOUT THIS WEEKEND INCLUDING SUMMARY OF LABS, NUTRITIONAL SUPPORT, FUNCTIONAL NEUROLOGICAL APPLICATIONS AND XYMOGEN SPECIFIC PRODUCTS. DR. BERRY WILL ATTEMPT TO COVER THEM BEFORE THE END OF THE SEMINAR.

*SPECIAL THANKS TO DR. CHERYL BURDETTE FOR HER EXTENSIVE WORK AND EDUCATIONAL MATERIALS

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ALL 3 WAVELENGTHS POST INJURY



THE BRAIN-GUT AXIS





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VAGAL NERVE STIMULATION **AND BARRIER SYSTEMS**



ERCHONIA



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VAGAL NERVE STIMULATION

Jinflamm 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 CAN with entry of Trevor Berry, DC, DACNB is prohibited.



VAGAL NERVE STIMULATION



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

VAGAL NERVE STIMULATION



Highlights

Transcutaneous auricular vagus nerve stimulation (taVNS) can emulate interoceptive signals.

taVNS boosts stomach-brain coupling in the brainstem, midbrain, and transmodal cortex.

taVNS-induced changes in stomach-brain coupling mirror changes in hunger ratings.

non-invasive stimulation of the vagus nerve can strengthen the communication between the stomach and the brain within minutes. Using non-invasive

techniques to record the stomach and the brain, the researchers were also

able to demonstrate how this coupling effect spreads across sub-cortical and cortical regions of the brain.

The findings could have therapeutic applications, as some mental disorders are characterized by reduced sensory input to the brain.

VOLUME 15, ISSUE 5, P1279-1289, SEPTEMBER 01, 2022

ophie J. Müll

Vagus nerve stimulation increases stomach-brain coupling via a vagal afferent pathway

VAGAL NERVE STIMULATION



We knew that the vagus nerve played a vital role in regulating the speed of the digestive tract. In previous work, we had also shown that non-invasively stimulating vagal afferents at the ear can reduce the frequency of the pacemaker cells of the stomach. These gastric myoelectric signals can be recorded using electrogastrography, a classical method that is conceptually similar to an ECG, but for the stomach. The mechanism that adjusts the speed of the digestive tracts is known as the vago-vagal reflex because it is modulated via the brainstem, a small part of the brain that is directly innervated by vagal afferents.

However, we did not fully understand whether vagal stimulation leads to more widespread effects in subcortical or cortical regions of the brain that are involved in higherorder cognitive functions, including goal-directed behavior.

This delay may lead to long periods without improvements for non-responders to the first line of treatments. Therefore, there is a huge unmet demand for mechanistic treatments that can lead to more rapid improvements in key symptoms of depression. We are currently running two studies that include patients with MDD, where we try to emulate motivational signals from the gut using either administration of ghrelin, a neuropeptide that is synthesized in the stomach, or vagus nerve stimulation. We are positive that the first insights from those studies can be published in late 2023 and hope that this may open new avenues for treatments.

VAGAL NERVE STIMULATION



Shock. 2013 Aug;40(2):144-51. doi: 10.1097/SHK.0b013e318299e9c0.

Vagus nerve stimulation attenuates intestinal epithelial tight junctions disruption in endotoxemic mice through α7 nicotinic acetylcholine receptors.

Author information Abstract

We tested the effect of vagus nerve stimulation in endotoxin-induced intestinal tight junction injury in mice challenged with lipopolysaccharide (LPS) and examined the

role of α 7 nicotinic acetylcholine receptors (α 7nAchR) in this process. Endotoxemia was induced by intraperitoneal injection of LPS (10 mg/kg) in male Balb/c mice. Samples were collected 12 h after LPS treatment. Endotoxemia was associated with intestinal barrier dysfunction, as evidenced by increased amount of fluorescein isothiocyanate-dextran in circulation. Western blot and immunofluorescence was performed, and the results demonstrated decreased expression of occludin and zonula occludens 1 along intestinal epithelium in endotoxemic mice. The ultrastructure of tight junction was disrupted as shown by transmission electron microscopy, which was associated with increased intestinal permeability. Stimulation of the right cervical vagus nerve ameliorated the damage of tight junction ultrastructure, which was consistent with decreased permeability to fluorescein isothiocyanate-dextran, and also reversed the decreased expression of tight junction proteins occludin and zonula occludens 1. Vagus nerve stimulation inhibited the upregulated activity of myosin light chain kinase and nuclear factor kB. In contrast, α -bungarotoxin (a specific α 7nAchR antagonist, 0.1 µg/mouse) administered before vagus nerve

stimulation significantly abolished these protective effects of vagus nerve stimulation. Our results for the first

time confirmed that vagus nerve stimulation attenuated the disruption of tight junction in intestinal epithelium in endotoxemic mice, which was mediated through suppressing translocation of nuclear factor κ B p65, downregulating myosin light chain kinase, and the α 7nAchR may play an important role in this process. Duplication, citation or distribution#0 this material in whole or in part

VAGAL NERVE STIMULATION



Stimulating the Central Nervous System to Prevent Intestinal Dysfunction After **Traumatic Brain Injury**

A. Leaky gut 6 hours after TBI with no vagal stimulation

B. Tight junction integrity 6 hours after TBI with vagal stimulation



Vagus Stimulation + TBI

Stimulating the central nervous system to prevent intestinal dysfunction after traumatic brain injury.

Bansal V, et al. J Trauma. 2010.

Abstract

BACKGROUND: Traumatic brain injury (TBI) causes gastrointestinal dysfunction and increased intestinal permeability. Regulation of the gut barrier may involve the central nervous system. We hypothesize that vagal nerve stimulation prevents an increase in intestinal permeability after TBI.

CONCLUSION: In a mouse model of TBI, vagal stimulation prevented TBIinduced 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



Brain Stimul. 2018 Jul - Aug;11(4):689-698. doi: 10.1016/j.brs.2018.01.034. Epub 2018 Feb 15. Non-invasive vagus nerve stimulation reduces bloodbrain barrier disruption in a rat model of ischemic stroke. 1, ____2, ___2, ___2, ___2, ___2, ___3, ___4.

Life Sci. 2013 Mar 12;92(4-5):289-97. doi: 10.1016/j.lfs.2013.01.009. Epub 2013 Jan 16. Vagus nerve stimulation inhibits seizure activity and protects blood-brain barrier integrity in kindled rats with cortical dysplasia.

Brain Behav Immun. 2019 Oct;81:444-454. doi: 10.1016/j.bbi.2019.06.041. Epub 2019 Jul 2.

Vagus nerve regulates the phagocytic and secretory activity of resident macrophages in the liver.
INTESTINAL PERMEABILITY "LEAKY GUT"





APPROXIMATELY 2 YEARS TO FORM TIGHT JUNCTION BARRIERS AS A NEWBORN **OTHER BIRTH CONSIDERATIONS...** DELIVERY METHOD, BREAST FEEDING, TOXIN/ANTIBIOTIC EXPOSURE

VAGAL NERVE STIMULATION



Front Immunol. 2017; 8: 598. Published online 2017 May 23. doi: 10.3389/fimmu.2017.00598

eaky Gut As a Danger Signal for Autoimmune Diseases

Abstract

The intestinal epithelial lining, together with factors secreted from it, forms a barrier that separates the host from the environment. In pathologic conditions, the permeability of the epithelial lining may be compromised allowing the passage of toxins, antigens, and bacteria in the lumen to enter the blood stream creating a "leaky gut." In individuals with a genetic predisposition, a leaky gut may allow environmental factors to enter the body and trigger the initiation and development

of autoimmune disease. Growing evidence shows that the gut microbiota is important in supporting the epithelial barrier and therefore plays a key role in the regulation of environmental factors that enter the body. Several recent reports have shown that probiotics can reverse the leaky gut by enhancing the production of tight junction proteins; however, additional and longer term studies are still required. Conversely, pathogenic bacteria that can

facilitate a leaky gut and induce autoimmune symptoms can be ameliorated with the use of antibiotic treatment. Therefore, it is

hypothesized that modulating the gut microbiota can serve as a potential method for regulating intestinal permeability and may help to alter the course of autoimmune diseases in susceptible individuals.

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Without permission of Trevor Berry, DC, DACNE is prohibited ALZHEIMER'S ETIOLOGIES



New model of Alzheimer's as an autoimmune disease September 27, 2022

University Health Network

Scientists have proposed a new mechanistic model (AD2) for Alzheimer's, looking at it not as a brain disease, but as a chronic autoimmune condition that attacks the brain.

• The research team conducted a comprehensive search, looking at a wide-ranging review of both journal and patient literatures combined with their own studies, to develop a thorough mechanistic model of Alzheimer's.

• The AD2 model endeavours to harmonize other mechanistic propositions (including proteopathy, synaptotoxicity and mitochondriopathy), while recognizing beta-amyloid as a physiologically oligomerizing immunopeptide and part of a much larger and broad, highly-interconnected immunopathic process.

• Within the AD2 model, amino acid metabolism of L-tryptophan and L-arginine emerge as innate immunity regulators, thus pointing to new diagnostic and therapeutic approaches.

. Donald F. Weaver. Alzheimer's disease as an innate autoimmune disease (AD 2): A new molecular paradigm. *Alzheimer*'s & *Dementia*, 2022; DOI: 10.1002/alz.12789

ALZHEIMER'S AS AN AUTOIMMUNE DISEASE



Abstract

A new model of Alzheimer's disease (AD) is presented: Alzheimer's disease as an autoimmune disease (AD2). In response to pathogen-/damage-associated molecular patternstimulating events (e.g., infection, trauma, ischemia, pollution), amyloid beta (A β) is released as an early responder cytokine triggering an innate immunity cascade in which A β exhibits immunomodulatory/antimicrobial duality. However, A β 's antimicrobial properties result in a misdirected attack upon "self" neurons, arising from the electrophysiological similarities between neurons and bacteria in terms of transmembrane potential gradients and anionic charges on outer membrane macromolecules. *The subsequent breakdown products of necrotic neurons elicit further release of A\beta leading to a chronic, self-perpetuating cycle*. In AD2, amino acid (trp, arg) metabolism is a central control player in modulating AD autoimmunity. AD2 includes A β as an important molecular player, but rejects the "amyloid hypothesis," recognizing A β as a physiologically oligomerizing cytokine and part of a larger immunopathic conceptualization of AD.

Alzheimer's disease as an innate autoimmune disease (AD2): A new molecular paradigm

Donald F. Weaver First published: 27 September 2022 https://doi.org/10.000/12.41090.citation or distribution of distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ALZIEMER'S'AND^{IB is prohibite} AUTOIMMUNITY



Alzheimer Disease Pathogenesis: The Role of Autoimmunity

Bryant Lim, Ioannis Prassas, Eleftherios P Diamandis The Journal of Applied Laboratory Medicine, Volume 6, Issue 3, May 2021, Pages 756–764, https://doi.org/10.1093/jalm/jfaa171 Published: 24 November 2020

We present research suggesting the association between autoimmunity and AD and demonstrate the need for new laboratory techniques to further characterize potential brain antigen-specific autoantibodies. Uncovering the putative autoimmune components of AD may be crucial in paving the way to new concepts for pathogenesis, diagnosis, and therapy.

A compromised blood-brain barrier, which would normally block those autoantibodies from affecting the organ, is seen in Alzheimer's sufferers. Indeed, athletes suffering from serious brain injury, which can disrupt the barrier, are more likely to develop the disease, said Prassas.

The Toronto team's hypothesis is that the autoantibodies cause neuro-inflammation and somehow trigger death of neurons or destruction of the synapses that connect them.

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BACTERIA AND ALZHEIMER'S



Researchers discover vital new contributor to Alzheimer's disease

Jan. 12, 2023: Leslie Capo, Louisiana State University Health Sciences Center]

In this study, the researchers detail the pathway of BF-LPS from the gut to the brain and its mechanisms of action once there. BF-LPS leaks out of the GI tract, crosses the blood brain barrier via the circulatory system, and accesses brain compartments. Then it increases inflammation in brain cells and inhibits neuron-specific neurofilament light (NF-L,) a protein that supports cell integrity.

Front, Neurol., 24 June 2022 Sec. Autonomic Neuroscience https://doi.org/10.388/fneur.2022.00048 This article is part of the Research Topic Gut Microbiota and the Nervous System, Volume II Viewall S Articles BIG 3 FOR LPS: FIX LEAKY GUT LOW LEVEL LASER THERAPY PASSIVE IMMUNOGLOBULIN (IG) THERAPIES

Downregulation of Neurofilament Light Chain Expression in Human Neuronal-Glial Cell Co-Cultures by a Microbiome-Derived Lipopolysaccharide-Induced miRNA-30b-5p

LPS INFLAMMATION AND ALZHEIMER'S



Open Access Published: 07 December 2021

Gram-negative bacteria and their lipopolysaccharides in Alzheimer's disease: pathologic roles and therapeutic implications

 Hyeon soo Kim, Sujin Kim, Soo Jung Shin, Yong Ho Park, Yunkwon Nam, Chae won Kim, Kang won Lee, Sung-Min Kim, In Duk Jung, Hyun Duk Yang, Yeong-Min Park & Minho Moc Translational Neurodegeneration

volume

10, Article number: 49 (2021)

Abstract

Alzheimer's disease (AD) is the most serious age-related neurodegenerative disease and causes destructive and irreversible cognitive decline. Failures in the development of therapeutics targeting amyloid-β (Aβ) and tau, principal proteins inducing pathology in AD, suggest a paradigm shift towards the development of new therapeutic targets. The gram-negative bacteria and lipopolysaccharides (LPS) are attractive new targets for AD treatment. Surprisingly, an altered distribution of gram-negative bacteria and their LPS has been reported in AD patients.

Moreover, gram-negative bacteria and their LPS have been shown to affect a variety of AD-related pathologies, such as Aβ homeostasis, tau pathology, neuroinflammation, and neurodegeneration. Moreover, therapeutic approaches targeting gram-negative bacteria or gramnegative bacterial molecules have significantly alleviated AD-related pathology and comitive dysfunction.

pathology and cognitive dysfunction. Despite multiple evidence showing that the gram-negative bacteria and their LPS play a crucial role in AD pathogenesis, the pathogenic mechanisms of gram-negative bacteria and their

LPS have not been clarified. Here, we summarize the roles and

pathomechanisms of gram-negative bacteria and LPS in AD.

Furthermore, we discuss the possibility of using gram-negative bacteria and gram-negative bacterial molecules as novel therapeutic targets and new pathological characteristics for AD.

VAGAL NERVE STIMULATION



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-kB) 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-kB 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-kB activation. The results showed that 635 nm irradiation led to a decrease in the HSP27 phosphorylation, reactive oxygen species (ROS) generation, I-kB kinase (IKK)/inhibitor of kB (IkB)/NF-kB phosphorylation, NF-kB 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/IkB/NF-kB phosphorylation, NF-kB 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-KB signaling pathway via HSP27 in inflammation. Thus, HSP27 may play a role in regulating the anti-milammatory esponse of LLLT.

VAGAL NERVE STIMULATION



Rheumatoid arthritis linked with gut bacteria imbalance

Impressive new research led by a team from University College London is suggesting bacterial **imbalances in the gut microbiome may play a major role in the development of rheumatoid arthritis.** The preclinical study found damage to the gut lining directly correlates with joint inflammation and arthritis severity.

those suffering from rheumatoid arthritis had *higher blood levels of lipopolysaccharide (LPS), LPS binding protein (LBP), and intestinal fatty acid binding protein. All these molecules are known biomarkers of intestinal damage*, and LBP levels in particular were found to correlate with acute disease severity.

"Our findings suggest that the intestinal lining is a therapeutic target," says Mauri.

CLINICAL AND TRANSLATIONAL ARTICLEI_VOLUME 2, ISSUE 7, P864-883.E9, JULY 09, 2021

Intestinal barrier dysfunction plays an integral role in arthritis pathology and can be targeted to ameliorate disease

Open AccessPublished:May 13, 2021DOI:https://doi.org/10.1016/j.medj.2021.04.013

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VAGAL NERVE STIMULATION



<u>Fasono A</u>, Shea-Donohue T. "Mechanisms of Disease: the role of intestinal barrier function in the pathogenesis of gastrointestinal autoimmune diseases." Nat Clin Prac Gastro & Hep Sept 2005 Vol 2 No 9.

http://www.nature.com/nrgastro/journal/v2/n9/full/ncpgasthep0259.html SUMMARY

The primary functions of the gastrointestinal tract have traditionally been perceived to be limited to the digestion and absorption of nutrients and electrolytes, and to water homeostasis. A more attentive analysis of the anatomic and functional arrangement of the gastrointestinal tract, however, suggests that another extremely important function of this organ is its ability to regulate the trafficking of macromolecules between the environment and the host

THE AUTOIMMUNE PROCESS CAN <u>BE ARRESTED</u> IF THE INTERPLAY BETWEEN GENES AND ENVIRONMENTAL TRIGGERS IS PREVENTED BY RE-ESTABLISHING INTESTINAL BARRIER FUNCTION.

Understanding the role of the intestinal barrier in the pathogenesis of gastrointestinal disease is an area of translational research that encompasses many fields and is

PRIMARY TARGET MODALITY IN ALL AUTOIMMUNE CONDITIONS: FIX INTESTINAL AND BBB PERMEABILITY THROUGH VAGAL NERVE STIMULATION AND OTHER BARRIER REPAIR PROTOCOLS!

Total IgG (mg/dL) 970 Duplication, citation or distribution of this material-jag whole get in the without permission of Trevor Berry, DC, DACNE IIIs previous Total IgA (mg/dL) 124 61-356 560 2357 10 08/25/2018 Test name In Control Moderate High Risk Range In Control 08/25/2018 Moderate High Risk Range Previous Transglutaminase 2 IgG 0.60 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgG 0.25 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgG 0.25 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgG 0.25 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgA 0.18 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgA 0.18 \$0.94 0.95-1.05 ≥1.06 08/25/2018 DGP IgA 0.18 \$0.94 0.95-1.05 ≥1.06 08/25/2018 Transglutaminase 2 IgA 0.30 \$0.89 0.90-1.10 ≥1.11 08/25/2018 DGP IgA </th <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th>									
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Test name In Control Moderate High Risk In Control Range Moderate Range High Risk Range Previous Traneglutaminase 2 lgG 0.60 \$0.94 0.95~1.05 \$1.06 08/25/2018 Traneglutaminase 2 lgA 0.38 \$0.94 0.95~1.05 \$1.06 08/25/2018 DGP lgG 0.25 \$0.94 0.95~1.05 \$1.06 08/25/2018 DGP lgA 0.18 \$0.94 0.95~1.05 \$1.06 08/25/2018 DGP lgA 0.18 \$0.94 0.95~1.05 \$1.06 08/25/2018 Test name In Control Moderate High Risk In Control 0.95~1.05 \$1.06 08/25/2018 TG/DOP Fusion Peptde 0.30 \$0.94 0.95~1.05 \$1.06 08/25/2018 tTG/DOP Fusion Peptde 0.30 \$0.89 0.90~1.10 \$1.11 08/25/2018 tTG/DOP Fusion Peptde 0.20 \$0.89 0.90~1.10 \$1.11 08/25/2018 Tast name In Control Moderate High Risk Range									
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DGP IgG 0.25 \$0.94 0.95~1.05 ≥1.06 08/25/2018 DGP IgA 0.18 \$0.94 0.95~1.05 ≥1.06 1.20 Test name In Control Moderate High Risk In Control Moderate High Risk Previous tTG/DGP Fusion Peptide 0.30 \$0.89 0.90~1.10 ≥1.11 08/25/2018 tTG/DGP Fusion Peptide 0.20 \$0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin (ng/mL) 81.5 \$45.3 45.4~55.3 >55.4 08/25/2018 Anti-Zonulin IgA 0.60 \$0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 \$0.89 0.90~1.10 ≥1.11 0.67 Moderate I	ellad	Tranaglutaminase 2 IgA	0.38			≤0.94	0.95~1.05	≥1.06	2.80 08/25/2018
DGP IgA 0.18 ≤0.94 0.95~1.05 ≥1.06 1.20 08725/2018 Test name In Control IgG Moderate High Risk High Risk In Control Range Moderate Range High Risk Range Previous 1TG/DGP Fusion Peptide IgG 0.30 ≤0.89 0.90~1.10 ≥1.11 0.16 08725/2018 0.16 IgG 0.20 ≤0.89 0.90~1.10 ≥1.11 0.85 08725/2018 Test name In Control Moderate High Risk Range In Control Previous Test name In Control Moderate High Risk In Control Range Moderate Range High Risk Range Previous Zonulin (ng/mL) 81.5 ≤45.3 45.4~55.3 ≥55.4 08/25/2018 Anti-Zonulin IgG 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgG 1.52 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 0.09 08/25/2018 Anti-Actin IgA 0.74 ≤0.89	5	DGP IgG	0.25			≤0.94	0.95~1.05	≥1.06	2.00 08/25/2018
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Test name In Control Moderate High Risk In Control Moderate High Risk Previous tTG/DGP Fusion Peptide IgG 0.30 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 tTG/DGP Fusion Peptide IgA 0.20 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 tTG/DGP Fusion Peptide IgA 0.20 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Test name In Control Moderate High Risk In Control Moderate Range	10								
Tig/DGP Fusion Peptide 0.30 ≤0.89 0.90~1.10 ≥1.11 0.16 (B/25/2018) tTG/DGP Fusion Peptide 0.20 ≤0.89 0.90~1.10 ≥1.11 0.85 (B/25/2018) Test name In Control Moderate High Risk In Control Range Moderate Range High Risk Range Previous Zonulin (ng/mL) 81.5 ≤45.3 45.4~55.3 ≥55.4 08/25/2018 Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018	š	Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
Tig/DGP Fusion Peptide 0.20 ≤0.89 0.90~1.10 ≥1.11 0.85 08/25/2018 Test name In Control Moderate High Riek In Control Moderate High Riek Range High Risk Previous Zonulin (ng/mL) 81.5 ≤45.3 45.4~55.3 ≥55.4 08/25/2018 Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 68/25/2018	Idmo	tTG/DGP Fusion Peptide IgG	0.30			≤0.89	0.90~1.10	≥1.11	0.16 08/25/2018
Test name In Control Moderate High Riek In Control Range Moderate Range High Risk Range Previous Zonulin (ng/mL) 81.5 ≤45.3 45.4~55.3 ≥55.4 08/25/2018 Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgG 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgG 1.52 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 08/25/2018	ŏ	tTG/DGP Fusion Peptide IgA	0.20			≤0.89	0.90~1.10	≥1.11	0.85 08/25/2018
Test name In Control Moderate High Risk Range In Control Range Moderate Range High Risk Range Previous Zonulin (ng/mL) 81.5 ≤45.3 45.4~55.3 ≥55.4 08/25/2018 Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018	1		-						
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Anti-Zonulin IgG 1.14 ≤0.89 0.90~1.10 ≥1.11 0.77 08/25/2018 Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgG 1.52 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 08/25/2018		Zonulin (ng/mL)			81.5	≤45.3	45.4~55.3	≥55.4	<10.0 08/25/2018
Anti-Zonulin IgA 0.60 ≤0.89 0.90~1.10 ≥1.11 0.20 08/25/2018 Anti-Actin IgG 1.52 ≤0.89 0.90~1.10 ≥1.11 0.67 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 0.87 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 08/25/2018		Anti-Zonulin IgG			1.14	≤0.89	0.90~1.10	≥1.11	0.77 08/25/2018
Anti-Actin IgG 1.52 ≤0.89 0.90~1.10 ≥1.11 0.67 08/25/2018 Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 0.09 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 08/25/2018	ভ	Anti-Zonulin IgA	0.60			≤0.89	0.90~1.10	≥1.11	0.20 08/25/2018
Anti-Actin IgA 0.74 ≤0.89 0.90~1.10 ≥1.11 0.09 08/25/2018 Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 08/25/2018	Pan	Anti-Actin IgG			1.52	≤0.89	0.90~1.10	≥1.11	0.67
Anti-LPS IgA (U/ml) 60.0 ≤30.0 ≥30.1 61.0 08/25/2018		Anti-Actin IgA	0.74			≤0.89	0.90~1.10	≥1.11	0.09
Unit with the second seco		Anti-LPS IgA (U/ml)			60.0	≤30.0		≥30.1	61.0 08/25/2018

Management of a "leaky gut"

≤281.0

86.0 08/25/2018

≥281.1

- Identify the sources of inflammation and reactivity and remove them (Wheat and Gut Zoomer, Food Sensitivity Panel, Stress, SAD diet, Antibiotics, Chemical Stressors, AND MOST IMPORTANT; INFECTIOUS AGENTS LIKE BACTERIA, VIRUSES, FUNGI, PARASITES ETC.)
- PHASE 1: Gut repair, liver detox and inflammation reduction. Month 1 Xymogen 6 DAY DETOX KIT. Contains Opticleanse GHI (for GI function and repair, Liver Detox and Inflammation cytokine regulation), Drainage (homeopathic for colon, kidney and liver detox), ColonX (like a colonic without the hassle) and ProbioMax DF (30 Billion CFU including HOWARU Bifido Lactis HN019). Consider 1 -> 3 day fast with water or continue Opticleanse GHI,1 scoop 2-3 x/day. HistDAO 1-4 daily depending on histamine sensitivity. Vitamin A, LIQUID D 7K/DAY. Omega MonoPure 1300 EC (For the duration of care). S-Acetyl Glutathione/NAC (1each daily for duration of care). Probiomax Ig26 DF (2+ Caps/day) once daily formula complete. Optimagneuro 1 scoop or less daily. Oncoplex (2 caps daily). *NEW GI BALANCE, 2 SCOOPS/DAY INITIAL 14 DAYS THEN 1 SCOOP FOR REMAINDER OF 3 MONTHS
- May need other management such as adrenal support/stress reduction, sleep help like Melatonin CR
- CONSIDER MEDPAX FOR PATIENT COMPLIANCE!

Anti-LPS (IgG + IgM) (U/ml) 89.0

- Continue for 3 months minimum. Retest Barrier Systems via Neural and Wheat Zoomers. Retest food sensitivities.
- MUST ADDRESS BRAIN-GUT AXIS! VAGAL STIM. STRESS REDUCTION.



FASTING: INTERMITTENT *16:8", 1 DAY, 3 DAY+

GUT REPAIR: NEW GI BALANCE

Amount Per Serving

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 (<i>Ulmus fulva</i>)(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 (<i>Actinidia deliciosa</i>)(fruit)(62,500 AU actinidin)s1	400 mg	**
Marshmallow (<i>Althaea officinalis</i>)(root)	400 mg	**
Licorice Extract (<i>Glycyrrhiza glabra</i>)(root)(3.5% glabridin)s2	300 mg	**
Quercetin (as quercetin dihydrate)	200 mg	**
Prune (<i>Prunus domestica</i>)(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	**

VAGAL NERVE STIMULATION



%DV

ALTHOUGH A COMPELLING ARGUMENT COULD BE MADE TO PERMANENTLY ELIMINATE A FOOD SOURCE IF YOU HAVE DEVELOPED IGG ANTI-BODIES TO YOU, SOME EXPERTS STATE A PATIENT COULD POTENTIALLY RE-INTRODUCE FOOD IF THE BARRIER SYSTEMS HAVE BEEN REPAIRED, GUT DYSBIOSIS BALANCED AND A HEALTHY BRAIN-GUT AXIS WITH AUTONOMIC BALANCE

> FOOD ELIMINATION PROTOCOL FOR RE-INTRODUCTION: PERMANENTLY STAY OFF THE FOOD SOURCE OR



AFTER 3 MONTHS OF GUT REPAIR PROTOCOLS AND 3 MONTHS MAINTENANCE OF STRICT FOOD ELIMINATION CONFIRM VAGAL/PARASYMPATHETIC BALANCE (HRV, BP, BPI, PALATAL TONE ETC) PICK FOODS THAT WANT TO BE RE-INTRODUCED (BACON!) CONSUME AND MARK THE DATE RE-RUN FOOD SENSITIVITY TEST 12+ DAYS LATER

VIBRANTS FOOD SENSITIVITY AND ADVANCED FOOD ZOOMER TESTS without permission of Trevor Berry, DC, DACNB is prohibited.

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insglutaminase Panel	Test name	In Control	vi llooertup e	r Migbstich	o r Freyo r	Benygebo	HIACNB	i Brptoh ibi
	Transglutaminase 3 IgG			1.53	≤0.89	0.90~1.10	≥1.11	0.63 08/25/2018
	Transglutaminase 3 IgA		1.02		≤0.89	0.90~1.10	≥1.11	0.42 08/25/2018
	Transglutaminase 6 IgG			1.15	≤0.89	0.90~1.10	≥1.11	0.29 08/25/2018
Ĕ	Transglutaminase 6 IgA	0.66			≤0.89	0.90~1.10	≥1.11	0.23 08/25/2018



Wheat Germ Panel	Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
	Wheat Germ Agglutinin IgG			1.32	≤0.89	0.90~1.10	≥1.11	0.57 08/25/2018
	Wheat Germ Agglutinin IgA		1.02		≤0.89	0.90~1.10	≥1.11	0.21 08/25/2018

	Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
	Alpha Gliadin IgG			1.72	≤0.89	0.90~1.10	≥1.11	0.79 08/25/2018
	Alpha Gliadin IgA			1.27	≤0.89	0.90~1.10	≥1.11	0.39 08/25/2018
	Alpha-Beta Gliadin IgG			1.32	≤0.89	0.90~1.10	≥1.11	0.40 08/25/2018
	Alpha-Beta Gliadin IgA		1.02		≤0.89	0.90~1.10	≥1.11	0.97 08/25/2018
anel	Gamma Gliadin IgG			1.67	≤0.89	0.90~1.10	≥1.11	0.41 08/25/2018
lin P	Gamma Gliadin IgA			1.42	≤0.89	0.90~1.10	≥1.11	0.34 08/25/2018
Gliac	Omega Gliadin IgG			1.30	≤0.89	0.90~1.10	≥1.11	0.06 08/25/2018
	Omega Gliadin IgA		1.07		≤0.89	0.90~1.10	≥1.11	0.62 08/25/2018
	Gluteomorphin IgG			1.43	≤0.89	0.90~1.10	≥1.11	0.77 08/25/2018
	Gluteomorphin IgA		1.09		≤0.89	0.90~1.10	≥1.11	0.43 08/25/2018
	Prodynorphin IgG			1.48	≤0.89	0.90~1.10	≥1.11	0.12 08/25/2018
	Prodynorphin IgA			1.17	≤0.89	0.90~1.10	≥1.11	0.02 08/25/2018

Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
Wheat Allergen IgE (kU/L)	⊲0.10			≤0.34	0.35~3.49	≥3.50	<0.10 08/25/2018

_	Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
Iutenin Pane	HMW Glutenin IgG	0.68			≤0.89	0.90~1.10	≥1.11	0.90 08/25/2018
	HMW Glutenin IgA	0.69			≤0.89	0.90~1.10	≥1.11	0.25 08/25/2018
	LMW Glutenin IgG			1.50	≤0.89	0.90~1.10	≥1.11	0.32 08/25/2018
Ö	LMW Glutenin IgA			1.15	≤0.89	0.90~1.10	≥1.11	0.86 08/25/2018

	Test name	In Control	Moderate	High Risk	In Control Range	Moderate Range	High Risk Range	Previous
	Serpin IgG			1.30	≤0.89	0.90~1.10	≥1.11	0.87 08/25/2018
_	Serpin IgA		1.02		≤0.89	0.90~1.10	≥1.11	0.36 08/25/2018
Pane	Farinins IgG			1.23	≤0.89	0.90~1.10	≥1.11	0.18 08/25/2018
Wheat F	Farinins IgA		0.93		≤0.89	0.90~1.10	≥1.11	0.37 08/25/2018
	Amylase/Protease Inhibitors IgG			1.25	≤0.89	0.90~1.10	≥1.11	0.01 08/25/2018
lute	Amylase/Protease Inhibitors IgA		0.94		≤0.89	0.90~1.10	≥1.11	0.99 08/25/2018
on-G	Globulins IgG			1.30	≤0.89	0.90~1.10	≥1.11	0.58 08/25/2018
ž	Globulins IgA		0.98		≤0.89	0.90~1.10	≥1.11	0.48 08/25/2018
	Purinin IgG			1.40	≤0.89	0.90~1.10	≥1.11	0.96 08/25/2018
	Purinin IgA		1.00		≤0.89	0.90~1.10	≥1.11	0.87 08/25/2018



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LEAKY BRAIN AND ALZHEIMER'S



New hypothesis argues leaky blood brain barrier triggers Alzheimer's

"A clear conclusion to be drawn from the model is that the primary focus in prevention and early treatment of AD should be on protecting, and if possible, repairing the BBB," concluded Rudge. "Diagnosing BBB disruption at the earliest stage is critical to fighting AD, certainly in the case of LOAD [late-onset

Alzheimer's disease], since relying on other signs, in the form of mild cognitive impairment, plaques, or NFTs, is quite likely, in most cases, to result in treatment starting too late to prevent further serious cognitive decline."

A New Hypothesis for Alzheimer's Disease: The Lipid Invasion Model

Journal: Journal of Alzheimer's Disease Reports, vol. 6, no. 1, pp. 129-161, 2022 Received 1 October without performance of distribution of di



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, H2O2 and lipid peroxidation) and immunity (lysozyme,

melatonin and endorphin) in gold-striped amberjack, *Seriola Ialandi*. 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-Goup Keang Rock of Tais Abar Stranger and Stranger Strange

MOTOR NEURON DISEASE



'Abnormal' Protein Could Be Common Link Between All Forms of Motor Neuron Disease

FeaturedGeneticsNeurologyNeuroscience·August 13, 2022

Researchers have found an abnormal protein usually linked to a rare inherited form of motor neuron disease is present in all types of motor neuron disease, suggesting a common link between the different forms of the disease.

Normally, the protein superoxide dismutase 1 (SOD1) protects cells, but a mutation in its gene is thought to make the protein "toxic"; this toxic protein form is associated with hereditary forms of ALS. Abnormal mutant SOD1 is only found in regions of the spinal cord where nerve cells die, implicating this abnormal protein in cell death.

GREEN LIGHT AND IMMUNITY



We used green and red LED exposure at two intensities (0.3 and 0.5W/m(2)) for 1, 3, and 5 days. We measured liver mRNA expression and plasma levels of antioxidant enzyme superoxide dismutase (SOD) and caspase-3. Furthermore, we measured plasma levels of hydrogen peroxide (H2O2), lipid peroxidation (LPO), melatonin, and immunoglobulin M (IgM). DNA damage and apoptotic activity were measured

However, we found that exposure to green LED light effectively reduced the detrimental effects induced by BPA, including decreasing DNA damage, apoptotic activity, SOD mRNA expression, and plasma levels of SOD, H2O2, and LPO. Likewise, the plasma levels of melatonin and IgM increased. Thus, our results indicate that green light conditions effectively reduces oxidative stress and promotes the immune function in juvenile rock bream.

Environ Toxicol Pharmacol 2016 Jul;45:140-9. doi: 10.1016/j.etap.2016.05.026. Epub 2016 May 31.

Effects of various LED light spectra on antioxidant and immune response in juvenile rock bream, Oplegnathus fasciatus exposed to bisphenol A

Duplication, citation or distributioned in material in whole or in part Ji Yong Choi 1, Tae Hwan Kim 1, Young Witle Other Mission Kim Tregoring Provided by the source of the source of

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"LEAKY BRAIN"

ZOOMER PLUS



Blood Brain Barrier Disruption		Blood Brain Barrier Disruption					
Anti-s100b		Anti-s100b					
		Anti-Glial fibrillary acidic protein	Anti-Microglia				
		Anti-Glucose regulated protein 78					

ROOT CAUSES:

- INFLAMMATION FROM FOOD INTOLERANCES

- HIGH BLOOD SUGAR - OBESITY AND HIGH CALORIE DIETS

- HIGH HOMOCYSTEINE. - LEAKY GUT

- STRESS AND POOR SLEEP. INFECTIONS AND TOXINS
- NMDA EXCITOXICITY/GLUTAMATE FOODS

- POOR CEREBRAL CIRCULATION. - GLUTEN (WGA)

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- EMF EXPOSURE - ENVIRONMENTAL TOXINS - FLUORIDATED WATER

Positive		Moderate								
(IgG + IgA)	lgM	(IgG + IgA)	lgM		iveg	auve	Vibro	170		
	Blood Brain Barrier Disruption	Demyelination Antigens	Demyelination Antigens	In children of the sec	Demyelinat	vibra	ntAmerica			
	Anti-s100b Peripheral Neuropathy Anti-GM1	Anti-Tubulin Blood Brain Barrier Disruption Anti-s100b Anti-Glucose regulated protein 78 Brain Autoimmunity Anti-Amyloid beta (25-35)	Anti-Tubulin Blood Brain Barrier Disruption Anti-s100b	Anti-Tubulin Blood Brain Barrier Disruption Anti-s100b	Anti-Tubulin Anti-Myelin Blood Brain proteolipid parrier Disruption Anti-Other Anti-Myelin proteolipid protein Neuroparadar	Anti-Myelin basic protein	oligodendrocyte glycoprotein Blood Brain Ba	Anti-Neurofascin	Anti-MAG	NEURAL ZOOMER
	Neuromuscular disorders Anti-Voltage gated potassium		disorders n Anti- Acetylcholine receptors	Anti-Glial fibrillary acidic protein Optic	Anti-Microglia al and Autonomic	order	PLUS			
	channels		Brain Autoimmunity Anti-RAGE	Anti-Neuron specific enolase	Anti-Aquaporin4	Anti-Recoverin	Anti-CV2			
THE			0.4		Peripheral	Neuropatny	Anti			
IHE	NZ+ CAN E	SE USED A	IS A	Anti-GM2	Anti-Hu	Anti-Ri	Amphiphysin			
COI	NFIRMATIO	N TEST O	A F		Neuromuscu					
PREDICTI BEST	VE MODEL	CLINICA AN UPSTR	LLY, IT IS EAM	Anti-Muscle specific kinase	Anti-Voltage gated calcium channels	Anti-Titin				
		E END ST	CE		Brain Auto	oimmunity				
				Anti-HSV1	Anti-Cerebellum	Anti-Purkinje cell	Anti-Yo			
INFLAM	MATION AF		IMUNE	beta (1-42) Anti- Hydroxytryptami ne Anti-NMDA recentor	Anti-Tau	Anti-Glutamate	Anti-Dopamine			
TRIG	GERS. IF I	SEE POSI	TIVE		Anti-Alpha-	adrenergic	Anti-Endothelin			
	IGS I STAR	T TO LOOP	(FOR		synuclein	receptors	A receptor			
					Brain Infl	ammation				
AND PAT	HOGENS C	OR OTHER	TOXINS	Anti-AMPA receptor	Anti-Dopamine receptor 1	Anti-Dopamine receptor 2	Anti-GABA receptors			
TRIGGE	RING MOLE	ECULAR M	IMICRY	Anti-Dipeptidyl aminopeptidase like protein 6	Anti-Glycine receptor	Anti-Neurexin 3	Anti-Contactin- Associated Protein-like 2			
ANI	D OFTEN PI	RECEDED	BY	Anti-Leucine-			Antibodies			
DISRUPTI	DISRUPTION OF THE GUT AND BLOOD-			rich glioma- inactivated	Anti-Ma					
BRAIN	BARRIERS!	NORMAL	LY THE	protein 1 (Anti- LGI1)						
BRAIN HAS THE LUXURY OF SITTING										
BEHIND	A PROTEC	TIVE VAUL	T FREE	Anti-HSV2	Anti-EBV	Anti-CMV	Anti-HHV 6			
OF IMM	IUNOLOGI	CAL REAC	JWITY ission	n of Trevor Berr	y, DC, DACNE	is prohibited.	ai t			

Demyelination Antigens	with	মিনিস্elinatissientiজন্য revor Berry, I	DC, DACNB is prohi				
			· ·				
Anti-Tubulin	Anti-Myelin basic protein	Anti-Tubulin	Anti-Myelin basic protein				
		Anti-Myelin oligodendrocyte glycoprotein	Anti-Myelin proteolipid protein				
		Anti-MAG	Anti-Neurofascin				
Blood Brain Barrier Disrup	otion	Blood Brain Barrier Disruption					
Anti-s100b		Anti-s100b					
		Anti-Glial fibrillary acidic protein	Anti-Microglia				
		Anti-Glucose regulated protein 78					
Optical and Autonomic ne disorder	rvous system	Optical and Autonomic nervous system d	isorder				
Anti-Neuron specific enolase		Anti-Neuron specific enolase					
		Anti-Aquaporin4	Anti-Recoverin				
		Anti-CV2					
Peripheral Neuropathy		Peripheral Neuropathy					
Anti-GM1	Anti-GM2	Anti-GM1	Anti-GM2				
		Anti-Hu	Anti-Ri				
		Anti-Amphiphysin					
Neuromuscular disorders	- I	Neuromuscular disorders					
		Anti-Acetylcholine receptors	Anti-Muscle specific kinase				
		Anti-Voltage gated calcium channels (Anti- VGCC)	Anti-Voltage gated potassium channels				
		Anti-Titin					
Brain Autoimmunity		Brain Autoimmunity					
Anti- HSV1	Anti- Cerebellum	Anti- HSV1	Anti-Cerebellum				
Anti- Purkinje cell		Anti- Purkinje cell					
		Anti-Yo	Anti-Amyloid beta (25-35) (Alzheimer's)				



NEURAL **ZOOMER PLUS**

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	Anti-Amyloid beta (Alzheimer's)	Anti-RAGE peptide (Alzheimer's)
	Anti-Tau (Alzheimer's)	Anti-Glutamate
	Anti-Dopamine (Parkinson's)	Anti-Hydroxytryptamine (Anti-5-HT)
	Anti-Alpha-synuclein (Alzheimer's and (Parkinson's))	Anti-α1 and β2 adrenergic receptors (Alzheimer's)
	Anti-Endothelin A receptor	
Brain Inflammation	Brain Inflammation	
	Anti-NMDA receptor	Anti-AMPA receptor
	Anti-Dopamine receptors	Anti-GABA receptors
	Anti-Dipeptidyl aminopeptidase like protein 6	Anti-Glycine receptor
	Anti-Neurexin 3	Anti-Contactin-Associated Protein-like 2 Antibodies (CNTNAP2)
	Anti-Leucine-rich glioma-inactivated protein 1 (Anti-LGI1)	Anti-Ma
Neural Genetics	Neural Genetics	
Apolipoprotein E	Apolipoprotein E	



NEURAL ZOOMER PLUS

Clinical Value

Demyelination Antigens The upgraded Neural Zoomer plus contains four extra antigens that provide clinical value in identifying both common and the upgraded Neural Zoomer plus contains four extra antigens that provide clinical value in identifying both common and consistently present in The upgraded Neural Zoomer plus contains four extra antigens that provide clinical value in identifying both common and rare demyelinating diseases. Anti-Myelin oligodendrocyte glycoprotein (anti-MGG) has been found consistently present in many acquired demyelinating CNS syndromes with predominance in pediatric patients'. Some patients tested negative for anti-myelin basic protein (anti-MBP), the most frequently used marker for multiple sclerosis (MS) and optic neuritis, showed positive for anti-myelin proteolipid protein (anti-PLP). Hence, anti-PLP adds value in identifying MS patients that would have neglected if only tested for anti-MBP. Anti-neurofascin is clinically important in identifying condition, combined central and peripheral demyelination (CCPD), and early detection is important in identifying of a rare disease called anti-MADe pripheral neuropathy that has slow progression. Hence, detection of this biomarker helps identifying the best treatment strategies such as simple exercises if the condition is mild or drug therapies if the disease is severe.

severe. Blood Brain Barrier (BBB) Disruption

Blood Brain Barrier (BBB) Disruption The new markers of BBB disruption provide more sensitivity towards identification of possible BBB disruptions and associated conditions. The presence of Anti-s100b, anti-glial fibrillary acidic protein autoantibodies, anti-microglia, glucose-regulated protein 78-targeted antibodies reflect BBB disruption that may allow neuroinflammation. The symptoms of BBB disruption may vary from migraines, disziness, brain fog, sleep disturbances, fatigue, anxiety, and depression to more complex neuroinflammatory disorders. Presence of these markers indicates brain inflammation and oxidative stress that can complex neuroinitalimatory disorders. Presence of these markers indicates brain initialimation and oxidative stress that can be treated by treating leaky gut, reduce alcohol use, and sugar imbalances or identifying triggers including mycotoxins (mold), toxins from the environment, such as chemicals and metals, and co-infections like Lyme disease etc., treatments with simple BBs sealers and antioxidant drug therapies. Glucose-regulated protein 78-targeted antibodies could trigger blood-brain barrier breakdown and development of hallmark anti-aquaporin-4 autoantibody in Neuromyelitis optica (NWO) pathology, hence the presence of anti-GRP 78 autoantibodies can be helpful in targeted treatments.²

pathology, hence the presence of anti-GRP 78 autoantibules can be received and autonomic nervous system disorder Optical and Autonomic nervous system disorder Neural Zoomer plus contains three extra optical and autonomic nervous system disorder markers that has added clinical values. Anti-Aquaporin4 is a well-established optication optication of the streated optication optication of the streated optication opt

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BLOOD BRAIN BARRIER



ERCHONIA RED LASER BASE BRAIN 1-10-40-60. 12+ VISITS TRANSCRANIAL BASE PROTOCOLS (UP TO 12 MINUTES HAND HELD/10 MINUTES FX RED, 6 MIN VIOLET AND GVL)

VAGAL STIMULATION ERCHONIA LASER (10 HZ) 2 MINUTES SCM/2 MINUTES GUT/PERCUSSOR (UP TO 10 MINUTES)

XYMOGEN SUPPORT (3 MONTH MINIMUM):

- MEMORALL 1 DAILY
- GASTRACID 1 DAILY (BEFORE PROTEIN MEALS)
- OPTIMAG NEURO 1 SCOOP DAILY
- COQMAX OMEGA 100 MG 2 DAILY

90 DAYS GI BALANCE

ONCOPLEX ES/CURCUPLEX -95 1 DAILY

OR NRF2 ACTIVATOR 1 DAILY

DIOVASC 1 DAILY

- SUPPORT IF LAB INDICATES: VIT D3, B ACTIV (HOMOCYSTEINE ELEVATION), ALAMAX CR (HBAIC ELEVATION/DIABETIC), MELATONIN CR (IF POOR SLEEP)
- LAB TEST CONFIRMATION VIBRANT NEURAL ZOOMER PLUS. ALSO CONSIDER IF ANTI-ZONULIN ANTIBODIES ON WHEATER ZOOMER/INTESTINAL PERMEABILITY
- ADDRESS ROOT CAUSES ...

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MARKERS FOR LEAKY BARRIERS REVIEW



GUT:

- ZONULIN -ANTI-ZONULIN ANTIBODIES -ANTI-ACTIN ANTIBODIES -ANTI-LIPOPOLYSACCHARIDE (LPS) ANTIBODIES WHEAT ZOOMER/INTESTINAL PERMEABILITY PANEL

BRAIN:

-ZONULIN (NOT ON NZ+) -ANTI-ZONULIN ANTIBODIES (NOT ON NZ+) -ANTI-GFAP (GLIAL FIBRILLARY ACID PROTEIN) ANTIBODIES -ANTI-MICROGLIA -ANTI-GLUCOSE REGULATED PROTEIN 78 NEURAL ZOOMER PLUS

VAGAL NERVE STIMULATION



HOW DO WE ACCESS THE VAGUS NERVE?

VAGAL NERVE STIMULATION



Brain Stimul. 2017 Jan - Feb;10(1):19-27. doi: 10.1016/j.brs.2016.10.008. Epub 2016 Oct 20.

Access to Vagal Projections via Cutaneous Electrical Stimulation of the Neck: fMRI Evidence in Healthy Humans.

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Conclusion:

The present findings provide evidence in humans that cervical vagal afferents can be accessed non-invasively via transcutaneous stimulation of the anterolateral surface of the neck (SCM), which overlies the course of the nerve, suggesting an alternative feasible method



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VAGAL NERVE STIMULATION



RCHONIA

ASSESSING THE AUTONOMIC EFFECT OF VAGAL NERVE STIMULATION WITH LOW LEVEL LASERS BY HEART RATE VARIABILITY MACHADO ET AL.

ABSTRACT: VAGUS NERVE STIMULATION (VNS) HAS BEEN APPROVED TO TREAT REFRACTORY EPILEPSY, AND FOR OTHER CONDITIONS. THE INVASIVE NATURE OF THE ELECTRICAL STIMULUS, WHICH REQUIRES SURGICAL IMPLANTATION OF ELECTRODES AROUND THE CERVICAL VAGUS NERVE, IS A TECHNICAL LIMITATION. FIFTEEN NORMAL PARTICIPANTS FROM 22 TO 46 YEARS DIVIDED INTO THREE GROUPS OF 5 SUBJECTS EACH, PAIRED IN AGE AND GENDER, WERE STUDIED APPLYING VNS USING LLLT BY LASERS OF DIFFERENT FREQUENCIES; RED LASER (5 SUBJECTS), VIOLET LASER (5 SUBJECTS) AND RED-VIOLET COMBO (5 SUBJECTS). THE STUDY INCLUDED 3 EXPERIMENTAL CONDITIONS; BASAL RECORD (10 MIN), VNS (10 MIN), AND POST-VNS (10 MIN). THE LF/HF RATIO WAS CONSIDERED BECAUSE IT PROVIDES A MEASUREMENT OF PARA-SYMPATHETIC BALANCE. 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.**

INSPIRATION IS SYMPATHETIC TONE, EXPIRATION PARASYMPATHETIC (1:2) ACH BATH ON VAGAL TONE. COMA PROGNOSIS/THERAPEUTIC WINDOW... 177

VAGAL NERVE STIMULATION

SUBJECT 10



QEEG RESULTS USING LLLT WITH RED LASER

SUBJECT O1



When stimulating with **RED** LASER, in 7 cases

brain activity increased progressively from BASAL, to VAGAL STIMULATION, and POST-VAGAL STIMULATION records. In the 3 remaining cases, we found a similar pattern as when the **BLUE**

(VIOLET) LASER was applied, a

progressive reduction brain

activity from BASAL, to VAGAL STIMULATION, and POST-VAGAL STIMULATION records.

CALIXTO MACHADO, INSTITUTE OF NEUROLOGY AND NEUROSURGERY

VAGAL NERVE STANDLATION **10 HZ FREQUENCY**



Long Term Effects of Low Frequency (10 Hz) Vagus Nerve Stimulation on EEG and Heart Rate Variability in Crohn's Disease: A Case Report

November 28, 2000: 55 (10) EXPEDITED PUBLICATION

Effects of vagus nerve stimulation on respiration during sleep

A pilot study

B.A. Malow, J. Edwards, M. Marzec, O. Sagher and G. Fromes

Autonomic Regulation Therapy via Left or Right Cervical Vagus Nerve Stimulation in Patients With Chronic Heart Failure: Results of the ANTHEM-HF Trial Author links open overlay panel Rajendra K.Premchand/MDiKamalSharmaMD2SanjayMittalMD3RufinoMonteiroMD4SatyajitDixitMD2ImadLibbusPhD5Lorenzo A.DiCarloMD6Jeffrey

LArdellPhD7Thomas S.RectorPharmD, PhD8BadriAmurthurMS5Bruce H.KenKnightPhD5Inder S.AnandMD, DPhil (Oxon), FRCP8

Vagus nerve stimulation inhibits cytokine production and attenuates disease severity in rheumatoid arthritis

Chronic vagus nerve stimulation reduces body fat, blood cholesterol and triglyceride levels in rats fed a high-fat diet

Article (PDF Available) in Folia medica Cracovie HOME IDEAS FOR STIMULATING VAGAL NERVE/ POTENTIAL VAGAL/PARASYMPATHETIC POINTS FOR LLLT: PARASYMPATHETIC: 4:4:8 BREATHING - GARGLE - GAG - MEDITATION - COLD TRANSCRANIAL AURICULAR SHOWERS YAW AXIS ROTATIONS - NO NO'S - SINGING - HUMMING SCM (ADD RIGHT CAROTID REFLEX) THYMUS TAPPING - LAUGHTER - AVOID FIGHT OR GI SYSTEM/EPIGRASTRIC FLIGHT **ILIOCECAL VALVE (SIBO)** *CO-ACTIVATE WITH OTHER VAGAL TONE STIMULATION **PURCHASE AN ERCHONIA LASER**

VAGAL NERVE STIMULATION: MASTER PROTOCOL



VAGAL NERVE STIMULATION USING ERCHONIA LASERS:

CONSIDER "VAGUS" SETTING 10-10-10-10 OR "MASTER BRAIN" 1-10-40-60 OR "REST-DIGEST" 1-10-1-10

MINIMUM 2 MINUTES OVER THE SCM AND **2 MINUTES OVER THE GUT** STACK WITH YOUR PERCUSSOR

UP TO 10 MINUTES TOTAL CONSIDER PREFRONTAL CORTEX THEN BASE OF SKULL/MEDULLA THEN OVER THE EAR/TRAGUS REGION THEN SCM THEN OVER THE GUT RULE OF 2'S



RIGHT VERSUS LEFT? BOWEL SOUNDS/ MOVEMENTS, LUBRICATION/ED ASSESS - BILATERAL BP, PULSE OX/BPI, PALATAL PARESIS, CAPILLARY REFILL ETC **CLINICAL CONSIDERATIONS -CONTRALATERAL CORTICAL +,**

ALWAYS MONITOR AUTONOMICS PULSE OX WITH BLOOR REFELICION INPEX of % of this material in whole or in PSILATERAL - without permission of Trevor Berry, DC, DACNB is prohibited.



ERCHONIA

THE VAGAL NERVE STACK:

- 1. ERCHONIA LLLT TO THE SCM AND GUT WITH ANY OF THE 10 HZ SETTINGS. MINIMUM 2 MINUTES EACH (DID 10 MINUTES IN HRV STUDY)
- 2. PERCUSSOR GI. 4 LAPS AROUND THE GUT IN A CLOCKWISE FASHION. FINISH OVER THE UMBILICUS

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- 3. HOOLEST PRIME TO SCM (#3 SETTING)
- 4. REZZIMAX TO HEAD/THYMUS/VAGUS
- 5. BOX BREATHING/RELAXATION MUSIC

VAGAL NERVE STIMULATION PROTOCOL



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VAGAL NERVE STIMULATION



REZZIMAX TUNER PRO



ERCHONIA PERCUSSOR AND LASER







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VAGAL NERVE STIMULATION





CLINICAL GEM: WHILE LASERING THE VAGUS NERVE AT THE SCM STACK IT WITH "NO-NO" VOR'S, 4:4:8 BREATHING, TONGUE TOOTH COUNT HUM, GI STIMULATION (PERCUSSOR)

THIS CAN BE EFFECTIVE IN ALL VAGAL NERVE STIMULATION SCENARIOS AND SUPRASEGMENTAL PAIN INHIBITION (RAPHE NUCLEI)

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GUT HEALTH



ERCHONIA



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GI ISSUES AND SPENDING



Each Dollar Spent on OTC Products Saves the U.S. ~ \$7.33

Healthcare System Value \$ Saved Per \$ Spent on OTC By Category

Category	Savings Size (Bil.)	% of Savings	Annual OTC Spend (Bil.)	\$ Saved Per \$ Spent on OTC (2022)	\$ Saved Per \$ Spent on OTC (2018)
Allergy	\$20.6	12.3%	\$3.9	5.3	5.4
Analgesics	\$29.9	17.9%	\$4.3	6.9	7.4
Antifungals	\$10.8	6.5%	\$0.7	14.6	11.4
Cough / Cold / Flu	\$21.8	13.0%	\$5.1	4.3	4.7
Lower GI	\$18.3	11.0%	\$2.7	6.8	7.2
Medicated Skin	\$28.8	17.2%	\$1.5	19.3	19.7
Upper GI	\$21.0	12.6%	\$2.6	8.1	7.3
Sleep	\$14.4	8.6%	\$0.9	15.9	12.6
Smoking Cessation	\$1.4	0.8%	\$1.1	1.3	1.5
Total	\$167.1	100%	\$22.8	7.33	7.2



Source: IRINGP Consume Survey, CHPA OTC Value Study, Oct 2018 & July 2022; IRI Market Advantage POS Data, Symphony Health Pharmaceulcal Audis, Fair Health.Org/Internet Literature Resear HealthSystemTracker, PwC. Puplication, citation or distribution of this material in whole or in part 2022 Value of OTCs Study 3.3 without permission of Trevor Berry, DC, DACNB is prohibited.

GUT MICROBIOME AND ALZHEIMER'S



HOME<u>HEALTH NEWS</u>

Research Shows Gut Bacteria Affect Brain Health – Reveals New Approach to Treating Alzheimer's Disease

"We gave young mice antibiotics for just a week, and we saw a permanent change in their gut microbiomes, their immune responses, and how much neurodegeneration related to a protein called tau they experienced with age," said senior author David M. Holtzman, MD

"This study may offer important insights into how the microbiome influences tau-mediated neurodegeneration, and suggests therapies that alter gut microbes may affect the onset or progression of neurodegenerative disorders," said Linda McGavern, PhD, program director at the National Institute of Neurological Disorders and Stroke (NINDS), which provided some of the funding for the study.

The findings suggest a new approach to preventing and treating neurodegenerative diseases by modifying the gut microbiome with antibiotics, probiotics, specialized diets or other means.

Reference: "ApoE isoform– and microbiota-dependent progression of neurodegeneration in a mouse model of tauopathy" by Dong-oh Seo, David O'Donnell, Nimansha Jain, **13 January 2023**, *Science*. DOI: 10.1126/science.add1236

UPDATES ON GUT HEALTH



Gut Microbiome at the Center of Parkinson's Disease

Pathogenesis

Investigators found an overabundance of opportunistic pathogens and immunogenic components, which suggest infection and inflammation at play, overproduction of toxic molecules, and overabundance of the bacterial product curli. This induces PD pathology and dysregulation of neurotransmitters, including L-dopa. At the same time, there was a shortage of neuroprotective molecules and anti-inflammatory components, which makes recovery difficult.

The researchers studied 257 species of organisms in the microbiome, and of these, analysis indicated 84, more than 30%, were associated with Parkinson's disease.

"Of the 84 PD-associated species, 55 had abnormally high abundance in persons with PD, and 29 were depleted," Payami said. "We found that over 30% of the micro-organisms and bacterial genes and pathways tested have altered abundances in Parkinson's disease. ORAL BASED SPECIES! ALL CAN BE IMPROVED BY LLLT

At one end of the spectrum, **Bifidobacterium dentium was elevated by sevenfold**, **Actinomyces oris by 6.5-fold and Streptococcus mutans by sixfold**. At the other end of the spectrum, Roseburia intestinalis was reduced by 7.5-fold and Blautia wexlerae by fivefold. Overall, 36% of PD-associated species had higher than twofold change in abundance, reflecting a 100% to 750% increase or decrease in PD versus the healthy control group.

Published: 15 November 2022

Metagenomics of Parkinson's disease implicates the gut microbiome in multiple disease mechanisms

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Zachary D. Wallen, Ayse Demirkan, Guy TwaWithouts permission of Trevor, Berry, DGrdDAGNBristprohibited. Haydeh Payami

GUT HEALTH: PBM ON THE GUT AND ALZHEIMER'S SIGNS

An abdominal irradiation targeting gut flora was designed for Alzheimer's disease.

Gut flora-targeted photobiomodulation therapy at 630 nm and 730 nm improved AD.

Increase of Helicobacter and decrease of Rikenella in AD were reversed by gf-PBM.

Abstract

Methods

PBM was performed on the abdomen of the mice at the **wavelengths of 630 nm, 730 nm, and 850 nm at 100 J/cm₂ for 8 weeks**. Morris <u>water maze</u> test, immunofluorescence and <u>proteomic</u> of <u>hippocampus</u>, and intestinal flora detection of fecal were used to evaluate the treatment effects of gf-targeted PBM on AD rats.

Results

PBM at all three wavelengths (especially 630 nm and 730 nm) significantly **improved learning retention** as measured by the Morris water maze. **In addition, we found reduced amyloidosis** and tau phosphorylation in

the hippocampus by immunofluorescence in AD mice. By using a <u>quantitative proteomic</u> analysis of the hippocampus, we found that gf-targeted **PBM significantly altered the expression levels of 509 proteins** (the same differentially expressed proteins in all three wavelengths of PBM), which **involved the pathways of <u>hormone</u>**

synthesis, phagocytosis, and metabolism. The 16 s <u>rRNA</u> gene sequencing of fecal contents showed that PBM significantly altered the diversity and abundance of intestinal flora. Specifically, **PBM treatment**

reversed the typical increase of <u>Helicobacter</u> and uncultured Bacteroidales and the decrease of Rikenella seen in AD mice.

ConclusionsOur data indicate that gf-targeted PBM regulates the diversity of intestinal flora, which may improve damage caused by AD. *Gf-targeted PBM has the potential to be a noninvasive <u>microflora</u> regulation method for AD patients.*

Journal of Photochemistry and Photobiology B: Biology

Volume 216, March 2021, 112152

Gut flora-targeted photobiomodulation therapy improves senile dementia in an Aβ-induced Alzheimer's disease animal modeL

GUT HEALTH



ERCHONIA

A Protective Probiotic for ALS Found

FeaturedNeurologyNeuroscience December 20, 2022

Summary: Lacticaseibacillus rhamnosus HA-114, a non-commercial probiotic reduces neurodegeneration and has neuroprotective effects in lab models of ALS.

Source: University of Montreal

APPLY ALL 3 ERCHONIA WAVELENGTHS!

A probiotic bacterium called Lacticaseibacillus rhamnosus HA-114 prevents neurodegeneration in the C. elegans worm, an animal model used to study amyotrophic lateral sclerosis (ALS).

That's the finding of a new study at Canada's CHUM Research Center (CRCHUM) led by Université de Montréal neuroscience professor Alex Parker and published in the journal *Communications Biology*.

He and his team suggest that the disruption of lipid metabolism contributes to this cerebral degeneration, and show that the neuroprotection provided by HA-114, a non-commercial probiotic, is unique compared to other strains of the same bacterial family tested.

"When we add it to the diet of our animal model, we notice that it suppresses the progression of motor neuron degeneration," said Parker, the study's lead author. "The particularity of HA-114 resides in its fatty acid content."

CLINICAL CONSIDERATION: HIGH DOSE L-SERINE SUPPLEMENTATION (30 GRAMS DAILY) REFERENCE THE RESEARCH FROM PAUL COX RUN THE NEURAL ZOOMER PLUS FOR DDX

GUT MICROBIOME AND MIGRAINES

Results

In IHGC datasets, ten, five, and nine bacterial taxa were found to have a causal association with migraine, MA, and MO, respectively, (IVW, all *P* < 0.05). *Genus.Coprococcus3* and *genus.Anaerotruncus* were validated in FinnGen datasets. Nine, twelve, and seven bacterial entities were identified for migraine, MA, and MO, respectively. The causal association still exists in *family.Bifidobacteriaceae* and *order.Bifidobacteriales* for migraine and MO after FDR correction. The heterogeneity and pleiotropy analyses confirmed the robustness of IVW results. Conclusion

Our study demonstrates that gut microbiomes may exert causal effects on migraine, MA, and MO. We provide novel evidence for the dysfunction of the gut-brain axis on migraine.

Migraine	Class						
& MA	Actinobacteria	0.89	25.78	IVW	16	0.0141	1.13(1.03-1.25)
	Family						
	ClostridialesvadinBB60group	0.50	22.03		15	0.0445	1.09(1.00-1.18)

МО	Class						
	Actinobacteria	0.93	26.16	IVW	15	0.0142	1.25(1.05-1.50)
	Melainabacteria	0.31	22.05		10	0.0410	0.87(0.76-0.99)
	Family						
	BacteroidalesS24.7group	0.36	23.14	IVW	8	0.0491	0.84(0.70-1.00)
	FamilyXI	0.16	22.42	IVW	8	0.0480	0.89(0.80-1.00)

Open access

Published: 17 July 2023

A causal effects of gut microbiota in the development of migraine

Qiang He, Wenjing Wang, Yang Xiong, Chuanyuan Tao, Lu Ma, Junpeng Ma, Chao You & The International Headache Genetics Consortium
The Journal of Headache and Pain

volume 24, Article number: 90 (2023) 193

GUT HEALTH AND COVID



A recent study of about 1,700 hospitalized coronavirus patients published in the journal *The Lancet* showed just how prevalent and long-lasting the condition may be. According to the findings, 76 percent COVID-19 patients were still dealing with at least one symptom six months after "recovering" from the virus. But what makes someone more likely to suffer from COVID for months? New research out of Hong Kong has found that one key factor may indicate the

severity and longevity of a patients' COVID symptoms: their gut health

Patients with COVID were found to have higher numbers of certain bacteria, including *Ruminococcus gnavus*, *Ruminococcus torques*, and *Bacteroides dorei*. They also had far lower levels of *Bifidobacterium adolescentis*, *Faecalibacterium prausnitzii*, and *Eubacterium rectale*, bacteria known to modify the immune system. The former two bacteria were particularly associated with infection severity.

THE RESEARCHERS ALSO NOTE THAT ANTIBIOTIC USE DURING RECOVERY COULD MAKE THEIR OUTCOMES WORSE AND PROBIOTICS COULD POTENTIALLY CONFER PROTECTION AND RESOLUTION

Conclusion Associations between gut microbiota composition, levels of cytokines and inflammatory markers in patients with COVID-19 suggest that

the gut microbiome is involved in the magnitude

of COVID-19 severity possibly via modulating host immune responses. Furthermore, the gut microbiota dysbiosis after disease resolution could contribute

to persistent symptoms, highlighting a need to understand how gut microorganisms are involved in inflammation and COVID-19.

Gut microbiota composition reflects disease severity and dysfunctional immune

Dufesponses in patients with GOVID-19 in part

Journal GutionYune Kiteyeon NOW Big 2020

GUT HEALTH



The gut microbiota influences blood-brain barrier permeability in mice

Viorica Braniste, Maha Al-Asmakh, [...], and Sven Pettersson Sci Transl Med. Author manuscript; available in PMC 2015 Apr 14. Published in final edited form as: Sci Transl Med. 2014 Nov 19; 6(263): 263ra158.

we demonstrated that lack of gut microbiota is associated with increased BBB permeability and altered expression of tight junction proteins. Fecal transfer from mice with pathogen-free gut flora into germ-free mice or treatment of germ-free mice with bacteria that produce short chain fatty acids (SCFA) decreased the permeability of the BBB.

GUT HEALTH



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.

<u>Bicknell B</u>1,2, <u>Liebert A</u>3,4, <u>Johnstone D</u>5, <u>Kiat H</u>6,7. 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

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, <u>THE POSSIBILITY EXISTS FOR PBMT</u> <u>TO BE USED AS AN ADJUNCT THERAPY IN TREATMENT OF OBESITY AND OTHER LIFESTYLE-RELATED DISORDERS, AS WELL AS CARDIOVASCULAR AND NEURODEGENERATIVE DISEASES</u>. THE CLINICAL IMPLICATIONS OF ALTERING THE MICROBIOME USING PBM WARRANTS FURTHER INVESTIGATION.

GUT HEALTH

Photobiomodul Photomed Laser Surg. 2019 Nov 1; 37(11): 681–693. Published online 2019 Nov 12. doi: 10.1089/photob.2019.4628

PMCID: PMC6859693 PMID: 31596658

ERCHONIA

"Photobiomics": Can Light, Including Photobiomodulation, Alter the Microbiome?

Ann Liebert, PhD,1,2 Brian Bicknell, PhD,3 Daniel M. Johnstone, PhD,4 Luke C. Gordon, BEHons, BMedSc,4 Hosen Kiat, MBBS, DMedSc,5,6 and Michael R. Hamblin, PhD7,8,9

Results: Recent work by our research group has demonstrated that PBM (red and NIR light) delivered to the abdomen in mice, can alter the gut microbiome in a potentially beneficial way. This has also now been demonstrated in human subjects.

Conclusions: In consideration of the known effects of PBM on metabolomics, and the now demonstrated effects of PBM on the microbiome, as well as other effects of light on the microbiome, including modulating circadian rhythms, the present perspective introduces a new term "photobiomics" and looks forward to the application of PBM to influence the microbiome in humans. Some mechanisms by which this phenomenon might occur are considered.

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WALL PROTEIN FRAGMENTS CALLED **MUROPEPTIDES ARE CARRIED IN** THE BLOOD STREAM TO THE **HYPOTHALAMUS BY A BILE ACID** CARRIER MOLECULE.

Bacterial sensing via neuronal Nod2 regulates appetite and body temperature ILANA GABANYI HTTPS://ORCID.ORG/0000-000 Displication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



A Look Inside Vibrant GZ 3.0

- Part 1: Commensal Bacteria (DNA)
- Part 2: Pathogens (DNA)
- Part 3: Functional Markers (Mass Spec)
- An immense amount of data summarized into 3 very concise, actionable pages

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VibrantWellness

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THE 5 R'S OF GUT HEALTH 1. REMOVE

1. REMOVE: ELIMINATE OFFENDING FOODS, ALLERGENS AND UNNECESSARY OTC MEDICATIONS COMMONLY USED NUTRITIONAL SUPPORT

BERBEMYCIN: BROAD SPECTRUM KILL AGENT

Amount Per Serving %Daily Value Oregon Grape (Mahonia aquifolium)(roots)

Grapefruit 5:1 Extract (Citrus × paradisi)(seed)

Berberine HCI

Uva Ursi Extract (Arctostaphylos uva-ursi)(leaves)(16% arbutin)

PROBIOMAX IG 26 DF: PASSIVE IG THERAPY PLUS SPORE BASED PROBIOTIC

FIXES LEAKY GUT AND INOCULATED AGAINST THE 29 MOST COMMON PATHOGENS INCLUDING C. DIFF! AVIAN GRAS Y IMMUNOGLOBULIN THERAPY IS MORE IMMUNE NEUTRAL TOWARDS HUMAN IMMUNOLOGY

> CELLCORE BIOSCIENCES FOUNDATIONAL PROGRAMS OTHER TARGET SPECIFIC PROTOCOLS LIKE CANDICIDAL LAURICIDIN/MONOLAURIN



RCHONIA

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390 mg **

350 mg

250 mg



5 R'S 2. REPLACE: FORTIFY NORMAL DIGESTIVE FUNCTION



RUN VIBRANT WHEAT AND GUT 3.0 ZOOMER, FOOD SENSITIVITY AND MICRONUTRIENT PANELS



BASIC GUT HEALTH XYMOGEN MEDPAX: 1 PROBIOMAX IG 26 DF 1 XYMOZYME HCL 1 HISTDAO





GI Balance

150 Vegetarian Dietary Capsules Sundement

5 CAPS/DAY 30 DAYS

Gl Balance Powder

2 SCOOPS/DAY

14 DAYS

Net WL. Dietary 517 or (192 g) Supplement EXCLUSIVE PROFESSIONAL FORMULAS



HistoDAO™ is a patented enzyme formula containing diamine oxidase (DAO)—the main enzyme responsible for the degradation of ingested histamine. This enzyme has been clinically tested and found to break down food-derived histamine in the digestive tract. DAO is not absorbed and does not have systemic activity. HistDAO does not manage or address antibodyrelated or IgE-related food allergies.* ERCHONIA VAGAL PROTOCOL SCM AND INTESTINAL REGION 10 HZ SETTING (10-10-10-10) 2+ MINUTES EACH REGION EVERY VISIT PERCUSSOR GI/VAGAL STIM



ProbioMax® IG 26 DF features clinically validated ingredients to support microbiome wellness and overall immune health. LactoSpore® (*Bacillus coagulans* MTCC5856) is a unique strain of shelf-stable L (+) lactic acid-producing bacteria with a naturally protective spore coating. IG 26 DF (IgY Max™), hyperimmunized egg powder, provides immunoglobulins and immune cofactors to support the body's natural defenses by limiting non-beneficial microbial adhesion.*

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Amount Per Serving %DV Calories 40 Total Carbohydrate

 7 g 3%†
 CANCER, GLUTAMATE ETC)

 Dietary Fiber
 Green Kiwifruit (Actinidia deliciosa)(fruit)(62,500 AU actinidin)S1

 400 mg ***
 400 mg ***

 1 g
 Marshmallow (Althaea officinalis)(root)

 2 inc (as zinc I-carnosine)
 400 mg ***

 32.25 mg 293%
 Licorice Extract (Glycyrrhiza glabra)(root)(3.5% glabridin)S2

 293%
 300 mg ***

 L-Glutamine
 300 mg ***

 4 g Slippery Elm (Ulmus fulva)(bark)
 200 mg ***

 2 g
 Prune (Prunus domestica)(fruit)

 N-Acetyl-D-Glucosamine
 200 mg

** Chamomile Extract (*Matricaria recutita*)(flowering head)(1% apigenin-7-glucoside)

Aloe Vera 200:1 Extract (Aloe barbadensis)(leaf gel)

Cat's Claw (Uncaria tomentosa)(bark)

00

Porcine Gastric Mucin

400 ma

200 mg Duplication, citation or distributio?96f this material in whole or in part

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4. REINOCULATE (CONSIDER ROTATION)



CONSIDER SOLUTIONS FOR LONG TERM GUT HEALTH:

1. ALWAYS ASSESS AUTONOMIC FUNCTION AND THE BRAIN GUT AXIS (HRV, BP, CAROTID RESPONSE). A HEALTHY BRAIN AND VAGAL OUTPUT IS ESSENTIAL FOR PROPER DIGESTIVE FUNCTION AND TONE! VAGAL NERVE PROTOCOLS.

5. REBALANCE

2. EDUCATE PATIENT OF THE HAZARDS OF A STRESSFUL LIFESTYLE (JOBS, RELATIONSHIPS, EVEN RELAXED EATING. TAKE TIME TO ENJOY MEALS. DON'T EAT SCARED!) USE FOOD SENSITIVITY AND ALLERGY TESTING AS THE GOLD STANDARD FOR ELIMINATION. WHEN IN DOUBT, TRY GLUTEN FREE, DAIRY FREE AND SOY FREE.

3. CONSIDER A FULL DETOX PROGRAM. 1-3 DAY WATER FASTS ARE AN EFFECTIVE REPAIR AND RESET IDEA. XYMOGEN'S DETOX RENEWAL MICRO-KIT IS AN EASY TO FOLLOW PROGRAM.

4. WATCH FOR CO-MORBIDITIES AND PATHOLOGY. MOLD AND OTHER PATHOGENS MAY BE IN PLAY. THE PATIENT MAY NEED A REFERRAL.

5. PROPER BOWEL MOVEMENTS ARE A MUST TO ELIMINATE WASTE AND TOXINS. MAGNESIUM AND FIBER SUPPORT (XYMOGEN'S OPTIFIBER LEAN) MAY BE INDICATED.



ERCHONIA

ERCHONIA

UPDATES ON GUT HEALTH



RCHONIA

Chemical residue on clean plates

A typical cycle in a commercial dishwasher involves circulating hot water and detergent for around 60 seconds at high pressure. Afterwards, there is a second 60-second washing and drying cycle in which water and a rinse agent are applied.

"What's especially alarming is that in many appliances, there's no additional wash cycle

to remove the remaining rinse aid," says Cezmi Akdis, UZH professor of experimental allergology and immunology and director of the SIAF, who led the study. "This means that potentially toxic substances remain on the dishes, where they then dry in place."

The result was that high doses of rinse agents killed the intestinal epithelial cells and lower doses made it more permeable. Researchers also

observed the activation of several genes and cell signaling proteins that could trigger inflammatory responses. A more

detailed analysis showed that one component of the rinse agent - alcohol ethoxylates -

was responsible for this reaction.

"It is important to inform the public about this risk, since alcohol ethoxylates seem to be commonly used in commercial dishwashers."

Ismail Ogulur, Yagiz Pat, Tamer Aydin, Duygu Yazici, Beate Rückert, Yaqi Penq, Juno Kim, Urszula Radzikowska, Patrick Westermann, Milena Sokolowska, Raja Dhir, Mubeccel Akdis, Kari Nadeau, Cezmi A. Akdis. Gut epithelial barrier damage caused by dishwasher detergents anggrase aids. The Journal of Allergy and Clinical Immunology. 1 December 2022. DOI: 10.1016/j.jaci.2022.10.020

GUT HEALTH



Volume 14, Issue 6, June 2015, Pages 479-489

ELSEVIER Changes in intestinal tight junction permeability associated with industrial food additives explain the rising incidence of autoimmune disease

Author links open overlay panel AaronLerneraTorstenMatthiasb

Abstract

The incidence of autoimmune diseases is increasing along with the expansion of industrial food processing and food additive consumption.

The intestinal epithelial barrier, with its intercellular tight junction, controls the equilibrium between tolerance and immunity to non-self-antigens. As a result, particular attention is being placed on the role of tight junction dysfunction in the pathogenesis of AD. Tight junction leakage is enhanced by many luminal components, commonly used industrial food additives being some of them.

Glucose, salt, emulsifiers, organic solvents, gluten, microbial transglutaminase, and nanoparticles are extensively and increasingly used by the food industry, claim the manufacturers, to improve the qualities of food. However, all of the aforementioned additives increase intestinal permeability by breaching the integrity of tight junction paracellular transfer. In fact, tight junction dysfunction is common in multiple autoimmune diseases and the central part played by the tight junction in autoimmune diseases pathogenesis is extensively described. It is hypothesized that commonly used industrial food additives abrogate human epithelial barrier function, thus, increasing intestinal permeability through the opened tight junction, resulting in entry of foreign immunogenic antigens and activation of the autoimmune cascade. Future research on food additives exposure-intestinal permeability–autoimmunity interplay will enhance our knowledge of the common mechanisms associated with autoimmune progression.

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GUT HEALTH



Food Dyes and Pigments Acid Blue #3 (Patent Blue V) Acid Red #14 (Carmoisine) Annatto **Beta-Carotene** Blue #1 (Brilliant Blue) Blue #2 (Indigo Carmine) Brilliant Black **Cochineal Extract** Green #3 (Fast Green) Red #2 (Amaranth Red) Red #3 (Erythrosine) Red #4 (Carmine) Red #40 (Allura Red) Yellow #5 (Tartrazine) Yellow #6 (Sunset Yellow) Pesticides Deltamethrin Glyphosate Sweeteners Acesulfame K Aspartame Erythritol Mannitol Monk fruit Saccharin Sorbitol Stevia Sucralose (Splenda) **Xylitol** Elements Fluoride **Nickel Sulfate** Titanium dioxide



2. GUT DYSBIOSIS/PATHOGENS/ MALABSORPTION (GZ3.0)

3. ADDITIVES TEST AND ELIMINATE

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4. VAGAL TONE (FN REFERRAL?)

Preservatives and Antioxidants Benzoic Acid Butylated Hydroxyansiole (BHA) Butylated Hydroxytoluene (BHT) **Citric Acid** Sodium Sulfite Sorbic Acid Formaldehyde Sodium Benzoate Sodium Nitrate **Emulsifiers and Surfactants** Lecithin (Soy) Lecithin (Egg yolk) Polysorbate 80 Flavor Enhancers Monosodium Glutamate (MSG) Ammonium Chloride Sodium Citrate Gums and Thickening Agents **Gum Arabic** Guar Gum Gum Tragacanth Locust Bean Gum Mastic Gum Xanthan Gum Beta-Glucan Carrageenan Cottonseed **Fibrous Additives** Ispaghula or Psyllium Husk Other Latex **Bisphenol A (BPA)**

ARTIFICIAL SWEETENERS



Common sweetener now linked to impaired memory and learning

Researchers at Florida State University (FSU) College of Medicine have found that male mice

consuming aspartame at significantly lower levels than deemed safe by the Food and Drug Administration (FDA) passed on spatial learning and memory deficits to their offspring.

Open Access

Published: 31 August 2023

Learning and memory deficits produced by aspartame are heritable via the paternal lineage

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ARTIFICIAL SWEETENERS



Chemical found in 'widely used sweetener' damages human DNA, new study says

North Carolina State University shared the findings of its new study in a news release. It names

"sucralose," said to be the most widely used artificial sweetener in the United States, as the "issue."

To put this in context, the European Food Safety Authority has a threshold of toxicological concern for **all genotoxic** substances of 0.15 micrograms per person per day," Schiffman explains. "Our work suggests that the trace amounts of sucralose-6-acetate in a single, daily sucralose-sweetened drink exceed that threshold. And that's not even accounting for the amount of sucralose-6-acetate produced as metabolites after people consume sucralose."

"When we exposed sucralose and sucralose-6-acetate to gut epithelial tissues – the tissue that lines your gut wall - we found that both chemicals cause 'leaky gut.' Toxicological and pharmacokinetic properties of sucralose-6-acetate and its parent sucralose: in vitro screening assays

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Susan S. Schiffman, Elizabeth H. Scholl, Terrence S. Furey &H. Troy Nagle Published online: 29 May 2023

ARTIFICIAL SWEETENER WARNING!



From 2009 to 2021, researchers in France studied more than

100,000 adults and their intake of artificial sweeteners. They reported that, based on the results, artificial sweeteners may lead to an increased risk of cancer, specifically

breast and obesity-related cancers. People who consumed any type of "high-intensity sweetener" had up to a 14% greater chance of developing cancer. **EXAMPLES OF COMMONLY USED ARTIFICIAL SWEETENERS:**

saccharin, aspartame, acesulfame potassium, sucralose, and neotame.

OPEN ACCESS PEEB-BEVIEWED RESEARCH ARTICLE

Artificial sweeteners and cancer risk: Results from the NutriNet-Santé populationbased cohort study

- Charlotte Debras Eloi Chazelas,
- Published: March 24, 2022 Duplication, citation or distribution of this material in whole or in part https://doi.org/10.1371/journal.pmed.1003950 thout permission of Trevor Berry, DC, DACNB is prohibited.



Just One Diet Coke Or Pepsi Max A Day Can Triple Your Risk Of Stroke

This US-based study found that people who consume only one diet drink per day triple their chances of suffering from the most commonly occurring stroke compared to people who don't consume any diet drinks. It turns out that they are also 2.9 times more likely to develop Alzheimer's.

Diet drinks are just bad all around. <u>Besides these deadly</u> <u>diseases</u>, they are also known to cause weight gain, diabetes, and heart issues.

Daily Consumption of Sodas, Fruit Juices and Artificially Sweetened Sodas Affect Brain

Posted 6 years ago on Thursday, April 20th, 2017 in Featured, In the Media, Research

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ARTIFICIAL SWEETENER WARNING!



Humans' Diet Coke addiction linked to rising anxiety due to artificial sweetener

Researchers at **Florida State University** say they tested the put aspartame in mice's drinking water, only to find they began to display anxious behaviour while the part of their brains linked to anxiety and fear changed.

The **scientists** found changes in the genes in the mice's amygdala - which processes potential threats and triggers our fight or flight responses.

Scientist Dr Pradeep Bhide of the University's Centre for Brain Repair, who led the study, said they **tested the** human equivalent of "two to four" small cans of diet fizzy drinks' worth of aspartame on the mice.

Most shockingly in the study, male mice which displayed brain changes after drinking the sweetener appeared to pass those on to their offspring, suggesting anxiety could be inherited from parents.



J Clin Gastroenterol. 2015 Aug;49(7):571-6. doi: 10.1097/MCG.00000000000257.

Small Intestinal Transit Time Is Delayed in Small Intestinal Bacterial Overgrowth.

Roland BC¹, Ciarleglio MM, Clarke JO, Semler JR, Tomakin E, Mullin GE, Pasricha PJ.



MANY GI CONDITIONS ARE ASSOCIATED WITH SLOWED GI TRANSIT TIME. ALWAYS CONSIDER VAGAL TONE AND FUNCTIONAL NEUROLOGICAL APPLICATIONS (VAGAL PROTOCOLS)

ACID SUPPRESSION CAN CAUSE GUT DYSBIOSIS AND ALTERED TRANSIT TIME

WATCH GLP-1 AGONISTS (OZEMPIC, BERBERINE) AND SIBO/DYSBIOSIS DUE TO THEIR SLOWING OF GI FUNCTION!

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



VAGAL (10HZ) +

LINE BEAM

LINEAR DOWN

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

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.



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



ESOPHOGEAL/ EPIGASTRIC REGION 2 MINUTES/ VISIT VIBRANT LAB



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 REGIONATION FOR SPINE BIB/REGIANDEGI PELEASE without permission of Trevor Berry, DC, DACNB is prohibited.
ADIPOSE REDUCTION





ZERONA Z6 (OTC)

EMERALD (MEDICAL)

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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.





Harmful Body Fat Not Only Increases Your Waistline, but Also Your Risk of Dementia

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

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 17:01 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," <u>says researcher</u> <u>Simone Kaltenhauser</u>, 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 the 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 hungcler porties after fatvbgBerry, DC, DACNB is prohibited.

BRAIN AND FAT



Scientists Discover a Secret Messenger Between Fat And The Brain

01 September 2022

Scientists thought they understood how our brains kept tabs on the levels of fat in our bodies: by monitoring fat-associated hormones in our bloodstreams.

But in a new study, researchers now discovered an entire additional messaging system.

It turns out we have a whole sensory system dedicated to carrying messages from our fat tissues (adipose) to our brains.

"The discovery of these neurons suggests for the first time that your brain is actively surveying your fat, rather than just passively receiving messages about it," says Scripps Research Institute neuroscientist Li Ye.

The role of somatosensory innervation of adipose tissues

Yu Wang, Verina H. Leung, Yunxiao Zhang, Victoria S. Nudell, Meaghan Loud, M. Rocio Servin-Vences, Dong Yang, Kristina Wang, Maria Dolores Moya-Garzon, Veronica L. Li, Jonathan Long, Ardem Patapoutian & Li Ye
Nature (2022)
 221

ADIPOSE REDUCTION





THE BRAIN-ADIPOSE COMBO: WE HAVE ONE OF OUR FX-635s IN THE SAME ROOM AS OUR ZERONA AND COMBINE THE TWO TOGETHER. A VERY POWERFUL CLINICAL TOOL THAT SAVES

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Open Access
 Published: 31 August 2022

without permission of Trever Berry, DC, DACNB is prohibited. LONG COVID AND OBESITY ardiovasc. Med., 07 February 2023



Sec. Thrombosis and Haemostasis Volume 10 - 2023 | https://doi.org/10.3389/fcvm.2023.106249 This article is part of the Research Topic COVID-19 and Thrombo-inflammatory Responses View all 12 drites

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.

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

<u>Trends Parasitol. 2017 Apr; 33(4): 276–284.</u> Published online 2016 Dec 19. doi: <u>10.1016/j.pt.2016.11.008</u>

PMCID: PMC5376508 NIHMSID: NIHMS834449 PMID: <u>28007406</u>

Adipose tissue - a safe haven for parasites?

Herbert B Tanowitz,1 Philipp E Scherer,2 Maria M Mota,3 and Luisa M Figueiredo3. Duplication, citation or distributio??® fithis material in whole or the without permission of Trevor Berry, DC, DACNB is prohibited.

CONSIDER ZERONA FOR CHRONIC PATHOGEN AND TOXIN BURDEN PROTOCOLS

ADIPOSE REDUCTION AND ARTHRITIS PAIN



J Atheroscler Thromb. 2017 Nov 1; 24(11)

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 fat1, 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 resistance3, 4).



(B) In leptin resistant obese individuals, effects of leptin, though its plasma levels are increased, is decreased in hypothalamus. Insufficiency of leptin signaling causes hyperinsulinemia in pancreatic β -cells, which lead to subsequent impairment of insulin secretion and diabetes mellitus. Leptin resistance increases productions of leptin in white adipose tissue, but cannot abolish fat

via HSL. Combined above mechanisms, leptin

resistance instigates ectopic fat disposition in the liver and the skeletal muscle, provoking insulin resistance and chronic inflammation.

ADIPOSE REDUCTION AND ARTHRITIS PAIN



Nat Rev Rheumatol

2011 Mar;7(3):161-9. doi: 10.1038/nrrheum.2010.213. Epub 2011 Jan 4.

The role of mitochondria in osteoarthritis

Abstract

Francisco J Blanco 1, Ignacio Rego, Cristina Ruiz-Romero

Mitochondria are important regulators of cellular function and survival that may have a key role in aging-related diseases. Mitochondrial DNA (mtDNA) mutations and oxidative stresses are known to contribute to aging-related changes. Osteoarthritis (OA) is an aging-associated rheumatic disease characterized by articular cartilage degradation and elevated chondrocyte mortality. Articular cartilage chondrocytes survive and maintain tissue integrity in an avascular, low-oxygen environment. Recent ex vivo studies have reported mitochondrial dysfunction in

human OA chondrocytes, and analyses of mitochondrial electron transport chain activity in these cells show decreased activity of Complexes I, II and III compared to normal chondrocytes.

This mitochondrial dysfunction may affect several pathways that have been implicated in cartilage degradation, including exidative stress, defective chondrocyte biosynthesis and growth responses, increased cytokine-induced chondrocyte inflammation and cartilage matrix calcification, and increased chondrocyte apoptosis. Mitochondrial dysfunction in OA chondrocytes may mutations in the mtDNA or from the direct effects of proinflammatory mediators such as cytokines, prostaglandins, rea nitric oxide. Polymorphisms in mtDNA may become useful as biomarkers for the diagnosis and prognosis of OA, and biomarkers by mtDNA haplogroups supports the concept that mtDNA haplogroups may define specific OA phenotype process.



CONSIDER THE ERCHONIA GVL LASER FOR OSTEOARTHRITIC JOINTS!

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ADIPOSE REDUCTION



Basic Clin Pharmacol Toxicol actions

. 2014 Jan;114(1):103-8. doi: 10.1111/bcpt.12160. Epub 2013 Nov 20.

Leptin - a link between obesity and osteoarthritis. applications for prevention and treatment

Katriina Vuolteenaho 1, Anna Koskinen, Eeva Moilanen

Abstract

Osteoarthritis (OA) is the most common cause of musculoskeletal disability and pain in the world. The current drug treatment for OA is symptom relieving, and there is an urgent need for treatments that could retard, prevent or repair cartilage destruction in OA. Obesity is a major risk factor for OA. Traditionally, it has been thought to contribute to the development of OA by increasing the load on weight-bearing joints. However, this appears to be an over-simplification, because obesity is also linked to OA in the hand and

finger joints. Recent studies have shown that adipocytokine leptin is a possible link between obesity and OA: Leptin levels in synovial fluid are increased in obese patients, leptin receptor (Ob-R) is expressed in cartilage, and leptin induces the production of matrix metalloproteinases (MMPs), pro-inflammatory mediators and nitric oxide (NO) in chondrocytes. Furthermore, according to the very recent findings, not only leptin levels in the joint

but also leptin sensitivity in the cartilage are enhanced in obese OA patients. The findings supporting leptin as a causative link between obesity and OA offer leptin as a potential target to the development of disease-modifying drugs for osteoarthritis (DMOAD), especially for obese patients.

JOINT FORMULAS LIKE CHONDROITIN AND GLUCOSAMINE SULFATE ACTUALLY ACT ON THE HYPOTHALAMUS TO PROMOTE SENSITIVITY TO LEPTINS (THEIR ACTION IS NOT LIMITED TO THE CARTILAGE ITSELF)!

ADIPOSE AND PAIN



The prevalence of obesity skyrocketed over the past decades to become a significant public health problem. Obesity is recognized as a low-grade inflammatory disease and is linked with several comorbidities such as diabetes, circulatory disease, common neurodegenerative diseases, as well as chronic pain. Adipocytes are a major neuroendocrine organ that continually, and systemically, releases pro-inflammatory factors. While the exact mechanisms driving obesity-induced pain remain poorly defined, nociceptor hypersensitivity may result from the systemic state of inflammation characteristic of obesity as well as weight surplus-induced mechanical stress. Obesity and pain also share various genetic mutations, lifestyle risk factors, and metabolic pathways. For instance, fat pads are often found hyper-innervated and rich in immune cell types of multiple origins. These immunocytes release cytokines, amplifying nociceptor function, which, in turn, via locally released neuropeptides, sustain immunocytes' function. Here, we posit that along with mechanical stress stemming from extra weight, the local neuro-immune interplay occurring within the fat pads maintains the state of chronic low-grade inflammation and heightens sensory hypersensitivity. Overall, stopping such harmful neuro-immune crosstalk may constitute a novel pathway to prevent obesity-associated comorbidities, including neuronal hypersensitivity.

SUMMARY: FAT = INFLAMMATION



Front Hum Neurosci. 2020; 14: 181. Published online 2020 Jun 9. doi: <u>10.3389/fnhum.2020.00181</u>

PMCID: PMC7295985 PMID: <u>32581740</u>

Neuro-Immunity Controls Obesity-Induced Pain

Tuany Eichwald

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ADIPOSE REDUCTION AND VISCERAL FAT



Published: 18 August 2021

Neuro-mesenchymal units control ILC2 and obesity via a brain–adipose circuit

• Filipa Cardoso, Roel G. J. Klein Wolterink,

Nature (2021)

In recent years, scientists have begun to suspect the nervous system and the immune system are both working together to control visceral fat. After all, this deep yellow matter not only houses nerve fibers, it also contains immune cells.

"The neurons and the immune cells were not talking to each other," explains molecular biologist Ana Filipa Cardoso from the Champalimaud Center for the Unknown in Portugal. "So we investigated other candidates in the tissue, finally coming across a rather unexpected 'middleman'."

What was once thought to be a cellular bystander, has now turned out to be a critical mediator. In fact, mesenchymal cells (MSCs) were all but ignored by scientists until fairly When researchers burned away mesenchymal cells in the deep fat of mice, the nearby immune cells, known as type 2 innate lymphoid cells (ILC2), stopped regulating the growth of the fat

Effect of low-level laser therapy on mesenchymal stem cell proliferation: a systematic review

Fernanda Ginani, Diego Moura Soares, Mardem Portela e Vasconcelos Barreto & Carlos Augusto Galvão Barboza Galvão Barboza

 The analysis of the papers showed a concentration of experiments using LLLT on stem cells derived from bone marrow, dental pulp, periodontal ligament, and adipose tissue. It can be concluded that the laser therapy positively influences the in vitro proliferation of stem cells studied

ADIPOSE REDUCTION, LLLT AND PERIPHERAL INSULIN RESISTANCE



Lasers Med Sci

. 2018 Aug;33(6):1245-1254. doi: 10.1007/s10103-018-2465-1. Epub 2018 Feb 23.

The effects of exercise training associated with low-level laser therapy on biomarkers of adipose tissue transdifferentiation in obese women

Abstract

Investigations suggest the benefits of low-level laser therapy (LLLT) to improve noninvasive body contouring treatments, inflammation, insulin resistance and to reduce body fat. However, the mechanism for such potential effects in association with exercise training (ET) and possible implications in browning adiposity processes remains unclear.

LLLT promotes an improvement in body composition and inflammatory processes as previously demonstrated. The Phototherapy group especially presented positive modifications of WNT5 signaling, FGF-21, and ANP, possible biomarkers associated with browning adiposity processes. This suggests that this kind of intervention promotes results applicable in clinical practice to control obesity and Petricel claim or fight the material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ADIPOSE REDUCTION, LELT AND PERIPHERAL INSULIN RESISTANCE



PBM therapy improved glucose tolerance and phosphorylation of Akt (Ser473) and reversed the HFDinduced reduction of GLUT4 content and phosphorylation of AS160 (Ser588). Also, PBM therapy reversed the increased area of epididymal and mesenteric adipocytes. The results showed that chronic PBM therapy improved parameters related to obesity and insulin resistance in HFD-induced obesity in mice.

Lasers Med Sci

. 2018 Apr;33(3):559-571. doi: 10.1007/s10103-017-2408-2. Epub 2017 Dec 15.

Infrared photobiomodulation (PBM) therapy improves glucose metabolism and intracellular insulin pathway in adipose tissue of high-fat fed mice

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.

<u>Bicknell B</u>1,2, <u>Liebert A</u>3,4, <u>Johnstone D</u>5, <u>Kiat H</u>6,7. 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, <u>THE POSSIBILITY EXISTS FOR PBMT</u> <u>TO BE USED AS AN ADJUNCT THERAPY IN TREATMENT OF OBESITY AND OTHER LIFESTYLE-RELATED DISORDERS, AS WELL AS CARDIOVASCULAR AND</u> <u>NEURODEGENERATIVE DISEASES</u>. THE CLINICAL IMPLICATIONS OF ALTERING THE MICROBIOME USING PBM WARRANTS FURTHER INVESTIGATION.



ADIPOSE REDUCTION



BASIC 8 TREATMENT PLATFORM: 2X/WEEK FOR 3 WEEKS 1X/WEEK FOR 2 WEEKS FOLLOW ERCHONIA'S TREATMENT PLAN (LYMPHATICS, WALK, H20 ETC) PRE-SCREEN MAF

> OTHER CONSIDERATIONS: EXTENDED TX PLANS BASED ON SIZE AND GOALS REFERRAL VISITS "MEMBERSHIP MODEL"

0.50







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WEIGHT LOSS



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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

WEIGHT LOSS

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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
- 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 WATCH BERBERINE IN THI DOMINANT CONDITIONS

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ERCHONIA

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

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

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



ADIPOSE REDUCTION

THE 30-30-30 FAT MELTING WORKOUT ROUTINE





The 4-Hour

OTHY FERRI

BEAD BY ZACH MICLARTY

Beta-hydroxy-beta-methylbutyrate PROTECT MUSCLE WHILE WHILE BURNING FAT. ALSO USED IN CATABOLIC STATES LIKE CANCER CACHEXIA, AIDS AND AGING. 3 GRAMS/DAY

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)

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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 <u>Cell Reports</u>, 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

Published: 28 April 2023

XYMOGEN QR CODE FOR NON-PHYSICIAN ACCOUNT PURCHASES



ALTHOUGH IT IS HIGHLY ENCOURAGED TO OPEN YOUR OWN WHOLESCRIPTS ACCOUNT TO BENEFIT FROM XYMOGEN PLUS 99 OTHER PRODUCT LINES, THIS QR CODE WILL LINK YOU TO DIRECT PURCHASE IF YOU WANTED TO TRY PRODUCTS FIRST OR FOR THOSE THAT DON'T HAVE AN NPI NUMBER TO OPEN THEIR OWN ACCOUNT SUCH AS PATIENTS IN ATTENDANCE OR NON-MEDICAL PROVIDERS



NOTE: THIS IS DR. BERRY'S WHOLESCRIPTS ACCOUNT

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ERCHONIA

ERCHONIA ZERONA Z6 AND EMERALD

BODY CONTOURING VS WELLNESS PROGRAM

MAKE YOUR Z6 OR EMERALD THE CENTERPIECE OF A GLOBAL WELLNESS PLATFORM, BRAIN AND BODY HEALTH



BREAK

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PAIN MANAGEMENT





PERIPHERAL SENSITIZATION DOWN REGULATION

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our new study, published July in the *Lancet* medical journal, found opioids do not relieve "acute" low back or neck pain (lasting up to 12 weeks) and can result in worse pain.

Prescribing opioids for low back and neck pain can also cause harms ranging from common side effects – such as nausea, constipation and dizziness – to misuse, dependency, poisoning, and death.

After six weeks of treatment, taking opioids did not result in better pain relief compared to the placebo.

Nor were there benefits to other outcomes such as physical function, quality of life, recovery time, or work absenteeism.

More people in the group treated with opioids experienced nausea, constipation, and dizziness than in the placebo group.

More people in the opioid group reported pain at one year: 66 people compared to 50 in the placebo group.

Opioid analgesia for acute low back pain and neck pain (the OPAL trial): a randomised placebo-controlled trial

LANCE

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CHRONIC PAIN AND MITOCHONDRIA



Mitochondrial and metabolic disturbances persist in sensory neurons after inflammation

•Disturbed redox balance in DRG predisposes to failing inflammatory pain resolution

•Mitochondrial ATPSc-KMT links to disturbed cellular redox balance

•ATPSCKMT knockdown or NAD+ supplementation restores failing pain resolution pathways



SUMMARY: ANIMAL STUDIES SHOW PRIMED DORSAL ROOT GANGLIA IN ACUTE PAIN FROM INFLAMMATION CONTRIBUTING TO CHRONIC PAIN AND HYPERALGESIC STATES RESPONDED WELL TO INJECTION OF NICOTINAMIDE RIBOSIDE TO HELP SUPPLY NAD+ PRECURSORS TO RESTORE MITOCHONDRIAL BALANCE IN THE PRIMED MICE.

Cell Rep Med. 2023 Nov 21; 4(11): 101265. Published online 2023 Nov 8. doi: 10.1016/j.xcrm.2023.101265

PMCID: PMC10694662 PMID: <u>37944527</u>

Inflammation-induced mitochondrial and metabolic disturbances in sensory neurons control the switch from acute to chronic pain

CHRONIC PAIN AND MITOCHONDRIA



2003 Aug;2(8):851-5. doi: 10.1039/b301233f.

Photochem Photobiol Sci

Modulation of macrophage structure and function by low level He-Ne laser irradiation

He-Ne laser (632.8 nm, approximately 10 W m(-2)) at energy densities ranging from 100 to 600 J m(-2) and the activities of lysozyme and cathepsin, phagocytosis, and cell spreading (markers of cell activation), as well as changes in NAD(P)H autofluorescence, were monitored. He-Ne laser irradiation was observed to lead to significant changes in all the parameters investigated.

Biochem Biophys Rep

2021 Jul 26:27:101085. doi: 10.1016/j.bbrep.2021.101085. eCollection 2021 Sep.

Effectiveness of low-power laser therapy in improvement of the peripheral neuropathy induced by xenobiotics in rats

NAD+ LEVELS CAN BE IMPROVED THROUGH DIET BUT IV DRIPS AND LOW LEVEL LASER THERAPY ARE MUCH MORE EFFECTIVE TO BRING NAD+ TO THERAPEUTIC LEVELS. STACK NAD+ IV AS DESCRIBED BELOW WITH YOUR LASER TREATMENTS FOR CHRONIC PAIN PATIENTS:

IV infusions: Contains 500mg of NAD in 500 ml of saline solution, typically administered multiple times over the course of several weeks.

Injections: A 500 mg or 1000mg vial is self-administered using a subcutaneous injection over a set period of time. (NOTE: IV DRIPS CAN TAKE 3-4 HOURS FOR ADMINISTRATION. CONSIDER MULTIPLE TREATMENTS - 4-6 TRIAL 1X/WEEK)

Nasal sprays: Contains 30 mg/spray in a 300 mg/ml bottle, typically administered once per day.

STACK WITH YOUR ERCHONIA LASER. COMMON MSK SETTING 9-16-42-53. BASE PAIN TREATMENT TRIAL 6 VISITS. CHRONIC OR COMPLEX PAIN PROTOCOLS USE A 12 VISIT TREATMENT TRIAL. ALSO CONSIDER THE "DOWNSTREAM" PROTOCOL

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LASER AND TOPICAL APPLICATIONS NEURO-MUSCULOSKELETAL CONSIDERATIONS



CURRENT BACK PAIN TREATMENT MODEL

ERCHONIA

<u>Surg Neurol Int.</u> 2021; 12: 132. Published online 2021 Mar 30. doi: <u>10.25259/SNI_167_2021</u>

Middle cluneal nerve entrapment neuropathy attributable to lumbar disc herniation

Juntaro Matsumoto, 1 Toyohiko. Isu, 1 Kyongsong Kim, 2 Koichi Miki, 1 and Masanori Isobe1 Author information Article notes Copyright and License information Disclaimer

Background:

Middle cluneal nerve entrapment neuropathy (MCN-EN) is a known cause of low back pain (LBP). Here, we succeeded in treating a patient with a lumbar disc herniation who actually had MCN-EN with a nerve block and neurolysis.

Case Description:

A 52-year-old female presented with severe left lower back and lateral thigh pain making it difficult to walk. Lumbar MRI

revealed a disc herniation on the left at the L5/S1 level. On palpation, we identified a trigger point on the buttock where the MCN penetrates the long posterior sacroiliac ligament between the posterior superior and inferior iliac spine. Two left-sided MCN blocks, followed by the left MCN neurolysis procedure, were performed under local anesthesia. At the last follow-up, 10 months after surgery, the LBP has not recurred, and she requires no medications for pain control.

Conclusion:

CURRENT BACK PAIN TREATMENT MODEL



Superior and Middle Cluneal Nerve Entrapment as a Cause of Low Back Pain Toyohiko Isu, Kyongsong Kim, Daijiro Morimoto, Naotaka Iwamoto Neurospine. 2018 Mar; 15(1): 25–32. Published online 2018 Mar 28. doi: 10.14245/ns.1836024.012 PMCID: PMC5944640 ArticlePubReaderPDF-230KCite

Select item 8053461

Middle cluneal nerve entrapment neuropathy attributable to lumbar disc herniation Juntaro Matsumoto, Toyohiko Isu, Kyongsong Kim, Koichi Miki, Masanori Isobe Surg Neurol Int. 2021; 12: 132. Published online 2021 Mar 30. doi: 10.25259/SNI_167_2021 PMCID: PMC8053461 ArticlePubReaderPDF-726KCite

Select item 5476578

3.

Anatomical study of middle cluneal nerve entrapment

Tomoyuki Konno, Yoichi Aota, Tomoyuki Saito, Ning Qu, Shogo Hayashi, Shinichi Kawata, Masahiro Itoh J Pain Res. 2017; 10: 1431–1435. Published online 2017 Jun 13. doi: 10.2147/JPR.S135382 PMCID: PMC5476578 ArticlePubReaderPDF–594KCite

Select item 5677392

4.

Anatomical etiology of "pseudo-sciatica" from superior cluneal nerve entrapment: a laboratory investigation Tomoyuki Konno, Yoichi Aota, Hiroshi Kuniya, Tomoyuki Saito, Ning Qu, Shogo Hayashi, Shinichi Kawata, Masahiro Itoh J Pain Res. 2017; 10: 2539–2545. Published online 2017 Nov 1. doi: 10.2147/JPR.S142115 PMCID: PMC5677392 ArticlePubReaderPDF=2.7MCite Select item 6773996

5.

Characteristics of Low Back Pain due to Superior Cluneal Nerve Entrapment Neuropathy Koichi Miki, Kyongsong Kim, Toyohiko Isu, Juntaro Matsumoto, Rinko Kokubo, Masanori Isobe, Tooru Inoue Asian Spine J. 2019 Oct; 13(5): 772–778. Published online 2019 May 14. doi: 10.31616/asj.2018.0324 PMCID: PMC6773996 ArticlePuReaderPDF=476KCite

Select item 4299373

6. 253 <u>Prospective study of superior cluneal nerve disorder as a potential cause of low back pain and leg symptoms</u> Hiroshi Kuniya Yoichi Aota Takuya Kawai Kan-ichiro Kaneko Tomovuki Konno Tomovuki Saito

Inflammation of Superficial Nociceptive nerves can masquerade as radiculopathy & "joint" pain.



Multiple tissues (tendon, periosteum, ligament etc) are innervated by nerves that converge as they approach/enter CNS (superficial inflammation can feel like "deep joint pain").





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COURSE OF SUPERIOR CLUNEAL NERVES

- Psoas Major
- (Lumbar Lordosis (hypo- vs. hyper-))
- Paraspinals (esp. adj. to L1-3, iliocostalis, & origin of Longissimus Thoracis)
- Thoracolumbar Fascia
- Quadratus Lumborum
- (Inferior Latissimus Dorsi)
- Iliac Crest.



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SECTION 1 | PRACTICAL LAB | LOWER BACK PAIN





CLINICAL NOTES: MIDDLE CLUNEAL NERVES

- The MCNs, especially the superior branches, often communicate with the medial superior cluneal nerve (Tubbs et al, 2010).
- MCNs are therefore frequently treated in conjunction with the superior cluneal nerves.
- The middle cluneals may be encountered during approaches to the dorsal sacrum, such as sacroiliac joint injections, or harvest procedures of the posterior iliac crest that approach the PSIS.
- MCN neuralgia can easily be misdiagnosed and sacroiliac joint pain.
- MCNs may be provoked by prolonged sitting and sports that involved the repetitive movement or prolonged posture in the seated position, such as rowing, jiu-jitsu, judo, cycling and driving.



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THE ERCHONIA ADVANTAGE



WITH ERCHONIA'S PATENTED LINE BEAM GENERATION OR FX SCANNING BEAMS TO FACILITATE SURFACE AREA EXPOSURE IN PAIN SYNDROMES,

COMBINED WITH THE PROPER LOW NON-THERMAL DOSE AND OPTIMAL MONOCHROMATIC WAVELENGTHS,

THEY CONTINUE TO BE THE INDUSTRY LEADER IN TREATMENT FOR ALL NEUROMUSCULOSKELETAL CONDITIONS!

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BASIC LOW BACK CASE: FX 405 SET UP



LASER NEUROLOGY

CHRONIC LOW BACK PAIN SOLUTION

- We have completed the chronic low back pain study.
- Level 1 studies are randomized parallel group, double-blind, placebo-controlled, multi-site studies that require rigorous quality control, resources and procedures.
- They are considered the most reliable as to whether or not a treatment is effective.
- You will be able to advertise directly to the consumer for a 170 billion dollar industry.



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Level 1 IRB regulated FDA Clinical Trial

- The IRB is the institutional review board responsible for the independent ethical review of biomedical research
- Level 1 trials are "Evidence Based Research" representing outcomes based on the most extensive criteria which minimizes bias
- Level 1 studies are randomized parallel group, doubleblind, placebo-controlled, multi-site studies that require rigorous quality control, resources and procedures
- They are considered the most reliable evidence of whether a treatment is effective

271

A double-blind, placebo-controlled randomized evaluation of the effect of the Erchonia® FX-635™on low back pain clinical study protocol

> LOW BACK PAIN CLINICAL STUDY RESULTS

ERCHONIA CORPORATION

January 15, 2018

(Study based on the ERCHONIA CORPORATION A double-blind, placebocontrolled randomized evaluation of the effect of the Erchonia® FX-635™on low back pain clinical study protocol: Version 4.2, January 7, 2017



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ASSOCIATION

Interesting Facts about Back Pain

- Worldwide, back pain is the single leading cause of disability, preventing many people from engaging in work as well as other everyday activities.2
- Back pain is one of the most common reasons for missed work. One-half of all working Americans admit to having back pain symptoms each year.3
- Back pain accounts for more than 264 million lost work days in one year—that's two work days for every full-time worker in the country.4
- Experts estimate that up to 80% of the population will experience back pain at some time in their lives.5
- Back pain can affect people of all ages, from adolescents to the elderly.5
- Back pain is the third most common reason for visits to the doctor's office, behind skin disorders and osteoarthritis/joint disorders.6
- Most cases of back pain are mechanical or non-organic—meaning they are not caused by serious conditions, such as inflammatory arthritis, infection, fracture or cancer.7
- Most people with low back pain recover, however reoccurrence is common and for small percentage of people the condition will become chronic and disabling.7
- Worldwide, years lived with disability caused by low back pain have increased by 54% between 1990 and 2015.7
- Low-back pain costs Americans at least \$50 billion in health care costs each year8—add in lost wages and decreased productivity and that figure easily rises to more than \$100 billion.9

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 the approximation of this material in whole or in part

	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 SD Mean 59.00 11.77 59.21 13.23 24.76 24.27 48.24 26.90 -34.24 25.94 -10.97 24.69

Table 17: Baseline and endpointVAS 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.

Mean VAS Low Back Pain Ratings for All Active Treatment Subjects Across Study Duration to 12 Months Follow-Up 59.96 37.61 26.91 VAS

1 Mth Post

Mean % Total Index Score on the ODI for Active Treatment Subjects Across Study Duration to 12 Months Follow-Up

2 Mths Post

12 Mths Post

0

15

10

Pre-Treatment

Pre-Treatment

Treatment End

Treatment End

ONE YEAR FOLLOW-UP 276

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

Form Approved: OMB No. 0910-0120277

erchonia

Expiration Date: 06/30/2020

See PRA Statement below.

Indications for Use

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 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 <u>ALL</u> NOCICEPTIVE MUSCULOSKELETAL PAIN CONDITIONS! ERCHONIA

CURRENT ERCHONIA RESEARCH PROJECTS AND COLLABORATIONS

Autism	Tinnitus	Neuropathy
Toe-Nail Fungus – Terbinafine Resistance	Erectile Dysfunction	Fat Loss with Mayo Diet EMERALD WITH MAYO DIET. LABS HBA1C, LIPIDS, CRP IL6 AND INSULIN PRE/POST
Low Back Pain with Biomarkers FX 405. PRE AND POST LABS A1A GLYCOPROTEINS, CRP HAPTOGLOBINS AND FULL CYTOKINE PANEL	Macular Degeneration	Severe spinal cord injuries



- 2002. Chronic neck and shoulder pain
- 2004. Post surgical pain following breast augmentation
- 2005. Acne Vulgaris
- 2008. Post surgical pain following liposuction
- 2010. Circumference reduction of the waist, hips and thighs
- 2012. Circumference reduction of the arms
- 2013. Circumference reduction waist, hips, thigh 532nm
- 2013. Cellulite

ERCHONIA

FDA CLEARANCES

- 2014. Chronic heel pain and plantar fasciitis
- 2014. Obesity circumferential reduction
- 2015. Zerona OTC (Over the Counter)
- 2016. Over all body indication for body contouring 635nm
- 2019. Chronic neck and shoulder pain 635nm & 405nm combo
- 2020. Over all body Non-Evasive Fat Loss for up to 40 BMI
- 2021. Overall musculoskeletal pain 635 & 405nm combo

ALL OTHER LASER COMPANIES COMBINED = 4

280

2015. Zerona EZ6 protocol 2016. Onychomycosis • 2018. Chronic lower back pain 2019. Overall musculoskeletal pain 635nm 2021. Post Operative Pain ERCHONIA FDA CLEARANCES = 22

SAFE AND EFFICACIOUS



1

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!



+ Supplemental content

Research

JAMA | Original Investigation

Effect of Opioid vs Nonopioid Medications on Pain-Related Function in Patients With Chronic Back Pain or Hip or Knee Osteoarthritis Pain The SPACE Randomized Clinical Trial

Erin E. Krebs, MD, MPH; Amy Gravely, MA; Sean Nugent, BA; Agnes C. Jensen, MPH; Beth DeRonne, PharmD; Elizabeth S. Gol/smith, MD, MS; Kurt Kroanka, MD: Matthew J. Bair; Sjamak Noorbaloochi, PhD

IMPORTANCE Limited evidence is available regarding long-term outcomes of opioids compared with nonopioid medications for chronic pain.

OBJECTIVE To compare opioid vs nonopioid medications over 12 months on pain-related function, pain intensity, and adverse effects.

DESIGN, SETTING, AND PARTICIPANTS Pragmatic, 12-month, randomized trial with masked outcome assessment. Patients were recruited from Veterans Affairs primary care clinics from June 2013 through December 2015; follow-up was completed December 2016. Eligible patients had moderate to severe chronic back pain or hip or knee osteoarthritis pain despite analgesic use Of 265 patients enrolled, 25 withdrew prior to randomization and 240 were randomized

INTERVENTIONS Both interventions (opioid and nonopioid medication therapy) followed a treat-to-target strategy aiming for improved pain and function. Each intervention had its own prescribing strategy that included multiple medication options in 3 steps. In theopioid group, the first step was immediate-release morphine, oxycodone, or hydrocodone/acetaminophen. For the nonopioid group, the first step was acetaminophen (paracetamol) or a nonsteroidal anti-inflammatory drug. Medications were changed, added, or adjusted within the assigned treatment group according to individual patient response

MAIN OUTCOMES AND MEASURES The primary outcome was pain-related function (Brief Pain Inventory [BPI] interference scale) over 12 months and the man secondary outcome was pain intensity (BPI severity scale). For both BPI scales (range, 0-10; higher scores = worse function or pain intensity), a 1-point improvement was clinically important. The primary adverse stcome was medication-related symptoms (patient-reported checklist; range, 0-19).

RESULTS Among 240 randomized patients (mean age, 58.3 years; women, 32 [13.0%]), 234 (97.5%) completed the trial. Groups did not significantly differ on pain-related function over 12 months (overall P = .58); mean 12-month BPI interference was 3.4 for the opioid group and 3.3 for the nonopioid group (difference, 0.1 [95% CI, -0.5 to 0.7]). Pain intensity was significantly better in the nonopioid group over 12 months (overall *P* = .03); mean 12-month BPI severity was 4.0 for the opioid group and 3.5 for the nonopioid group (difference, 0.5 [95% CI, 0.0 to 1.0]). Adverse medication-related symptoms were significantly more common in the opioid group over 12 months (overall P = .03); mean medication-related symptoms at 12 months were 1.8 in the opioid group and 0.9 in the nonopioid group (difference, 0.9 [95% CI, 0.3 to 1.5]).

CONCLUSIONS AND RELEVANCE Treatment with opioids was not superior to treatment with nonopioid medications for improving pain-related function over 12 months. Results do not support initiation of opioid therapy for moderate to severe chromic user the s osteoarthritis pain.

OPIOD GROUP PAIN SCALE WER³² FROM 54 TO 40 (14 POINTS) VS **ERCHONIA FX WENT FROM 61 TO 21 (40 POINTS)**

Disease Outcomes Researc olis Veterans Affairs Healt tem Minneanolis Minnesota

RESULTS DO NOT SUPPORT INITIATION OF **OPIATE THERAPY FOR MODERATE TO SEVERE CHRONIC BACK PAIN OR HIP OR KNEE OSTEOARTHRITIS PAIN**

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Affiliations Center fo

ely, Nugent, Jens oldsmith, Noorba

WHEREAS ALL OTHER LIGHT DEVICES FAILED...



Journal of Physiotherapy 🛢 (2020) 🛢 – 🛢



Journal of PHYSIOTHERAPY journal homepage: www.elsevier.com/locate/jphys

Research

Photobiomodulation therapy does not decrease pain and disability in people with non-specific low back pain: a systematic review

Shaiane Silva Tomazoni ^{a.b}, Matheus Oliveira Almeida ^a, Jan Magnus Bjordal ^b, Martin Bjørn Stausholm ^b, Caroline dos Santos Monteiro Machado ^{c.d}, Ernesto Cesar Pinto Leal-Junior ^{c.d}, Leonardo Oliveira Pena Costa ^a

³ Masters and Doctoral Programs in Physical Therapy. Universidade Cidade cé São Paulo, São Paulo, Brazil, ^b Physiotherapy Research Group, Department of Global Public Healts and Primary Care, University of Bergen, Bergen, Norway, 'Luboratory of Phototherapy and Innovative Technologies in Health, Nove de Julha University, São Paulo, Brazil, ^d Post-graduate Program in Rehabilization Sciences. Nove de Julha University, São Paulo, Brazil, ^e Post-graduate Program in Rehabilization Sciences. Nove de Julha University, São Paulo, Brazil

ABSTRACT

KEY WORDS

Low-level laser therapy Light emitting diode therapy Phototherapy Musculoskeletal disorders Rehabilitation Question: In people with non-specific low back pain (LBP), what are the effects of photobiomodulation therapy (PBMT) on pain, disability and other outcomes when compared with no intervention, sham PBMT and other rutations? Design: Systematic review of randomised trials with meta-analysis. Participants: People with acute/subacute or chronic non-specific LBP, Interventions: Anny type of PBMT (laser class I, II and III and light-emitting diodes) compared with no treatment, and when used as an adjunct to other treatment. Stam PBMT and ther types of treatment, usual when the set as an adjunct to other treatment. Jourcome measures: Pain intensity, disability, overall improvement, quality of Life, work absence and adverse effects. Results: Twelve randomised controlled trials were included (pooled n = 1/046). Most trials had low risk of blas. Compared with sham PBMT, the effect of PBMT on pain and disability was clinically unimportant in people with scienteliabouth or chronic (TBP, la conole with choose the treatment). Subsence and adverse effects. Results: Twelve randomised controlled trials were included (pooled n = 1/046). Most trials had low risk of blas. Compared with sham PBMT, the effect of PBMT on pain and disability was clinically unimportant in reamuer) and blass treatements or choose (TBP, la conole with choose the treatment. Concome constrained adverse declare treatment is an adverse to a treatment on constrained multiplance and adverse declare treatment is an adverse to a treatment on adverse treatment. Concome constrained adverse declare treatment and adverse declare treatment on adverse treatment on adverse treatment process and adverse declare treatment is an adverse to a treatment on adverse treatment on adverse treatment. Concome constrained adverse declare the treatment on adverse treatment on

IN SUMMARY. **ALL OTHER** FORMS OF LIGHT THERAPIES **OTHER THAN** THE ERCHONIA LASER. INCLUDING LED. **CLASS 3-4. IR/ NIR ETC HAVE** SHOWN IN **RESEARCH TO BE INEFFECTIVE** FOR LOW BACK PAIN!

Phototherapy Musculoskeletal disorders Rehabilitation

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

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 A∂ 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 (Figure 4). The cell bodies of neurons lie within the dorsal nerve root ganglion, but the elongated cytoplasm (axons) of the neurons extends from the cell body to the bare nerve endings in the surface of the skin. The direct effect of LLLT are initially at the level of the epidermal neural network, but the effects move to nerves in subcutaneous tissues, sympathetic ganglia, and the neuromuscular junctions within muscles and nerve trunks.

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SET IT AND FORGET IT



STANDARD BASE POSITION LOW BACK PAIN. MAX 20 MINUTES. 10 MINUTES AVERAGE



ÉRCHONIA

CURRENT BACK PAIN TREATMENT MODEL

OVERVIEW OF (LOW) BACK PAIN MANAGEMENT:

- 1. IS IT PERIPHERAL SENSITIZATION
- 2. UTILIZE ERCHONIA LASER DEVICES AS PART OF THE INITIAL TREATMENT TRIAL AND DIAGNOSTICS
- 3. APPLY PROLOGEL IF AVAILABLE
- 4. MAKE SURE THEY ARE PROPERLY OFFLOADING PARASPINAL MUSCLES AND PELVIC INSTABILITY WITH HIP EXTENSOR AND EXTERNAL ROTATION ACTIVATION (THE "GLUT CLAMSHELL LASER" PROTOCOL. MAKE SURE IT IS GLUT ISOLATED AND NOT RECRUITING PARASPINALS, QUADS, HAMSTRINGS...
- 5. ALWAYS GIVE AT LEAST 6 VISITS FOR LASER TREATMENT TRIALS, 12 FOR COMPLEX CONDITIONS LIKE CRPS
- 6. ASSUMING NO CONTRAINDICATIONS, REDUCE ANY BIOMECHANICAL FAULTS/SUBLUXATION SOURCES WITH CHIROPRACTIC CARE
- 7. ADDRESS THE PATIENTS INDIVIDUAL NEEDS AND GOALS
- 8. BASED ON THAT, RX HOME THERAPIES OF ACTIVITY THAT THE PATIENT WANTS TO PARTAKE IN
- 9. SEND THEM HOME WITH THEIR ACUTE/INITIAL TREATMENT PLAN KIT AND MUTUAL RECOMMENDATIONS. THE KIT SHOULD INCLUDE AN ERCHONIA HAND HELD LASER (RENTAL PROGRAM), RESISTANCE LAP BAND, BOTTLE OF PROLOGEL (RED LIDOCAINE VERSION IN SEVERE ACUTE CASES)
- 10. DAILY GLUT ISOMETRIC HOLDS SIDE LYING. WORK UP TO 20 MINUTES (10 MINUTES EACH SIDE, ONE MINUTE ON, ONE MINUTE OFF)
- 11.ONCE FINISHED GLUT ISO COMBO HAVE THE PATIENT LASER THE VAGUS NERVE AND PREFRONTAL CORTEX WHILE THEY VISUALIZE PERFORMING TASKS THAT THEY WANT TO DO WITHOUT PAIN, AND MEDITATIVE CBT FOR HAPPY INTROSPECTION
- 12. ENCOURAGE THEM TO GET POSITIVE REINFORCEMENT OF BASIC ADL'S (IE. IF IT USED TO HURT GETTING UP OFF OF THEIR FAVORITE CHAIR, HAVE THEM TRY IT AFTER DOING THEIR HOME LASER PROTOCOLS ABOVE)
- 13. AS THEY ESTABLISH POSITIVE REINFORCEMENT AND SAFTEY OF BASIC ADL'S, ENCOURAGE THEM TO PARTAKE IN PHYSICAL ACTIVITIES THEY ENJOY SUCH AS WALKING, HIKING, YOGA...WHATEVER THEIR FAVORITES ARE!

LOW BACK BACK PAIN PROTOCOLS





287

DYNAMIC LOW BACK PROTOCOL




ACUTE LOW BACK HOME PLAN







1. LASER RENTAL PROGRAM



XLR8 \$300/WK

EVKL \$400/WK

GVL \$500/WK





2. BOTTLE OF PROLOGEL IF NO CONTRAINDICATIONS TO LIDOCAINE USE RED FOR ACUTE PAIN





3. GIVE PATIENT RESISTANCE APPROPRIATE THERABAND FOR GLUT CLAMSHELLS

SUGGESTED PACKAGE PRICE FOR 1 WEEK RENTAL WITH XLR8, 1 BOTTLE OF PROLOGEL AND 1 BAND



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

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's Clinical Affairs Manager stated, "The latest 510(k) statement once again proves Erchonia's commitment to the science of low-level later therapy through blinded and gentralied clinical trials to obtain FDA 510(k) marketing statements. As the World Elevel France State France France France State Sta

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 118–136,91

GREEN LIGHT AND PAIN



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

Ibrahim, Mohab M. ...; Patwardhan, Amol...; Gilbraith, Kerry B.; Moutal, Aubin; Yang, Xiaofang; Chew, Lindsey A.; Largent-Milnes, Tally; Malan, T. Philip...; Vanderah, Todd W.ab; Porreca, Frankab; Khanna, Rajeshb

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 and 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 a single of the second s

GREEN LIGHT AND PAIN



<u>Research by Mary Heinricher</u> 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.

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PMCID: PMC4794405 NIHMSID: NIHMS743647 PMID: <u>26785323</u>

A possible neural mechanism for photosensitivity in chronic pain

Melissa E. Martenson,a

PAIN AND WAVELENGTHS



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

1 TREATMENT FOR NECK AND SHOULDER PAIN XRL8 VS EVRL

COMPARISON

ERCHONIA LASERS FOR PAIN



Medical Devices: Evidence and Research Dovepress open access to scientific and medical resea

Effects of a single treatment with two nonthermal laser

wavelengths on chronic neck and shoulder pain

Results: Among subjects treated with the red and violet lasers, mean VAS neck and shoulder pain scores decreased from 65.0 to 35.2 (p<0.0001). Most subjects in the study (75%) achieved ≥30% decrease in VAS scores. The decreased mean (SD) VAS scores remained 29.6 (16.7) and 29.3 (19.2) after 24 and 48 hrs, respectively. The secondary efficacy measures of change in range of motion ROM) and patient satisfaction also improved. There were no adverse events.

Conclusion: Overall, treatment with the red and violet lasers outperformed the FDAapproved red laser with respect to change in pain scores and improvement in shoulder ROM.



NMDA GLUTAMATE RECEPTORS AND 405 NM ON PAIN



Optical control of pain in vivo with a photoactive mGlu5 receptor negative allosteric modulator

Abstract

Joan Font, Marc López-Cano, Serena Notartomaso,

Light-operated drugs constitute a major target in drug discovery, since they may provide spatiotemporal resolution for the treatment of complex diseases (i.e. chronic pain). JF-NP-26 is an inactive photocaged derivative of the metabotropic glutamate type 5 (mGlu5) receptor negative allosteric modulator raseglurant.

Violet light illumination of JF-NP-26 induces a photochemical reaction prompting the active-drug's release, which effectively controls mGlu5 receptor activity both in ectopic expressing systems and in striatal primary neurons.

405 NM VIOLET LIGHT EFFECTIVELY TARGETS THE NMDA GLUTAMATE RECEPTOR SITES IN THE PERIPHERAL AND CENTRAL NERVOUS SYSTEMS MODULATING SENSITIZATION OF WDR'S AND OTHER EFFECTS OF GLUTAMATE EXCITOTOXICITY

EVRL ADVANCED PAIN APPLICATION





VIOLET 405 NM DIRECTLY OVER SPINAL CORD

RED 635 NM OVER ADJACENT NERVE ROOTS

SPAN MULTIPLE SEGMENTS



ERCHONIA

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%

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

COMPARISON

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PAIN AND WAVELENGTHS



RANGE OF MOTION (ROM)

Device	Degree improvement in shoulder ROM
XLR8	14.4°
EVRL	29.3°
GVL	31.9°

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PAIN AND WAVELENGTHS



SATISFACTION RATING

- All but 1 subject experienced improvement directly after treatment, after 24 hours post 100% subjects experienced improvement,

Device	Subjects that were Somewhat Satisfied or Very Satisfied
XLR8	89%
EVRL	89%
GVL	98%

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WATCH THE NEW KIRK GAIR GVL WEBINAR! THANK YOU DR. GAIR, DR. SILVERMAN AND DR. COMEY FOR THIS GREAT STUDY

	Red Only	Red & Violet	Green & Viole	DC
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%	
(%) Improvement in Pain from baseline to immediately after treatment	48%	45%	52%	
(%) Improvement in Pain from study endpoint to 48 hrs. post-treatment	43%	50%	65%	
(°) Improvement in Range of Motion]4º	29°	32°	

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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 <u>increased</u> <u>levels of activity in glial cells</u>, 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, PhD¹; Mark A. Lumley, PhD²; Roy H. Perlis, MD, MSc³; <u>et al</u> Conor Liston, MD, PhD²; Faith M. Gunning, PhD³; Tor D. Wager, PhD³ Author Affiliations Article Information

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CURRENT BACK PAIN TREATMENT MODEL



ERCHONIA

"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.

However, the study went even further. The research team in Frankfurt compared a third group of treatment methods alongside the standard and individualized ones. In this group, individualized training sessions were combined with cognitive behavioral therapy (CBT). This procedure – a type of talk therapy – is based on the assumption that negative thoughts and behaviors surrounding pain tend to exacerbate it. Through CBT, pain patients learn to change the way they handle it.

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 Behrgns and Daniel Niederer, **29 July 2022**, *Journal of Pain*. DOI: 10.1016/j.jpain.2022.07.005



without permission of Trevor Berry, DC, DACNB is prohibited. THE NEUROLOGY OF PAIN



DESCRIBE THE BASIC MECHANISMS OF NOCICEPTION AND PAIN:

- INJURY -> IMMUNE RESPONSE AND INFLAMMATION "SOUP"/NOXIOUS STIMULI
- INFLAMMATION CHEMICAL MESSENGERS BATH YOUR PERIPHERAL NERVE RECEPTORS. PRIMARILY THE CHANNELS (TRPV, TRPM, ASIC ETC) OF SLOW TYPE C NOCICEPTORS
- UPREGULATION OF SPINAL CORD MECHANISMS PRIMARILY WIDE DYNAMIC RANGE NEURONS USING EXCITATORY GLUTAMATE RECEPTORS (GLUT5 RECEPTOR) ***
- ASCENDING TRACTS BRING THE INFORMATION ROSTRAL TO THE BRAIN REGIONS PRIMARILY THROUGH THE SPINOTHALAMIC SYSTEM
- MUCH OF PAIN SUMMATES OR TERMINATES IN THE INSULAR AND BRAINSTEM REGIONS FOR "FIGHT OR FLIGHT, FEAR, MEMORY AND OTHER SURVIVAL RESPONSES". EX, RETICULAR FORMATION, CEREBELLUM, HYPOTHALAMUS, AMYGDALA AND CINGULATE REGIONS. (DO NOT UNDERESTIMATE pCBT TYPES OF THERAPIES IN CHRONIC PAIN SUFFERING)
- THESE PATHWAYS CONTINUE ON THE NEOCORTICAL REGIONS INCLUDING THE PARIETAL LOBE FOR LOCALIZATION (*) AND FRONTAL REGIONS FOR MOTOR RESPONSES



DO NOT FORGET SYSTEMIC SOURCES OF INFLAMMATION LIKE PATHOGENS, FOOD SENSITIVITIES, STRESS ETC.

PAIN MANAGEMENT



THE "DOWNSTREAM" APPROACH

ALWAYS GIVE YOURSELF AT LEAST 6 VISITS FOR BASIC MUSCOSKELETAL/PAIN CASES (12 FOR COMPLEX SUCH AS CRPS OR PHANTOM LIMB). IF YOU ARE NOT ACHIEVING OBVIOUS RESULTS WITHIN 3-4 VISITS, CONSIDER THE FOLLOWING...



THE MASTER PROTOCOL **NEUROMUSCULOSKELETAL**



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



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STROKE, CRPS, PHANTOM LIMB, DYSTONIA, FROZEN SHOULDER REHAB, POST-OP/IMMOBILIZE PROTOCOL BASE ALGORYTHM



PAGE 1

- 1. UPREGULATE THE VAGUS NERVE FOR 2 MINUTES BRAIN SETTING (1-10-40-60).
- 2. TRANSCRANIAL LASER BASE BRAIN SETTING 1-10-40-60 TO THE CORTEX OF THE INVOLVED SIDE (IE RIGHT MIDDLE CEREBRAL ARTERY OR INTERNAL CAPSULE INFARCT LASER RIGHT CORTEX). LASER POSITION CAN BE BIASED TOWARDS DESIRED REHAB (IE WHEN DOING LEFT HAND MOTOR REHAB LASER THE RIGHT FRONTAL LOBE). ALSO LASER THE CONTRALATERAL CEREBELLUM WITH BASE BRAIN SETTING (IE RIGHT CORTICAL = LEFT CEREBELLUM). WITH THE FX AND PLTOUCH THIS CAN BE DONE AT THE SAME TIME. PARIETAL LOBE IF PAIN BASED.
- 3. HAVE PATIENT PRE-VISUALIZE THE DESIRED MOVEMENT OF REHAB WITH THE EYES CLOSED. (IN PAIN SYNDROMS VISUAL MOVEMENT WITHOUT PAIN)
- 4. ACTIVATE THE FRONTAL LOBE WITH SACCADES (IE TO FIRE RIGHT FRONTAL LOBE USE LEFTWARD SACCADES. ANGLE UP AND AWAY FOR MORE LATERALIZATION).
- 5. MIRROR OVER THE TARGET LIMB SO THAT THE PATIENT HAS THE VISUAL PERSPECTIVE THAT IT IS THE INVOLVED LIMB BUT WILL ACTUALLY BE THE CONTRALATERAL "GOOD" LIMB.
- 6. HAVE THE PATIENT ACTIVELY MOVE THE GOOD LIMB IN WHATEVER DESIRED PATTERN YOU ARE TARGETING (IE ELBOW FLEXION). THE MIRROR IS PLACED IN A WAY THAT THE PATIENT IS LOOKING AT THE LIMB MOVING IN THE MIRROR AND THE BRAIN THINKS THE INJURED LIMB IS MOVING BUT IT'S ACTUALLY THE NON-INJURED LIMB. (NOTE: THIS CAN BE DONE FOR OTHER CONDITIONS LIKE CRPS, FROZEN SHOULDER, PHANTOM LIMB PAIN ETC).



MIRROR THERAPIES





STROKE, CRPS, PHANTOM LIMB, DYSTONIA, FROZEN SHOULDER REHAB, POST-OP/IMMOBILIZE PROTOCOL BASE ALGORYTHM



PAGE 2

7. REPEAT THE GOOD LIMB MOVEMENTS IN THE MIRROR WHILE THE PROVIDER OR FAMILY MEMBER MOVES THE INJURED LIMB BEHIND THE MIRROR FOR THE PATIENT PASSIVELY. LASER THE DISTAL TISSUE IN PAIN/POST-OP ETC. 8. SENSORY CO-ACTIVATION SHOULD BE APPLIED TO THE BAD SIDE DURING THIS

8. SENSORY CO-ACTIVATION SHOULD BE APPLIED TO THE BAD SIDE DURING THIS PROCEDURE. TENS, VIBRATION MASSAGE ETC CAN BE USED ON THE INJURED SIDE AT THE SAME TIME AS THE MOTOR/MIRROR REHAB.

9. IF THE PATIENT IS ABLE TO DEVELP NEUROPLASTICITY TO THE POINT OF GETTING THEIR OWN ACTIVE MOVEMENT ON THE INJURED SIDE YOU CAN START TO ADD ACTIVE THERAPY BEHIND THE MIRROR INSTEAD OF PASSIVE.

10. ONCE THEY ARE ABLE TO DO ACTIVE CARE, REMOVE THE MIRROR AND TRANSITION INTO "CROSS-CRAWL PATTERNS" (IE IF WORKING ON LEFT ELBOW FLEXION BEHIND THE MIRROR AND THE PATIENT IS ABLE TO START ACTIVATING THE LEFT ELBOW INTO FLEXION, REMOVE THE MIRROR AND HAVE THEM OBSERVE THE LEFT ELBOW INTO FLEXION WHILE THE RIGHT ELBOW GOES INTO EXTENSION. THIS CAN BE BUILT UP INTO FULL CROSS-CRAWL MARCHING). 11. OTHER CO-ACTIVATION MAY BE APPLIED SUCH AS HEMI-STIM VEP'S, OTHER SPECIAL SENSES (OLFACTION, AUDITORY ETC).

12. THIS MIRROR TECHNIQUE CAN BE DONE FOR ANY LIMB.



PAIN AND CNS NEUROCHEMISTRY





THE ALAMO FOR CHRONIC PAIN, ADDICTION, NEUROPSYCHOLOGY, PTSD AND BEHAVIORAL MODIFICATION: THE CLASSIC PSYCHEDELICS PSILOCYBIN LSD AYAHUASCA IBOGAINE MDMA

*THESE ARE SCHEDULE 1 SUBSTANCES AND, WITH A FEW EXCEPTIONS, ARE ILLEGAL IN THE USA. LEGAL APPLICATIONS AND A CONTROLLED ENVIRONMENT (SET AND SETTING) MUST BE CONSIDERED BEFORE UTILIZATION!

PAIN AND CNS NEUROCHEMISTRY



Randomized Controlled Trial

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

D W Barrett 1, <u>F Gonzalez-Lima</u> Abstract

This is the first controlled study demonstrating the beneficial effects of transcranial laser stimulation on cognitive and emotional functions in humans. Photobiomodulation with red to

near-infrared light is a novel intervention shown to regulate neuronal function in cell cultures, animal models, and clinical conditions. Light that intersects with the absorption spectrum of cytochrome oxidase was applied to the forehead of healthy volunteers using the laser diode CG-5000, which maximizes tissue penetration and has been used in humans for other indications. We tested whether low-level laser stimulation produces beneficial effects on frontal cortex measures of attention, memory and mood. Reaction time in a sustained-attention psychomotor vigilance task (PVT) was significantly improved in the treated (n=20) vs. placebo control (n=20) groups, especially in high novelty-seeking subjects. Performance in a delayed match-to-sample (DMS) memory task showed also a significant improvement in treated

vs. control groups as measured by memory retrieval latency and number of correct trials. The Positive and Negative Affect Schedule (PANAS-X), which tracks self-reported positive and negative affective (emotional) states over time, was administered immediately before treatment and 2 weeks after treatment. The PANAS showed that while

participants generally reported more positive affective states than negative, **overall affect improved**

significantly in the treated group due to more sustained positive emotional states as compared to the placebo control group. These data imply that transcranial laser stimulation could be used as a non-invasive and efficacious approach to increase brain functions such as those

related to cognitive and emotional dimensions. Transcranial infrared laser stimulation has also been proven to be safe and successful at improving neurological outcome in humans in controlled clinical trials of stroke. This innovative approach could lead to the development bitation invitative, gedistribution of the development of the

PAIN AND CNS NEUROCHEMISTRY



Published online 2018 Jan 11. doi:PMCID: PMC6041198NIHMSID: NIHMS934240PMID:

Brain Photobiomodulation Therapy: A Narrative Review

Effects on intrinsic brain networks

In the brain, a collection of distant but integrated structures provide widespread neuronal connections, which are called "intrinsic brain networks". The default mode network (DMN), salience network (SN) and central executive network (CEN) are the most important examples of these formations. These networks are not only activated upon stimulation by neural inputs, but also their activities are detectable even in the resting state [174]. This may indicate that cerebral networks through their dynamic activities and anatomical connectivity work together to regulate intrinsic brain activity [175]. Besides, these intrinsic networks are able to modulate higher levels of cognitive and emotional functions [176]. Both chronic neurodegenerative disease and acute

brain insults, cause an imbalance in the activity of these networks [177,178]. For instance, in TBI patients, abnormalities in higher level cognitive activities are associated with weak connections within and between the DMN, SN and CEN nodes, resulting in impaired dynamic interactions of these networks [179,180]. There is a hypothesis that the

geographical matching of light irradiation sites on the head, with the corresponding anatomical regions of intrinsic networks within the brain may allow re-establishment of these functions, and may have enhanced therapeutic benefit [174]. In this respect, Naeser et al. [30]

reported findings in TBI patients who received *transcranial therapy over the DMN, SN, and CEN* nodes and displayed enhanced cognitive functions, likely through the augmentation of metabolic capacity in these intrinsic networks. Moreover, Naeser et al

proposed that the ability of PBM to reduce PTSD symptoms might stem from the modulation of DMN and SN activities [30]. On the

other hand, application of PBM in stroke patients with aphasia demonstrated neurotherapeutic efficacy through the stimulation of cortical nodes within the CEN network [181].

LASERS A - Z MANUAL **JEROME RERUCHA**



BASE MUSCULOSKELETAL/PAIN DEFAULT UPREGULATION

9-16-42-53

TENDONITIS 1-21-250-2720

9-16-33-36

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 **NEUROGENIC INFLAMMATION/FASCIITIS**

WOUNDS 3-16-24-111



LOWER EXTREMITY





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SECTION 1 | PRACTICAL LAB | LOWER EXTREMITIES







PERCUSSOR, MOTOR

FX LE PROTOCOL BASIC





PERIPHERAL NEUROPATHY ETIOLOGIES



COMMON DIFFERENTIALS FOR PERIPHERAL NEUROPATHY:

-DIABETIC NEUROPATHY. COMMON LABS: HBAIC, FASTING GLUCOSE, C-PEPTIDE

-B VITAMIN/METHYL DONOR DEPLETION COMMON LABS: CBC WITH DIFF, HOMOCYSTEINE, MICRONUTRIENT PANEL, MMA ELEVATION (LOW B12), GENETICS LIKE MTHFR (VIBRANT NUTRIPRO)

-STATINS

-GLUTEN SENSITIVITY/TRANSGLUTAMINASE 6 COMMON LABS: WHEAT ZOOMER (VIBRANT)

-CHEMOTHERAPY INDUCED

-VITAMIN B6 TOXICITY

-EMF EXPOSURE

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without permission of Trevor Berry, DC, DACNB is prohibited. BLOOD SUGAR SUPPORT





ERCHONIA LLLT: 9-20-53-733 LIVER ADRENALS PANCREAS 1 MINUTE EACH

FOR PERIPHERAL NEUROPATHY/MSK 9-16-42-53 BASE PAIN OR 2-2-2-2

VIBRANT LAB CONSIDERATIONS: FASTING GLUCOSE <100-126+ LIPID PANEL/ CARDIOMETABOLIC PROFILE HBAIC (<5.6) C-PEPTIDE

BERGACOR PLUS BERBERINE w/IN SEA BERBERINE ES5 LEPTIN MANAGER XYMOGEN Formulas

OTHER CLINICAL CONSIDERATIONS: CALORIC RESTRICTION/INTERMITTENT FASTING "MOTHER NATURE" OR "KETOGENIC" DIETS EX얟위CISE!!! HIIT/CARDIO >23MIN/COMPLEX

RESTLESS LEG SYNDROME (WILLIS-EKBOM DISEASE, RLS) CLINICAL CONSIDERATIONS



-RULE OUT ANEMIA PATTERS ESPECIALLY IRON DEFICIENCY: #1 ENVIRONMENTAL TRIGGER (RUN CBC AND FULL IRON PANEL) -CONSIDER OTHER MICRONUTRIENT DEPLETION ESPECIALLY MAGNESIUM (RUN MICRONUTRIENT PANEL) -WATCH PROTON PUMP INHIBITORS/H2 BLOCKERS/ACID BLOCKERS (CAUSE CO-FACTOR DEPLETION AND OTHER POSSIBLE MECHANISMS) SEE STUDY BELOW. SUMMARY: THESE MEDS DOUBLE THE RISK OF RLS! -WATCH DOPAMINE (ARE THEY ON A MEDICATION DEPLETING DOPAMINE, PARKINSONIAN RELATED ETC)

> NUTRITION CONSIDERATIONS FOR RLS: IRON GLYCINATE (IF LAB OR FUNCTIONALLY LOW SERUM IRON) OPTIMAG NEURO (MAGTEIN, MALATE, GLYCINATE) OMEGA 3 FATTY ACIDS 3/DAY A GOOD MULTIVITAMIN (EX, VIT C HELPS IRON ABSORPTION) CALMING AGENTS LIKE VALERIAN, CHAMOMILE, EPSOM BATHS L-THEANINE (IMPROVES CALMING & SLEEP PATTERNS) DOPAMINE AGONISTS AS INDICATED CBD (FORTIFY ELITE 1-2 PM 25-50 MG) OR HEMP MONOPURE



TREATMENT CONSIDERATIONS FOR RLS: IN OFFICE - MASTER BRAIN SETTING FULL BRAIN/VAGAL PROTOCOL (1-10-40-60) FX PFC/VAGAL/CB 10 MIN 635, 6 MIN 405 STACK WITH GLUT ISOMETRICS (TBAND CLAMSHELL 30 SECOND HOLDS X3-4) WHILE DOING MULTIPLE SETS OF ANTI-SACCADES SWITCH LASER TO FULL LOWER EXTREMITY EXPOSURE (SEE LAST SLIDE) 5 MINUTES EACH LUMBOSACRAL PLEXUS-KNEE/BIFURCATION-TARSAL TUNNEL/PLANTAR

Association of proton pump inhibitor and histamine H-receptor antagonists with restless legs

syndrome

Eric J Earley, Maria Didriksen, Bryan R Spencer, Joseph E Kiss, Christian Erikstrup, Ole B Pedersen, Erik Sørensen, Kristoffer S Burgdorf, Steven H Kleinman, Alar E Mast Sleep, Volume 44, Issue 4, April 2021 without permission of Trevor Berry, DC, DACNB is prohibited.

DIABETIC PERIPHERAL NEUROPATHY



Diabetes Metab Syndr. 2019 Mar - Apr;13(2):1087-1091. doi: 10.1016/j.dsx.2019.01.022. Epub 2019 Jan 18.

Effect of Low Level Laser Therapy on serum vitamin D and magnesium levels in patients with diabetic peripheral neuropathy - A pilot study.

<u>Anju M1, Chacko L1, Chettupalli Y</u>1, <u>Maiya AG</u>2, <u>Saleena Ummer V</u>3. Author information

Abstract

BACKGROUND:

Diabetic Peripheral neuropathy (DPN) is the most distressing complication of diabetic population leading to loss of sensation, pain, and amputation. Low-level laser therapy (LLLT) has been used to manage nerve injuries as it holds the potential to induce a biostimulatory effect with no side effects. Hence we planned to study the biochemical effect and therapeutic outcomes of LLLT on patients with painful diabetic peripheral neuropathy as a preliminary work.

MATERIALS AND METHODS:

Pre-posttest analysis was done on 40 patients diagnosed with DPN confirmed using 10 g Monofilament test and Michigan Neuropathy Screening Instrument (MNSI). Vibration sensation and pain measured by Vibration perception threshold (VPT) and Numeric pain rating scale (NPRS). All patients were given LLLT (3.1 J/cm2) on plantar and dorsal of the foot for 10 days. Serum samples were collected at baseline and 4 weeks after LLLT to estimate Vitamin D and Magnesium and compared the results. RESULTS:

There was a significant increase in Vitamin D and Magnesium levels after LLLT. We observed a considerable improvement in the quality of life after LLLT demonstrated by a decrease in VPT(VIBRATION PERCEPTION THRESHOLD) and MNSI and a reduction in NPRS (PAIN SCALE) in DPN patients.

CONCLUSION:

In this study, we found that LLLT improved the QL and hence may be a useful therapeutic option in treating peripheral neuropathic pain in type 2 diabetic patients. The progress in the serum Magnesium and Vit. D levels were proportional to the QL and may be a good indicator of the prognosis of DPN³ after LLLT.

PERIPHERAL NERVES



Effects of Electrical Stimulation at Different Frequencies on Regeneration of Transected Peripheral Nerve

December 2007<u>Neurorehabilitation and Neural Repair</u> 22(4):367-73

Abstract and Figures

Electrical stimulation of damaged peripheral nerve may aid regeneration. The purpose of this study was to determine whether 1 mA of percutaneous electrical stimulation at 1, 2, 20, or 200 Hz augments regeneration between the proximal and distal nerve stumps. A10-mm gap was made in rat sciatic nerve by suturing the stumps into silicone rubber tubes. A control group received no stimulation. Starting 1 week after transection, electrical stimulation was applied between the cathode placed at the distal stump and the anode at the proximal stump every other day for 6 weeks. Higher frequency stimulation led to less regeneration compared to

lower frequencies. Quantitative histology of the successfully regenerated nerves revealed that the groups receiving electrical treatment, especially at 2 Hz,

had a more mature structure with a smaller cross-sectional area, more myelinated fibers, higher axon density, and higher ratio of blood vessel to total nerve area compared with the controls. Electrophysiology showed significantly shorter latency, longer

duration, and faster conduction velocity. Electrical stimulation can have either a positive or negative impact on peripheral nerve regeneration. Clinical trials that combine stimulation with rehabilitation must determine the parameters that are most likely to be safe and effective.

SET ALL DIODES AT 2 HZ AND APPLY TO TARGET PERIPHERAL NERVE RE-CHECKING MOTOR/SENSORY TESTS, NCV/SSEP ETC

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PERIPHERAL NERVES



Bioengineering (Basel). 2018 Jun 9;5(2). pii: E44. doi: 10.3390/bioengineering5020044. Photobiomodulation Therapy (PBMT) in Peripheral Nerve Regeneration: A Systematic Review. Rosso MPO1, Buchaim DV2,3, Kawano N4, Furlanette G5, Pomini KT6, Buchaim RL7,8. Author information

Abstract

Photobiomodulation therapy (PBMT) has been investigated because of its intimate relationship with tissue recovery processes, such as on peripheral nerve damage. Based on the wide range of benefits that the PBMT has shown and its clinical relevance, the aim of this research was to carry out a systematic review of the last 10 years, ascertaining the influence of the PBMT in the regeneration of injured peripheral nerves. The search was performed in the PubMed/MEDLINE database with the combination of the keywords: low-level laser therapy AND nerve regeneration. Initially, 54 articles were obtained, 26 articles of which were chosen for the study according to the

inclusion criteria. In the qualitative aspect, it was observed that PBMT was able to accelerate the

process of nerve regeneration, presenting an increase in the number of myelinated fibers and a better lamellar organization of myelin sheath, besides improvement of electrophysiological function, immunoreactivity, high functionality rate, decrease of inflammation, pain, and the facilitation of neural regeneration, release of growth factors, increase of vascular network and collagen. It was concluded that PBMT has beneficial effects on the recovery of nerve lesions, especially when related to a faster regeneration and functional improvement, despite the variety of parameters.

> CLINICAL CONSIDERATIONS: 2X/WEEK 2-2-2-2 SETTING 10 **MINUTES 8-12 VISITS (4-6 WEEKS) YOU SHOULD OBTAIN** SUBJECTIVE AND OBJECTIVE CHANGES DEPENDING ON SEVERITY OF INJURY AND HOW OLD THE INJURY IS. **CONSIDER NERVE ROOT UPREGULATION AND AT TARGET PERIPHERAL NERVE(S).** 333



GREEN WAVELENGTH



ERCHONIA

UUT

ORIGINAL ARTICLES

A LIGHT SOURCE FOR PHOTOTHERAPY

R. Lubart⁹, S. Rochkind¹, U. Sharon² and M. Nissan³ of Physics, Bar-Ban University, Israel, (Departments of Neurosurgery, Ichilov Hospital, Israel, «Pau Engineering Ltd., Frand, &Technion, Haifa, Israel

Kty words Low power light source Peripheral net (CAP) Porphyrads

ens and other irradiation sources light are used both clinically and throughout the world, but there are we results us to the beneficial effects surces.¹ The effect of light upon the es has been dealt with by various a Ren et al.² sectioned and sutured temporary suppression of clonus in appearance of laser-induced social botentials, using 1 mW HeNe pul g 0.5 Juen² transcutaneously, eported a fuilure in confirming Uso Basford⁵ did not find 1 mW He direct social particular social social social bits of the social social social social social social social bits of the social social social social social social social bits of the social social social social social social social bits of the social ing Walker's WHeNe laser al sensory latencies action potentials, and Currier⁹ used pulsed 1.R. laser d were unable to detect any change in

the best annow the effects of low power ap is working on the effects of low power nervous system. We performed most of us tests using HeNe laser (632.8 nm) as a detail in our previous papers.⁹ We a 1 mW HeNe laser cannot reach the to be present in every cell

Addressee for correspondence: Dr R. Lubart, Physics Department, Har-Han University, Ramat-Gan 52100, Israel.

0898-5901/91/010015-03\$05.00 (C) 1991 by John Wiley & Sons, Ltd.

The present work represen-to the effect of different w nerves. According to these re-from our understanding of h we propose a new light sour

Materials and Methods

The present study was carried of Sprague Dawley kind. The rats and the sciatic nerve was expose the norm CAP me this p

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Lubart et al examined the effects of red (632 nm), green (540 nm) and infrared (904 nm) light sources on the peripheral nervous system18.

No significant difference was found for the action potential of the nerve with the infrared and control group, which means the infrared light affects the nerve minimally or not at all.

Only the red and green wavelengths had an effect on the compound action potential (CAP) of the nerve, with green light much more effective than the red.

GREEN WAVELENGTH



INJURED NERVES

The continuation of this work with different wavelengths, showed that green 540 nm is more effective than 632 nm.

ADD THE GREEN WAVELENGTH OF THE GVL TO YOUR PERIPHERAL NERVE APPLICATIONS! CONSIDER 2-2-2-2 HZ FREQUENCY SETTING



REVIEW

New trend in neuroscience: Low-power laser effect on peripheral and central nervous system (basic science, preclinical and clinical studies)

Semion Rochkind and Georges E. Ouaknine

Department of homesingens, in Prive Storadig Meddel Center, Tarkwa University, Load The present review summarizes findings in a correlating study of the use of low-power later present review summarizes findings in a correlating study of the use of low-power later that the study of the study of the study of the study finding and the study of study of the study of the study of the study of the study of study of study of the study of the study of the study of study of the study of the study of the study of study of the study of the study of the study of the study of study of the study of s

Keywords: Low-power laser irradiation, peripheral nerve and spinal cord injury, spinal cord and brain transplantation. tethered spinal cord

Compendence and report requests to: D: Semian Richland, Department of Neurosurgery, Ichilov Hospital, Tel-Aviv Sourasky Medical Center, & Wersman Street, Tel-Aviv 6429, Izrael Accepted for publication July 1991.

Neurological Research, 1992, Volume 14, Marc

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...

LASERS FOR ALL TYPES OF PRACTITIONERS AND IN THE NEWS



Jennifer Lin 16h · 📀

Did anyone else watch Selma Blair dance on DWTS and wonder how she was able to move? It's awesome knowing that she credits the Erchonia lasers for helping her heal. It's so important for mainstream America to know that these tools exist and that you don't always need drugs or surgeries to manage pain. I added 5 lasers to my medical practice 7 months ago and have been grateful that they've allowed a 5 year-old autistic boy to speak real words for the first time, a gentleman with chronic plantar fasciitis walk pain-free for the first time in a decade, a woman with bone on bone knee pain tell me she was walking stadium stairs and standing for 8 hours without pain. Light energy works!



dailymail.co.uk Video: Selma Blair gets laser treatment for painful knee ah...

HEADACHES





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GREEN LIGHT AND HEADACHE PAIN



In conclusion, the authors reported that "Green light is least likely to exacerbate migraine headache and at low intensities it may even be therapeutic by reducing the headache intensity."1

A later study, published in 2018 in the journal Pain, took a deeper dive into light sensitivity in migraines, also with a focus on comparing the effects of different colors of light on migraines. The authors reported that approximately 80% of migraine patients experienced exacerbation of their symptoms in response to white, blue, amber, and red lights, while green light exacerbated headaches 40% of

the time. The same study reported that exposure to light-initiated headaches about 16% of the time, and that green light therapy initiated headaches about 3% of the time2.

References:

1. Noseda R, Bernstein CA, Nir RR, Lee AJ, Fulton AB, Bertisch SM, et al. Migraine photophobia originating in cone-driven retinal pathways. Brain. 2016;139(Pt 7):1971-1986.

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GREEN LIGHT AND HEADACHE PAIN



Long-time sufferers of migraines and other chronic pain conditions may benefit from exposure to green light. A new study, led by pharmacologist Mohab M. Ibrahim, M.D., Ph.D., found that the color green may be key to easing pain.

Green light changes the levels of serotonin and alters the endogenous opioid system, an innate pain-relieving system found throughout the central and peripheral nervous system, gastrointestinal tract and immune system, said Bing Liao, M.D., a neurologist at Houston Methodist Hospital.

After completing the green light therapy, their score dropped down to an average of 2.8. The frequency of headaches dropped from 19 to 6.5 per month, and overall quality of life climbed from 48 percent to 78 percent.

Ibrahim exposed 25 migraine volunteers first to white lights for two hours as a control, then to green LED lights. He measured multiple parameters, including pain reduction, frequency of migraines or headaches, frequency of fibromyalgia flare-ups, pain intensity and duration, and quality of **##**fe.

GREEN LIGHT AND HEADACHE PAIN



Discussion

Green light emitting diodes significantly reduced the number of headache days in people with episodic migraine or chronic migraine. Additionally, green light emitting diodes significantly improved multiple secondary outcome measures including quality of life and intensity and duration of the headache attacks. As no adverse events were reported, green light emitting diodes may provide a treatment

option for those patients who prefer non-pharmacological therapies or may be considered in complementing other treatment strategies.

Research article

First published online September 9, 2020

Evaluation of green light exposure on headache frequency and quality of life in migraine patients: A preliminary one-way cross-over clinical trial

Laurent F Martin https://orcid.org/0000-0001-7358-5335, Amol M Patwardhan, [...] View all authors and affiliations Volume 41, Issue 2

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THE NEW GVL ADVANCED PROTOCOL

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-QU ZHANG HTTPS://ORCID.ORG/0000-0001-6623-9629 <u>Authors Info & Affiliations</u> SCIENCE TRANSLATIONAL MEDICINE

7 Dec 2022 Vol 14, Issue 674

Abstract

Green light exposure has been shown to reduce pain in animal models. Here, we report a vision-associated enkephalinergic neural circuit responsible for green light-mediated analgesia. Full-field green light exposure at an intensity of 10 lux produced analgesic effects in healthy mice and in a model of arthrosis. Ablation of cone photoreceptors completely inhibited the analgesic effect, whereas rod ablation only partially reduced pain relief. The analgesic effect was not modulated by the ablation of intrinsically photosensitive retinal ganglion cells (ipRGCs), which are atypical photoreceptors that control various nonvisual effects of light. Inhibition of the retino-ventrolateral geniculate nucleus (vLGN) pathway completely abolished the analgesic effects. Activation of this pathway reduced nociceptive behavioral responses; such activation was blocked by the inhibition of proenkephalin (Penk)-positive neurons in the vLGN (vLGNPenk). Moreover, green light analgesia was prevented by knockdown of *Penk* in the vLGN or by ablation of vLGNPenk neurons. In addition, activation of the projections from vLGNPenk neurons to the dorsal raphe nucleus (DRN) was sufficient to suppress nociceptive behaviors, whereas its inhibition abolished the green light analgesia. Our findings indicate that cone-dominated retinal inputs mediated green light analgesia through the vLGNPenk-DRN pathway and suggest that this signaling pathway could be exploited for reducing pain.

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

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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 S-HT₃ receptor antagonists (e.g. ondansetron) Fenfluramine Ergotamine/methysergide Hallucinogens

e organ systems

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



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HEADACHES

SUPRATROCHLEAR NERVE





© 2021 All Rights Reserved | Please do not copy or redistribute without written consent of the presenters. ERCHONIA **HEADACHES 2 MINUTES ERCHONIA INFRATROCHLEAR** (GVL) HAND HELD **NERVE AND TRIGEMINAL** 1-10-40-60 **GANGLIA** Supraorbital nerve Supratrochlear nerve TRIGEMINAL GANGLION 100 Supraorbital nerve Supratrochlear nerve Occipital artery Infraorbital Infraorbital nerve nerve Greate occipital nerve Lesser Mental occipital nerve nerve Sternocleidomastoid Great-Mental nerve auricular muscle Supraclavicular nerve nerve CONSIDER MASTICATION RELEASE TECHNIQUES DURING THIS TIME (INTERNAL AND EXTERNAL) © 2021 All Rights Reserved | Please do not copy or redistribute without written consent of the presenters.

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COMBINE ERB'S POINT LLLT EXPOSURE WITH VAGAL NERVE STIMULATION

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

GVL FOR PAIN MANAGEMENT



Dr. Berry,

I thought you'd be interested in the study that I have attached below, and yet may be more interested in the results from application of learnings on a patient.

I received and read the study this morning and applied the learnings to a patient. He has ankylosing spondylitis and has had a low back surgery with a fusion that eliminated the radicular leg pain. Since the surgery he has had significant left sacroiliac pain as well as a broad, banded residual low back pain. In addition, he came in this morning with R leg radicular pain to the outside of the R foot. These three areas had pain as follows:

Area	Pre Tx. Pain VAS	Pain VAS-After GVL 3
minutes over closed eyes		
LSI	+2	+.5
R Low back pain with radiculopat	hy to foot +1-2	0 pain and no radicular
Broad banded LB pain	+2-3	+.5

I had sent you a previous study about birds/magnatite crystals and light activation in one eye. That's why this one caught my attention.

Sincerely,

Jim Hollingsworth, DC, ND

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HEADACHES



- 1. REDUCE ANY BIOMECHANICAL DYSFUNCTION, ESPECIALLY OF THE UPPER CERVICAL REGION. CHIROPRACTIC MANIPULATION!
- 2. IF MANUAL THERAPIES ARE NOT "HOLDING", ADDRESS UNDERLYING NEUROGENIC CAUSES. VESTIBULAR DYSFUNCTION/IMBALANCE, OCULOMOTOR IMBALANCE OR OTHER NEUROLOGICAL COMPENSATION CAUSES. (GAZE STABILITY, BROCK BEADS ETC)
- 3. DON'T FORGET OBVIOUS TRIGGERS OF DIET AND LIFESTYLE. DO THEY SLEEP ON THEIR STOMACH WITH HEAD TURNED, LACK OF WATER OR OTHER NUTRIENTS. WATCH ALCOHOL. CAFFEINE WITHDRAWAL OR FASTING. DRINK EVEN MORE WATER!
- 4. LABS: HORMONE IMBALANCE, ANEMIA PATTERNS ETC.
- 5. MINIMIZE STRESS, COMPUTER USE AND OTHER ENVIRONMENTAL TRIGGERS LIKE BLUE/INCANDESCENT LIGHT EXCESS.
- 6. APPLY TOPICALS TO THE AREAS INDICATED ON SLIDES.
- 7. 2 MINUTES ERCHONIA GVL LOW LEVEL LASER TO EACH OF THOSE REGIONS USING MASTER BRAIN SETTING 1-10-40-60
- 8. ADD 30 SECONDS OF GREEN "WISPING" OVER THE RETINA
- 9. MOST HEADACHES WILL RESPOND RAPIDLY EVEN WITHIN THE OFFICE VISIT BUT ALWAYS GIVE YOURSELF® A 6 VISIT TREATMENT "TRIAL".

HEAD AND FACE PAIN





Duplic

BILL GEED TRIGEMINAL PAIN EXACERBATED BY INFECTED TOOTH AND DENTAL REMOVAL 10/25/22



HEAD AND FACE PAIN



ERCHONIA

Hi Doc

3/8/2021 MILITARY VET PTSD AND POST-CONCUSSION

For some time now I have been remiss in expressing my everlasting gratitude and respect, from my early difficulties to the present day.

It has been an arduous journey indeed. Apart from my neck, shoulder and back injuries, I also suffered from 'brain fog'.

Admittedly I was somewhat skeptical as to how efficacious these "Laser Treatments" might be. To my astonishment, for the first time in years, I began to think more clearly. I experienced a fresh and new approach that harkened back to an earlier time in my life. This was something I thought I had lost.

Thanks to you Doctor, I am a man reborn.

To all skeptics, I am now a believer. I beseech all of you. Give the "Laser Treatments" a chance.

Most Respectfully Bill Geed U.S.M.C. 11/62-11/66

10/25/22

"YOUR VIOLET/RED LASER WAS A GAME CHANGER FOR MY TRIGEMINAL NEURALGIA. AFTER AN INFECTED TOOTH AND MULTIPLE SURGICAL PROCEDURES IT WAS BACK WITH A VENGEANCE. I TRIED THEIR MEDICATIONS, ICE AND EVERYTHING ELSE THEY RECOMMENDED TO NO AVAIL. ONE TREATMENT WITH YOUR NEW GREEN/VIOLET DEVICE₅AND I AM A NEW MAN!" BILL GEED

CHEMO BRAIN

CHEMO BRAIN:

ERCHONIA TRANSCRANIAL LASER 1-10-1-10 (2-10 MINUTES DEPENDING ON THEIR METABOLIC CAPACITY AND FATIGUE RATIOS)

NUTRTION SUPPORT

LOW SUGAR DIET. PROMOTE KETOSIS (MCT OILS) HIGH DOSE OMEGA 3 FATTY ACIDS MINMUM 3 GRAMS PER DAY (XYMOGEN HEMP MONOPURE IN PM, AND DHA MONOPURE AM RECOMMENDED) S-ACETYL GLUTATHIONE AND NAC AM AND PM OTHERS: VINPOCETINE, GINKGO BILOBA, BACOPA MONNIERI, PHOSPHATIDYLSERINE (ALL ARE IN XYMOGEN'S MEMORALL!)

NAD+ through NAD+ precursors, such as NMN (Nicotinamide mononucleotide) or Nicotinamide riboside Methylene blue 40-50 mg with 250 ml 5% dextrose over 45 minutes (CI: PREGNANCY, NEUROPSYCH MEDS)

*ALWAYS WATCH CONTRAINDICATIONS AND INTERACTIONS OF SUPPLEMENTS WITH CHEMOTHERAPY DRUGS, A GOOD REFERENCE IS "NATURALMEDICINES.COM" without permission of Trevor Berry, DC, DACNB is prohibited.

without permission of Trever Berry, DC, DACNB is prohibited TRIGEMINAL NEURALGIA



Methods: For two months, participants in the control group A received the medication only (oral hypoglycemic drugs, Analgesics, vitamin B12), participants in the study group B received the medications as in group A in addition to LLLT, and participants in the study group C received medication as in group A in addition to electromagnetic therapy (EMT). The primary outcome was the amplitude of compound muscle action potentials of temporalis and masseter muscles by using NEXUS 10 (Mind media). The secondary outcome was pain intensity by using the Visual Analog Scale (VAS).

Results: According to the results of this study, there is a statistically significant difference in visual analog scale scores and the amplitude of compound muscle action potentials of the temporalis and masseter muscles among groups in favor of group B.

conclusions: After treatment, all groups improved significantly, with the laser group outperforming the electromagnetic group by a large margin. For irradiation, LLLT was more effective than EMT in reducing diabetic patients' trigeminal pain, and increasing masseter and temporalis muscles compound action potential amplitude in diabetic patients with TN.

Randomized Controlled Trial

Eur J Phys Rehabil Med

2023 Apr;59(2):183-191. doi: 10.23736/S1973-9087.23.07501-9. Epub 2023 Feb 10.

Effect of electromagnetic therapy versus low-level laser therapy on diabetic patients with trigeminal neuralgia: a randomized control trial



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

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

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 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





ps://doi.org/10.33 This article is part of the Research Topic Towards an Understanding of Tinnitus Heterogeneity, Volume II

A randomized single-blind controlled trial of a prototype digital polytherapeutic for tinnitus

Results: Mean changes in TFI for the USL group at 6 (16.36, SD 17.96) and 12 weeks (17.83 points, SD 19.87) were clinically meaningful (>13 points reduction), the mean change in WN scores were not clinically meaningful (6 weeks 10.77, SD 18.53; 12 weeks 10.12 points, SD 21.36). A statistically higher proportion of USL participants achieved meaningful TFI change at 6 weeks (55%) and 12 weeks (65%) than the WN group at 6 weeks (33%) and 12 weeks (43%). Mean TFI, rating and COSIT scores favored the US group but were not statistically different from WN. Usability measures were similar for both groups.

After 20 years searching for a cure for tinnitus, researchers at the University of Auckland are excited by 'encouraging results' from a clinical trial of a mobile-phone-based therapy. The study randomised 61 patients to one of two treatments, the prototype of the new 'digital polytherapeutic' or a popular self-help app producing white noise.

Key to the new treatment is an initial assessment by an audiologist who develops the personalised treatment plan, combining a range of digital tools, based on the individual's experience of tinnitus.

"This is quicker and more effective, taking 12 weeks rather than 12 months for more individuals to gain some control." WATCH FOR THIS TO LAUNCH IN 2023

THE MASTER PROTOCOL **NEUROMUSCULOSKELETAL**



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 THE CITY OVER A FOUNDON IS HERE OF RECOMPART without permission of Trevor Berry, DC, DACNB is prohibited.



HANDS ON WORKSHOP

ERCHONIA

BEDSIDE NEUROLOGICAL EXAM

BRAIN BALANCING WITH MASTER LASER SETTING

"DOWNSTREAM" STACKING

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BREAK

DIET AND LIFESTYLE

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3 SUPPLEMENTS FOR BRAIN



WHAT WOULD BE A GOOD COMBO IF A PATIENT IS ONLY WILLING TO TAKE 3 SUPPLEMENTS FOR THEIR BRAIN HEALTH?



ALZHEIMER'S AND OMEGA 3 INTAKE



Omega 3 Intake Linked to Stable Memory Function in Alzheimer's Patients

FeaturedNeurologyNeuroscience[.]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

USE THE VIBRANT MICRONUTRIENT PANELS OR BRAIN SPAN FOR ACCURATE INTRACELLULAR ESSENTIAL FATTY ACID MARKERS AND RATIOS



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 Inês F. Sequeira

FISH SOURCE



SEATOPIA



Certified-clean, guilt-free, sushi-grade seafood.

Mercury safe, antibiotic free, & truly sustainable. SEATOPIA.COM

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OMEGA 3 SUPPORT MAXSIMIL TECHNOLOGY





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







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 \geq 65 years old), we examined the association of RBC DHA with incident AD, testing for an interaction with *APOE*- ε 4 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*- ε 4 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*- ε 4 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

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

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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	Х	
Macular Degeneration			XX
Alzheimer- Dementia		X	XX
Maternal- Childhood Devel.		x	хх

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



ARE HARSH EXTRACTED SOY BASED PHOSPHATIDYLCHOLINE. BIOPC USES GENTLE COLD PRESSED SUNFLOWER EXTRACTION AND IS BROAD SPECTRUM (CHOLINE, SERINE ETC).

MOST PHOSPHOLIPIDS



(CHOLINE, SERINE ETC) Duplication, citation or distributio#% f this material in whole or in without permission of Trevor Berry, DC, DACNB is prohibited

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

*CLINICAL PEARL



FOR EVEN BETTER FATTY ACID ABSORPTION, STACK OMEGA FATTY ACID SUPPLEMENTS WITH DIGESTIVE ENZYMES THAT PROMOTE FAT EMULSIFICATION AND



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 En Treating Cognitive Impairment in Older Adults: A Ranc 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 provide the provide provide

and there were BEST LAB TEST FOR MAGNESIUM?

Published online 2019 Nov 11. doi: <u>10.2147/NDT.S230688</u>

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.#

 $Magnesium \ L-three on the Prevents \ and \ Restores \ Memory \ Deficits \ Associated \ with \ Neuropathic \ Pain \ by \ Inhibition \ of \ TNF-\alpha$

Jun Wang, PhD, Yong Liu, PhD, LipJuptiZatton, PhD, iog West; Bhd Arteria Shant PhD, Bait Ping Pang, PhD, Xu-Hong Wei, PhD, Yong-Yong Li, and Xian-Guo Liw the permission of Trevor Berry, DC, DACNB is prohibited.

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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-I-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.





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 #123, 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 23, 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 Mg₂₊ levels in plasma, cerebrospinal fluid and in dorsal root ganglion neurons were significantly reduced. Oral magnesium-L-threonate normalized the neuroinflammation, synaptic potentiation and Mg₂₊ 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 Mg₂₊, and downregulated phospho-p65, tumor necrosis factor-alpha and interleukin-1 beta exclusively in the presence of extracellular Mg₂₊.

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 Duplication, citation or distribution of this material in whole or in part menopause-related neuronabel isonisters for trever Berry, DC, DACNB is prohibited.

MAGNESIUM



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 pathogeninfected 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, "Magnesiun sensing via LFA 1 regulates CD8+ T cell effector function."

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

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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.

VAGNESIUM 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.

 Open AccessReview
 MAGNESIUM OROTATE:
 MG AND OROTIC ACID.
 CARDIOVASCULAR SYSTEMS BENEFITS.

 Magnesium in Prevention and Therapy by Uwe Gröber 1,*,Joachim Schmidt 1 an@Kfaus Kisters 1,2
 Nutrients 2015, 7(9), 8199-8226; https://doi.org/10.3390/nu7095388
 https://doi.org/10.3390/nu7095388

3 SUPPLEMENTS FOR BRAIN 2. MAGNESIUM



GOOD: 1 GRAM MAGNESIUM L-THREONATE (MAGTEIN) BETTER: STACK L-THREONATE WITH MAGNESIUM MALATE BEST: STACK L-THREONATE AND MALATE WITH CHELATED GLYCINATE/LYSINATE

XYMOGEN OPTIMAG NEURO





OPTIMAG NEURO IS WIDELY CONSIDERED "BEST IN SHOW" FOR OVERAL BRAIN AND PHYSIOLOGY MAGNESIUM SUPPORT AS IT CONTAINS ALL 3 LISTED ABOVE. NOTE: THIS IS NOT THE PRIMARY MAGNESIUM FOR BOWEL MOVEMENTS NOR SHOULD IT HAVE A DIRECT EFFECT ON THAT SYSTEM

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3 SUPPLEMENTS FOR BRAIN 3.COMBINATION SUPPLEMENT



Supplement Facts Serving Size: 4 Capsules Servings Per Container: 30 START WITH 1/DAY AND WORK UP TO 2 IN AM WITH BREAKFAST

	Vitamin A (75% as natural beta-carotene and 25% as retinyl palmitate)	400 mcg	44%	Γ
	Vitamin C (as sodium ascorbate, potassium ascorbate, zinc ascorbate, and calcium ascorbate)	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 salts1 and as calcium folinate)	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 malates2, d-calcium pantothenate, and calcium ascorbate)	17 mg	2%	
	lodine (as potassium iodide)	17 mcg	11%	
	Magnesium (as di-magnesium malate)s2	65 mg	15%	
	Zinc (as zinc bisglycinate chelates2)	2 mg	18%	
	Selenium (as selenium glycinate complex)s2	17 mcg	31%	
	Manganese (as manganese bisglycinate chelate)s2	0.09 mg	4%	
	Chromium (as chromium nicotinate glycinate chelate)s2	89 mcg	254%	
	Molybdenum (as molybdenum glycinate chelate)s2	8 mcg	18%	
	Potassium (as potassium glycinate complexs2 and potassium ascorbate)	17 mg	0%	
N-	AcetvI-L-Cvsteine		200 ma	**
Ba	copa Extract (Bacopa monnieri)(whole plant)(8% bacosides)		200 ma	**
Al	pha-Lipoic Acid		100 ma	*1
Sł	ilaiit (10.3% Dibenzo-α pyrones (DBPs) & Dibenzo-α pyrone Chromoproteins (DCPs))(50% fulvic acid	ts with DBP Core	100 ma	*1
Th	eacrines4		100 mg	**
Mi	k Thistle Extract (Silybum marianum)(seeds)(30% silybins)		90 mg	**
Gr	een Tea Extract (Camellia sinensis)(leaf)(75% catechins, 45% EGCG)		45 mcg	**
Mi	xed Tocopherols		25 mg	**
Be	nfotiamine		10 mg	**
tra	ns-Resveratrol (from Polygonum cuspidatum)(root)		10 mg	*
Inc	psitol		6 mg	*
GI	ucoraphanin (from broccoli extract)(Brassica oleracea italica)(seed)s5		5.2 mg	**
Hu	perzine A (from Huperzia serrata)(whole plant)		200 mcq	**
Va	nadium (as vanadium nicotinate glycigate chelate)s2		134 mcg	**
				1

Highlights VITAMIN D AND ALZHEIMER'S



ERCHONIA

- 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 Ε ε4 non-carriers versus carriers.
- Vitamin D has potential for dementia prevention, especially in the high-risk strata.

Interpretation: Exposure to vitamin D supplementation was associated with a 40% lower dementia incidence rate than no exposure, providing strong support for supplementation. The results were consistent across three vitamin D formulations.

Alzheimers Dement (Amst). 2023 Jan-Mar; 15(1): e12404.

Published online **2023 Mar 1**. doi: <u>10.1002/dad2.12404</u>

Vitamin D supplementation and incident dementia: Effects of sex, *APOE*, and baseline cognitive status

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XYMOGEN QR CODE FOR NON-PHYSICIAN ACCOUNT PURCHASES



ALTHOUGH IT IS HIGHLY ENCOURAGED TO OPEN YOUR OWN WHOLESCRIPTS ACCOUNT TO BENEFIT FROM XYMOGEN PLUS 99 OTHER PRODUCT LINES, THIS QR CODE WILL LINK YOU TO DIRECT PURCHASE IF YOU WANTED TO TRY PRODUCTS FIRST OR FOR THOSE THAT DON'T HAVE AN NPI NUMBER TO OPEN THEIR OWN ACCOUNT SUCH AS PATIENTS IN ATTENDANCE OR NON-MEDICAL PROVIDERS



NOTE: THIS IS DR. BERRY'S WHOLESCRIPTS ACCOUNT

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THE ULTIMATE ANTIOXIDANT AND ANTI-AGING MECHANISM

WHAT MECHANISM IS THE PRIMARY FACTOR NEEDED FOR CYTO- AND CHEMOPROTECTIVE MECHANISMS IN NUTRIGENOMICS AND NUTRIGENETICS?





ERCHONIA

Amount Per Serving%DV

Turmeric Extract (*Curcuma longa*)(rhizome)(95% total curcumi curcumin)S1 400 mg**

Green Tea Aqueous Extract (*Camellia sinensis*)(leaf)(60% catec 400 mg**

trans-PterostilbeneS2 100 mg**

Glucoraphanin (from broccoli extract)(Brassica oleracea italica

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LOOK UP NRF2/ARE AND DISEASE ON PUBMED. THIS PROCESS COULD BE DESCRIBED AS THE MASTER DEFENSE MECHANISM AGAINST DISEASE STATES INCLUDING CANCER AND NEURODEGENERATION!

Int J Mol Sci

2021 Sep 4;22(17):9592. doi: 10.3390/ijms22179592.

An Overview of the Nrf2/ARE Pathway and Its Role in Neurodegenerative Diseases

Abstract

Emilia Zgorzynska 1, Barbara Dziedzic 1, Anna Walczewska 1

Nrf2 is a basic region leucine-zipper transcription factor that plays a pivotal role in the coordinated gene expression of antioxidant and detoxifying enzymes, promoting cell survival in adverse environmental or defective metabolic conditions. After synthesis, Nrf2 is arrested in the cytoplasm by the Kelch-like ECH-associated protein 1 suppressor (Keap1) leading Nrf2 to ubiquitin-dependent degradation. One Nrf2 activation mechanism relies on disconnection from the Keap1 homodimer through the oxidation of cysteine at specific sites of Keap1. Free Nrf2 enters the nucleus, dimerizes with small musculoaponeurotic fibrosarcoma proteins (sMafs), and binds to the antioxidant response

element (ARE) sequence of the target genes. Since oxidative stress, next to neuroinflammation and mitochondrial dysfunction, is one of the hallmarks of neurodegenerative pathologies, a molecular intervention into Nrf2/ARE signaling and the enhancement of the transcriptional activity of particular genes are targets for prevention or delaying the onset of age-related and inherited neurogenerative diseases. In this study, we review evidence for the Nrf2/ARE-driven pathway dysfunctions leading to various neurological pathologies, such as Alzheimer's, Parkinson's, and Huntington's diseases, as well as amyotrophic lateral sclerosis, and the beneficial role of natural and synthetic molecules that are able to interact with Nrf2 to enhance its protective efficacy.

THE ULTIMATE ANTIOXIDANT AND ANTI-AGING MECHANISM

- Provides Concentrated Glucoraphanin from Broccoli Seed Extract*
- Supports Healthy Cell-Life Cycles*
- Supports Phase II Detoxification Enzymes*
- Supports Extended Antioxidant Activity*
- Myrosinase Promotes Conversion of Glucoraphanin to Sulforaphane*

OncoPLEX[™] Plus Myrosinase features broccoli seed extract enhanced with myrosinase, the enzyme that promotes the conversion of naturally occurring glucoraphanin in broccoli to sulforaphane (SFN). A potent activator of antioxidant activity, healthy cell-life cycles, and the production of detoxification enzymes, SFN is linked to the many health benefits associated with cruciferous vegetables.*

-SULFORAPHANE IS THE MOST POTENT ACTIVATOR OF NRF2 ALONG WITH EXERCISE, FASTING AND 635 NM LLLT -THIS PROCESS CRITICAL FOR EVERYTHING FROM PHASE 2 LIVER DETOX, ANTI-OXIDANT ACTIVITY OF GLUTATHIONE, ANTI-AGING TO ANTI-CARCINOGENIC PROTECTION (EX. 2/3 OF CIGARETTE SMOKE BENZENE IS NEUTRALIZED BY SULFORAPHANES). THE EQUIVALENT OF 6 LBS OF BROCCOLI -PROTECTS AGAINST ESTROGEN MALCONVERSION QUINONES THAT CAN CAUSE DNA DAMAGE -TYPICAL ABSORPTION OF BROCCOLI DERIVED GLUCORAPHANINS IS ONLY ABOUT 10%

-MYROSINASE ENZYME INCREASES THE CONVERSION OF GLUCORAPHANIN TO SULFORAPHANE TO 40%!



ERCHONIA

-MYROSINASE IS EXTRACTED FROM MUSTARD SEED POWDER. VITAMIN C FURTHER PROMOTES THIS CONVERSION. VITAMIN E IS CONSIDERED THE GOLD STANDARD FOR ORAC VALUE (ANTI-OXIDANT VALUE). SUPER ANTIOXIDANTS LIKE ACAI HAVE ORACS AROUND 20-30K. SULFORAPHANES HAVE AN ORAC OF 300,000! -NO TOXICITY FOR SULFORAPHANES. ONLY NEED 1 CAP EVERY 2-3 DAYS! <\$20 FOR 30

-MUST INCREASE ANTI-OXIDANT RESPONSE ELEMENT TO FIGHT OFF ENVIRONMENTAL DAMAGE LIKE GLYPHOSA introduction of distribution of the state of this material in whole or in part DAMAGE LIKE GLYPHOSA introduct permission of Trevor Berry, DC, DACNB is prohibited. OST POTENT DEFENSE!



HOMOCYSTEINE: NOT UUST A CARDIOVASCULAR RISK FACTOR



Homocysteine is an amino acid that's heavily involved in a biological process called <u>methylation</u>. 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 <u>detoxification pathways</u>; 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 <u>can't optimize methylation</u>. 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 <u>12% to 31% of</u> <u>dementia cases</u> could be prevented by lowering fasting total homocysteine (tHcy) levels.

TAKE YOUR METHYLATED B'S!

LOW LEVEL LASER THERAPY AND CARDIAC FUNCTION



Abstract

Background: Low-level laser therapy (LLLT) is a promising noninvasive physiotherapeutic approach that has been demonstrated to improve cardiac performance. This study aimed to assess the impact of low-level laser therapy on cardiac functions and clinical status in patients with chronic left ventricular systolic heart failure who were not candidates for cardiac revascularization or resynchronization. A case series of 27 patients received a course of low-level laser physiotherapy, the clinical outcomes, echocardiographic parameters, and serum nitric oxide levels were evaluated before and after LLLT.

Results: Of the total patients enrolled in the study, 21 (or 77.8%) were male, with a mean age of 57.7 ± 6.89 years. NYHA classification significantly improved after low-level laser therapy, 15 patients were in class III, 12 were in class IV, and no one was in class II before laser

therapy while after laser therapy; 25 patients shifted to class II, two patients were in class III with P < 0.001, **Six-minute walk** distance test was performed, and the results showed that the mean of 6MWT was less than 200 m (148.556 ± 39.092) before the study but increased to more than 300 after laser therapy (385.074 ± 61.740), left ventricular ejection fraction before laser therapy was 26 ± 7.5 while after laser therapy it became 30 ± 8.6 but diastolic function did not change after low-level laser therapy, the mean peak TR pressure was 40.0 ± 9.0 mmHg and 33.0 ± 7.0 before and after laser therapy respectively P < 0.001. A significant change was observed in NO level from 4.1 ± 1.4 IU/mI before laser therapy to 5.2 ± 1.7 IU/mI after laser therapy P < 0.001.

Conclusions: Low-level laser therapy may add benefits to improve symptoms, clinical condition, and quality of life in patients with left ventricular systolic dysfunction, further studies are necessary to evaluate the changes in cardiac functions at a longer follow-up duration.

Egypt Heart J **2023 Feb** 13;75(1):12. doi: 10.1186/s43044-023-00337-6.

Effect of low-level laser physiotherapy on left ventricular function among patients with chronic systolic heart failure

Mahmoud Abdulbasser Sayed 1

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Light therapy or Cost of this material in whole or long the structure of the second restored of the second restored of the second restored of the second sec aging and fend off deadly disease



Abstract

Background

Photobiomodulation (PBM) therapy, a form of low-dose light therapy, has been noted to be effective in several age-associated chronic diseases such as hypertension and atherosclerosis. Here, we examined the effects of PBM therapy on age-associated cardiovascular changes in a mouse model of accelerated cardiac aging.

Methods

Fourteen months old Adenylyl cyclase type VIII (AC8) overexpressing transgenic mice (n=8) and their wild-type (WT) littermates (n=8) were treated with daily exposure to Near-Infrared Light

(850 nm) at 25 mW/cm2 for 2 min each weekday for a total dose of 1 Einstein (4.5 p. J/cm2 or fluence 3 J/cm2) and compared to untreated controls over an 8-month period. PBM therapy was administered for 3.5 months (Early Treatment period), paused, due to Covid-19 restrictions for the following 3 months, and restarted again for 1.5 months. Serial echocardiography and gait analyses were performed at monthly intervals, and serum TGF-81 levels were assessed following sacrifice.

Results

During the Early Treatment period PBM treatments: reduced the age-associated increases in left ventricular (LV) mass in both genotypes (p=0.0003), reduced the LV end-diastolic volume (EDV) in AC8 (p=0.04); and reduced the left atrial dimension in both genotypes (p=0.02). PBM treatments substantially increased the LV ejection fraction (p = 0.03), reduced the aortic wall stiffness (p = 0.001), and improved gait symmetry, an index of neuro-muscular coordination (p = 0.005). The effects of PBM treatments, measured following the pause, persisted. Total TGF- β 1 levels were significantly increased in circulation (serum) in AC8 following PBM treatments (p = 0.01). We observed a striking increase in cumulative survival in PBM-treated AC8 mice (100%; p = 0.01) compared to untreated AC8 mice (43%).

100% SURVIVAL OF THE TREATED ANIMALS VS 43% OF THE UNTREATED! Conclusion

PBM treatment mitigated age-associated cardiovascular remodeling and reduced cardiac function, improved neuromuscular coordination, and increased longevity in an experimental animal model. These responses correlate with increased TGF-B1 in circulation. Future mechanistic and dose optimization studies are necessary to assess these anti-aging effects of PBM, and validation in future controlled human studies is required for effective clinical translation.

Photobiomodulation therapy mitigates cardiovascular aging and improves survival

Sunayana Begum Syed PhD. Ismayil Ahmet PhD. Khalid Chakir PhD. Christopher H. Morrell PhD. Praveen R. Arany DDS, PhD. Edward G. Lakatta MD 385

First published: 23 February 2023

CARDIOVASCULAR DISEASE AND LLLT



Exploring A No	vel Approach	Using Red o	and Viole	t Laser T	herapy Fo
	Carotid Artery	Stenosis - A	A Case Se	eries	

Recent Advances in Clinical Trials

Andrzej Eberhardt¹ and Travis Sammons^{2*}

*Correspondence: Travis M. Sammons, Erchonia Corporation, 112 Southchase Bvld., Fountain Inn, SC 29644, Tel: 888-242-0571, Fax: ¹General surgery and Vascular, Vascular Surgery and Phlebology in Femmed Clinic, Warsaw, Poland, 321.473.1608 2Erchonia Corporation, Melbourne, FL, USA. ed: 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 worldwide [2]. Complications arising from unstable atheroselerotic 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 enterpresented to easily a strong 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 potentially slow ancroscierotic plaque progression [4]. Antipiateder 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 tinnius. For symptomatic patients with severe stenosis (70% to 99%), carotid endarterectomy, a surgical procedure to remove plaque, is often recommended [6]

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 res 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 can parent undervent beweeky unasound 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 topectivity and Coshi value is one or use ratio will windows will methods of studying atheroscientic 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 wide kiew is del e. e.t. where GSM value suggest softer plaques with a higher lipid or fluid content, potentially indicating a more unstable plaque and a worse prognosis for CS

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, by evaluating the potential therapeutic effects of LLLT as both smoking history, or diabetic status. These findings suggest that This study enrolled a total of 30 patients (17 male, 13 female) 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.

All participants received laser therapy using the Erchonia EVRL Dupplications citations or distribution for the Waterial OF WIRDEATME BATE BI-WEEKLY 15 MINUTES EVRL) Recent studies investigating the effects of low-level laser therapy (LLLT) on vascular health have shown promising results, and may violet with out permission of Trevor Berry, DC, DACNB is prohibited.



K2-D3 5000

120 Vegetarian Dietary Capsules Supplement

S-Acetvl

Glutathione

Some Statins Linked to Dementia Risk

The researchers found that of 300 older adults with mildly impaired thinking and memory, those using "lipophilic" statins were more likely to develop dementia over the next eight years.

Lipophilic statins include such widely used medications as simvastatin (Zocor), atorvastatin (Lipitor) and lovastatin (Altoprev).

In patients with mild cognitive impairment, taking lipophilic statins more than doubles their risk of developing dementia compared to those who do not take statins. According to research presented at the Society of Nuclear Medicine and Molecular Imaging 2021 Annual Meeting

"Lipophilic Statins in Subjects with Early Mild Cognitive Impairment: Associations with Conversion to Dementia and Decline in Posterior Cingulate Brain Metabolism in a Long-term Prospective Longitudinal Multi-Center Study," Prasanna Padmanabham, Stephen Liu and Daniel Silverman, University of California, Los Angeles, Los Angeles, California.

"Pharmacological evidence and clinical trial results support the interpretation that statins stimulate atherogenesis by suppressing vitamin K2 synthesis and thereby enhancing artery calcification. Statins cause heart failure by depleting the myocardium of CoQ10, Heme A and selenoproteins (ex. Glutathione Peroxidase), thereby impairing mitochondrial ATP production. In summary, statins are not only ineffective in preventing congestive heart disease events but instead are capable of increasing CHD and heart failure. "



Expert Review of Clinical Pharmacology 2015, Mar;8(2):189-99-Okuyama et al



Our data demonstrate that lovastatin concentrations able to suppress not only cholesterol but also geranylgeranylpyrophosphate formation may evoke phosphorylation of tau reminiscent of preclinical early stages of Alzheimer's disease and, when prolonged, apoptosis.

CARDIOVASCULAR SUPPORT



Big Pharma Suffers Another Major Blow As Study Debunks High Cholesterol Myths, Admitting Statins Are Totally Worthless





Study suggests that HDL or 'good' cholesterol is less beneficial than previously thought, especially for Black adults

Now, <u>research</u> published Monday in the Journal of the American College of Cardiology found that low levels of HDL cholesterol were associated with higher risk of heart attack among White adults, but the same was not true

among Black adults. Also, higher levels of HDL cholesterol were not found to reduce the risk of cardiovascular disease for either group.

SUMMARY: THE STANDARD LIPID PANEL IS AN ANTEQUATED ANALYSIS OF CARDIOVASCULAR RISK FACTORS. CONSIDER THE FULL BOSTON OR CLEVELAND HEART PANELS FOR A MORE ACCURATE, CURRENT AND COMPREHENSIVE ANALYSIS. AT MINIMUM, ADD OX-LDL, sLDL, HS-CRP, HBA1c, AND HOMOCYSTEINE TO YOUR STANDARD LIPID PANEL FOR CV RISKS

Race-Dependent Association of High-Density Lipoprotein Cholesterol Levels With Incident Coronary Artery Disease

Neil A. Zakai, Jessica Minnier, Monika M. Safford, Insu Koh, Marguerite R. Irvin, Sergio Fazio, Mary Cushman, Virginia J. Howard, and Nathalie Pamir

J Am Coll Cardiol. 2022 Nov, 80 (22) 2104-2915

CARDIOVASCULAR SUPPORT



- DON'T GET CAUGHT IN THE "ALL LDL IS BAD AND ALL HDL IS GOOD" TRAP. THE BRAIN AND BODY NEEDS LDL. LDL CAN BE HARMFUL IF SMALL AND "OXIDIZED" WHICH GET GOBBLED UP BY MACROPHAGES IN THE BLOOD VESSELS TURNING INTO TO FOAM CELLS WHICH CAN PROMOTE ATHEROSCLEROTIC PLAQUE FORMATION. HOMOCYSTEINE IS A MAIN PRECURSOR CAUSING ENDOTHELIAL DAMAGE. METHYLATION PRODUCTS ARE BEST (WATCH COMT GENE SNP - USE HYDROXYLATED B'S INSTEAD)

- SUGAR/CARBS SHOULD BE THE TARGET AS THE PRIMARY CULPRIT OF THIS INFLAMMATION OXIDIZING LDL PROCESS (SAD DIET OF AMERICA)
- NIACIN AND EXERCISE ARE YOUR BEST DEFENSE AGAINST "BAD HDL" FORMS
- STATINS SHOULD ONLY BE GIVEN (IF AT ALL), IF A GENETIC TEST HAS BEEN GIVEN TO RULE OUT INTOLERANCE (SLCO1B1 - CAN LEAD TO STATIN INDUCED MYOPATHY), AND ARE ON 200+ mg OF CO-Q10
- DON'T COLD TURKEY STOP HIGH DOSES OF CO-Q10. TITRATE DOWN
- CO-Q10 BY ITSELF CAN IMPROVE OXIDIZED LDL:HDL BY 42.5%
- RED YEAST RICE IS BASICALLY A NATURAL SIMVASTATIN
- LIPOPROTEIN A DOES NOT RESPOND TO STATINS
- TO PROPERLY ASSESS CVD RISK FACTORS, HOMOCYSTEINE MUST BE ADDRESSED. IT WILL DISRUPT THE ENDOTHELIAL LINING OF CV SYSTEM SETTING THE STAGE FOR ATHEROMA'S

RUN THE BOSTON OR CLEVELAND HEART PANEL FOR DUTHE MOST COMPREMENSIVE CARDIAC PROFILE without permission of Trevor Beny, DC, DACINB is prohibited.



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 = SLOW RELEASE 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.

DR. BROCK SEATTLE BULLET POINTS



CHRONIC SINUSITIS ADDRESS MOLD AND FUNGUS

ALT AND CRP SKYROCKETING SINCE COVID

VIOLET LASER BEST FOR NOS EXPRESSION, PREVENTING FIBROSIS AND ANTIMICROBIAL

FIBROSURE INSTEAD OF LIVER PUNCH BIOPSY

METALS HM COMPLEX FROM PURE ENCAPSULATIONS AND HEPATOSYNERGY FROM APEX FOR NUCLEAR WARHEAD FOR LIVER START WITH HM COMPLEX - PURE THEN APEX HEPATOSYNERGY

METHYLATION MAKES NEUROTRANSMITTER WATCH MTHFR COMT BREAKS DOWN NEUROTRANSMITTER AND HORMONES BREAKDOWN COMT CAN'T BREAK DOWN EPI/NOREPI SO STRESS HORMONES STAY IN FOR HOURS...PLASTICITY IN AMYGDALA

> WHEN COMING OF SSRI'S CB IS HIGHLY SEROTONERGIC SO THEY GET THE WHIRLY'S USING HYDROXYLATION AND LASER, GUT HEALTH DRUG DISCONTINUATION SYNDROME

STORED CORTISONE TURNS INTO CORTISOL. ADRENALS DON'T "FATIGUE". IT'S AN HP AXIS MALADAPTATION CAN USE ADAPTOGENS

STRESS CORTISOL ERODES GUT LINING IT CAUSES FAT AROUND VISCERA

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DR. BROCK SEATTLE BULLET POINTS



ALT IS THE PRIMARY ALCOHOL MARKER AL/ALCOHOL

FLUSHING NIACIN 750 MG IS THE ONLY MAJOR NUTRIENT KNOWN TO LOWER LPa LPa IS STATIN RESITANT

MRNA VACCINE => REPLICATION OF S2 SPIKE PROTEIN WITHOUT AN OFF SWITCH!

ENDOCALYX WAS FORMULATED TO HELP GLYCOMATRIX...SAME REASON AS HEPARIN NATTOKINASE IS ALSO A BIOFILM DISRUPTER

DON'T USE TOPICAL HRT, ACCUMULATES IN FAT CELLS, PELLETS CAN CAUSE UTERINE THICKENING ADIPONECTINS STOP INFLAMMATION BUT LEPTIN RESISTANCE => STOPS ADIPONECTINS = INCREASED CYTOKINE STORM PLUS INSULIN RESISTANCE...EQUALS URIC ACID ELEVATION

ADIPONECTIN PRODUCTION HELPTS INHIBIT INFLAMMATION

HE DOESN'T THINK IODINE FURTHER PROMOTES HASHIMOTOS HE HAS SHOWN OZEMPIC PROMOTES C PEPTIDE NUMBERS IN TYPE 1 DIABETICS!

FAVORITE CARDIAC STUDY IS A NUCLEAR STRESS TEST

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S2 SPIKE PROTEIN

Published online 2022 Aug 24. doi: 10.3390/molecules27175405

PMCID: PMC9458005 PMID: <u>36080170</u>

HONIA

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.



RECENT COVID VACCINE NEWS



1.98 g

L-Arginine alpha-Ketoglutarate

One in four who had Pfizer Covid jabs experienced unintended immune response

The code relating to the Covid vaccines was harmless and no issues were created. However the team say that subsequent mRNA vaccines used for other diseases or infections could, in theory, lead to viable proteins being created that are active in the body.

In this scenario not only is the vaccine not making the right protein, it could lead to a rogue protein being produced. The authors also found that there is an easy way to eradicate the frameshifting events which relies on changing the code of the mRNA drug to minimize the use of the problematic pseudo-uridine.

The study found evidence in mice and humans for the misreading of the modRNA contained within the Pfizer COVID-19 vaccine to inadvertently produce "off-target" proteins capable of eliciting "off-target" immune responses.

Ribosomal frameshifting and misreading of mRNA in COVID-19 vaccines produces "off-target" proteins and immune responses eliciting safety concerns: Comment on UK study by Mulroney et al

December 2023



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Dr. Mawkout permission or distribution of this material in whole or part amid:



DIET FOR REDUCTION OF DEMENTIA RISKS



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Mediterranean Diet Associated With Decreased Risk of

Dementia

FeaturedNeurologyNeuroscienceOpen Neuroscience ArticlesMarch 14, 2023

summary: Older adults who consumed a Mediterranean-style diet had a 23% lower risk of developing dementia than those who consumed a typical diet.

Eating a traditional Mediterranean-type diet – rich in foods such as seafood, fruit, and nuts – may help reduce the risk of dementia by almost a quarter, a new study has revealed.

This research, published today in *BMC Medicine*, is one of the biggest studies of its kind as previous studies have typically been limited to small sample sizes and low numbers of dementia cases.

Scientists analysed **data from 60,298 individuals** from the UK Biobank, a large cohort including individuals from across the UK, who had completed a dietary assessment.

The authors scored individuals based on how closely their diet matched the key features of a Mediterranean one. The participants were followed for almost a decade, during which time there were 882 cases of dementia.

The authors considered each individual's genetic risk for dementia by estimating what is known as their polygenic risk – a measure of all the different genes that are related to the risk of dementiation, citation or distributio#06 this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



Skipping breakfast is a daily habit that can increase your risk of dementia by four times!

BREAKFAST SUPERFOOD IDEAS:

- EGGS
- AVOCADO
- COFFEE FRIEND OR FOE?
- SOLUBLE FIBERS INCLUDING SEEDS LIKE FLAX, BEANS, NUTS, PEAS (ALL), OATS, FRUIT INCLUDING BERRIES, KIWI, BANANAS, PEARS, APPLES (ZONE AND WHOLE **NOT JUST JUICE)**
- WHOLE FOOD JUICING WITH IMMUNE NEUTRAL PROTEIN
- GET YOUR WATER STARTED FOR THE DAY (TARGET 24 OZ BEFORE YOU LEAVE THE HOUSE)

*NOTE: FOOD SENSITIVITY/ALLERGY LISTS SUPERSEDE GENERAL DIETARY **RECOMMENDATIONS!**

Relationship between lifestyle habits and dementia among community dwelling older adult : Findings from a 6-year follow up(Paper & Abstract September 17th)

Maki Hirano, Yuri Kawashima, Sumio Ito, Ryoji Sinohara, Yuka Sugisawa, Yuko Sawada, Yukiko Ishii, Lian Tong, Emiko Tanaka, Etsuko Tomisaki, Taeko Watanabe, Yoko Onda, Yukiko Mochizuki, Kentaro Morita, Mayumi Namba, Amarsanaa Gan-Yadam, Kentaro Tokutake, Bailiang Wu, Tokie Anme

Author information

Keywords: Cohort Study, Dementia, Elderly JOURNAL FREE ACCESS 2011 Volume 18 Issue 1 Pages 102-106

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"THE DIRTY DOZEN" 2024



THE DIRTY DOZEN

- 1. STRAWBERRIES
- 2. SPINACH

4. GRAPES

- 3. KALE, COLLARD, MUSTARD GREENS
- 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.

- 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

- **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**

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

adolescence. A strategie of the state of the



DR. BERRY'S MEDPAX 2024



ERCHON

PhosphaLine



ERCHONIA LLLT BRAIN/VAGAL, PAIN AREAS, IMMUNE BOOST ETC



AM:

- -1 OMEGAMONOPURE COQ10
- -1 ACTIV NUTRIENTS PERFORMANCE
- -1 ACTIV NUTRIENTS (MULTI WITHOUT COPPER AND IRON)
- **-1 S-ACETYL GLUTATHIONE**
- -1 OPTIMAG NEURO CAP (MG NORMALLY IN PM FOR MOST) -1 HISTDAO

PM:

- -1 NAC
- -1 CORTISOLV
- -1 K2-D3 5000
- -1 ONCOPLEX WITH MYROSINASE
- -1 PROBIOMAX IG 26 DF (ROTATE)
- -1 HEMP MONOPURE

= L-SERINE AND RESVERATROL

NON-ALCOHOLIC FATTY LIVER DISEASE

Fatty Liver Affects 80% of obese Women and 30% general population Over 40 — Here's How to Prevent and Reverse It

1. EAT EGGS AND OTHER HIGH CHOLINE FOOD SOURCES

University of North Carolina study, 80 percent of women with fatty liver who upped their choline intake had a 100 percent reversal of the condition, often within 30 days. 500 mg DAILY. *XYMOGEN PHOSPHALINE = 2.7 GRAMS! 2 DRINK COEEEE

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ERCHONIA LOW LEVEL LASER THERAPY: 12 VISIT TREATMENT TRIAL OVER 3 MONTHS 5 MINUTES WAND OR FX SCAN OVER LIVER/RUQ (ERCHONIA CLINICAL TRIAL IS 1X/WEEK FOR **10 MINUTES WITH EVRL)** FREQUENCIES 53-537-55-751

> PRE AND POST CHECK LIVER ENZYMES (ALT/AST/GGT) OTHER CONSIDERATIONS: HS-CRP, HBA1C, LIPID PANEL, ESR

CURCUPLEX-95, NRF2 ACTIVATOR

4. EXERCISE

Just 30 minutes of activity daily helps the liver burn 39 percent of its stored fat, say British scientists. STOP ALCOHOL/SUGAR INTAKE!

Eur Endocrinol, 2020 Oct; 16(2); 152-155.

Published online 2020 Oct 6. doi: 10.17925/EE.2020.16.2.152



DEMENTIA RISK INCREASE



Increased risk for all-cause dementia in people who abstain from alcohol

the researchers collected data from 15 prospective epidemiological cohort studies conducted across six continents to examine the relationship between alcohol and dementia.

A total of 24,478 individuals were included in the current study, with a mean age of 71.8 years at baseline. Among these, 58.3% were females, and 54.2% were current drinkers.

Conclusions

The study findings suggest that abstaining from alcohol may increase the risk for all-cause dementia. Furthermore, there was no evidence to suggest that the amount of alcohol consumed impacts the dementia risk.

The relationship between alcohol use and dementia in adults aged more than 60 years: a combined analysis of prospective, individual-participant data from 15 international studies

Louise Mewton, Rachel Visontay, Nicholas Hoy, Darren M. Lipnicki, Matthew Sunderland, Richard B. Lipton, Maëlenn Guerchet, Karen Ritchie, Jenna Najar ... See all authors First published: 22 August 2022

INTERMITTENT FASTING



Published: 17 November 2022

Intermittent fasting protects against Alzheimer's disease in mice by altering metabolism through remodeling of the gut microbiota

Abstract , Pap IOURNAL NATURE AGING

Alzheimer's disease (AD) is the most common form of dementia without effective clinical treatment. Here, we show that intermittent fasting (IF) improves cognitive functions and AD-like pathology in a transgenic AD mouse model (5XFAD). IF alters gut microbial composition with a significant enrichment in probiotics such as *Lactobacillus*. The changes in the composition of the gut microbiota affect metabolic activities and metabolite production. Metabolomic profiling analysis of cecal contents revealed IF leads to a decreased carbohydrate metabolism (for example, glucose) and an increased abundance in amino acids (for example, sarcosine and dimethylglycine). Interestingly, we found that the administration of IF-elevated

sarcosine or dimethylglycine mimics the protective effects of IF in 5XFAD mice, including the **amelioration of**

cognitive decline, amyloid- β (A β) burden and glial overactivation. Our findings thus demonstrate an IF regimen is a potential

approach to prevent AD progression, at least through the gut-microbiota-metabolites-brain axis, and constitutes an innovative AD therapeutic avenue.

CLINICAL RECOMMENDATION: 24 HOUR WATER FAST EVERY 2-3 WEEKS. COME OFF FAST USING BISON BONE BROTH

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Highly processed oils are often extracted from soybeans, corn, rapeseed (the source of canola oil), cottonseed, sunflower and safflower seeds, and contain a lot of omega-6 fatty acids. Excess consumption of omega-6s can trigger the body to produce chemicals that can lead to inflammation in the brain. If you're sautéing vegetables or grilling fish or meat, I recommend using olive, coconut or avocado oil.

2. Foods with added and refined sugars

Our brain uses energy in the form of glucose, a type of sugar, to fuel cellular activities. But a high sugar diet can lead to excess glucose in the brain. This can cause memory impairments and less plasticity of the hippocampus, the part of the brain that controls memory. Don't forget that many savory foods have hidden added sugars, too, like store-bought pasta sauces, ketchup, salad dressings and even canned soups. Swap these out for homemade items made with whole foods.

3. Processed foods

A diet high in ultra-processed foods may put you at risk of having shorter telomeres — or the "cap" on our DNA. Longer telomeres tend to promote healthy cellular aging. Shortening our telomeres may mean that we are at risk of degenerative disease earlier in life.A 2022 study also found that participants who consumed high amounts of ultraprocessed foods like baked goods and sodas were more likely to experience mild depression compared to those who consumed the least.Here's a tip: If you can't pronounce an ingredient, or have no idea what it is, it's often best to avoid it.

4. Foods with artificial sweeteners

When you use artificial sweeteners that have no nutritional value, they can increase "bad" gut bacteria which can negatively affect your mood. These sweeteners include saccharin, sucralose and stevia. Aspartame can be especially harmful, and has been directly linked with anxiety in research studies. It also causes oxidation, which increases harmful free radicals in the brain. Some alternatives to consider: Honey, monk fruit extract or coconut sugar. (Organic stevia, non aspartame gum, organic maple)

5. Fried foods

While items that are battered, crusted or deep-fried may be at the top of the comfort foods list, they can be damaging to the brain. A study of over 18,000 people found that a diet high in fried foods was linked to lower scores of memory and cognition. As an alternative, I suggest opting for baked, air-fried, or steamed versions of your favorite foods.

DIET VS. EXERCISE



Fitness: Which has more impact on long-term health — diet or exercise?

In a perfect world, we'd exercise and sit down to a homemade meal most if not all days of the week. But we're not perfect - far from it. study from Tehran University of Medical Sciences that compared the influence of four distinct lifestyles - active with a healthy diet, inactive with a healthy diet, active with an unhealthy diet and inactive with an unhealthy diet - on the mortality of 17,641 men and 23,051 women age 40 and older over a period of 13.9 years.

"Our study demonstrated a reduction of 21(BOTH), 16(EXERCISE) and 9 (DIET) percentage (points) in all-cause mortality in participants who engaged in both healthy behaviours, only physical activity and only a healthy diet, when compared to those with both unhealthy behaviours respectively," stated the researchers. Diet was analyzed based on the consumption of fruits, vegetables, low-fat dairy, whole grains, nuts and legumes, red and processed meat,

Diet was analyzed based on the consumption of fruits, vegetables, low-fat dairy, whole grains, nuts and legumes, red and processed meat, sugar, sweetened drinks and sodium. Physical activity was measured by calculating the amount of time spent being sedentary and in exercise of light, moderate and vigorous intensity. Also included in the data collection was personal information like gender, age, marital status, ethnicity, occupation, education, socio-economic status and medical history, and daily habits like smoking and drinking.

But the results weren't replicated when the researchers narrowed the scope to just cardiovascular disease. Only individuals with an active lifestyle reduced their risk of dying from heart disease. Diet — even a healthy one — had no impact on mortality.

Open Access

Published: 16 November 2022

Comparing the risk of cardiovascular diseases and all-cause mortality in four lifestyles with a combination of high/low physical activity and healthy/unhealthy diet: a prospective cohort study

Duplication, citation or distributio#98 f this material in whole or in part

- Asma Kazemi, Najmeh Sasani, Zeinab Moki 📶 Auto PRUTNISS 191. Value PRUTNISS 191. Value PRUSA PROVIDE A Malekzadeh

EXERCISE AND NEURODEGENERATION



Exciting new study shows how exercise protects against Alzheimer's and Parkinson's

Mar. 7, 2023:

Cathepsin B (CTSB), a myokine, and brain-derived neurotrophic factor (BNDF) have been found to possess robust neuroprotective effects. In a new study presented in the special issue, investigators looked at whether increasing aerobic exercise intensity would increase the amount of CTSB and BDNF circulating in the blood. Sixteen young healthy subjects completed treadmill-based aerobic exercise at maximum capacity and then at 40%, 60%, and 80% of capacity.

Circulating CTSB and BDNF were measured in blood samples taken after each bout of exercise, and CTSB protein, BDNF protein, and mRNA expression were measured in skeletal tissue. Researchers found that high intensity exercise elevates circulating CTSB in young adults immediately after exercise, and that skeletal muscle tissue expresses both message and protein of CTSB and BDNF.

"CTSB and BDNF are promising therapeutic targets that may delay the onset and progression of cognitive impairments," said lead investigator Jacob M. Haus, PhD, School of Kinesiology, University of Michigan.

Special Issue: Peripheral Factors and Neural Plasticity Guest Editors: Henriette van Praag and Christiane D. Wrann Brain Plasticity, Volume 8, Issue 1 409 Openly available at https://content.iospress.com/journals/brain-plasticity/8/1

RECEPTOR ACTIVATION AND NEUROPLASTICITY



Exercise Alters Brain Chemistry to Protect Aging Synapses

dNeuroscience 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

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OPTIMIZATION





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LLLT AND BRAIN



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

Wei-Tong Pan # 1

Conclusion

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.

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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.

LASERS AND OPTIMIZATION



Abstract

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?

Cleber Ferraresi 1 2 3, Ying-Ying Huang 1 2, Michael R Hamblin 1 2 4

KIRK GAIR SEMINAR FOR SPORTS RECOVERY AND PERFORMANCE

Affiliations expand Duplication, citation or distribution of this material in whole of A. • PMID: 27874264 PMCID: PMtboth كفته المعادية والمعادية والم

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 FOR 3 OLYMPICS! 4/20/24

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



LASERS AND BRAIN OPTIMIZATION



ERCHONIA

Int J Geriatr Psychiatry. 2018 Nov 26. doi: 10.1002/gps.5039. [Epub ahead of print]

Photobiomodulation improves the frontal cognitive function of older adults. Chan AS1,2, Lee TL1, Yeung MK1, Hamblin MR3,4,5.

Author information

Abstract

OBJECTIVES:

The frontal lobe hypothesis of age-related cognitive decline suggests that the deterioration of the prefrontal cortical regions that occurs with aging leads to executive function deficits. Photobiomodulation (PBM) is a newly developed, noninvasive technique for enhancing brain function, which has shown promising effects on cognitive function in both animals and humans. This randomized, sham-controlled study sought to examine the effects of PBM on the frontal brain function of older adults.

METHODS/DESIGNS:

Thirty older adults without a neuropsychiatric history performed cognitive tests of frontal function (ie, the Eriksen flanker and category fluency tests) **before and after a single 7.5-minute session of real or sham PBM.** The PBM device consisted of three

RESULTS: SIGNIFICANT GROUP (EXPERIMENTAL, CONTROL) × TIME (PRE-PBM, POST-PBM) INTERACTIONS WERE FOUND FOR THE FLANKER AND CATEGORY FLUENCY TEST SCORES. SPECIFICALLY, ONLY THE OLDER ADULTS WHO RECEIVED REAL PBM EXHIBITED SIGNIFICANT IMPROVEMENTS IN THEIR ACTION SELECTION, INHIBITION ABILITY, AND MENTAL FLEXIBILITY AFTER VS BEFORE PBM. CONCLUSIONS: OUR FINDINGS SUPPORT THAT PBM MAY ENHANCE THE FRONTAL BRAIN FUNCTIONS OF OLDER ADULTS IN A SAFE AND COST-EFFECTIVE MANNER.

LASERS AND BRAIN OPTIMIZATION



Front. Syst. Neurosci., 14 March 2014 | https://doi.org/10.3389/fnsys.2014.00036

Augmentation of cognitive brain functions with transcranial lasers

F. Gonzalez-Lima: and Douglas W. Barrett

Department of Psychology and Institute for Neuroscience, University of Texas at Austin, Austin, TX, USA

The purpose of this paper is to provide an update on LLLT's neurochemical mechanisms

supporting transcranial laser stimulation for cognitive-enhancing

applications. We will explain first LLLT's action on brain bioenergetics, briefly describe its bioavailability and dose-response, and finish with its beneficial effects on cognitive functions. Although our focus is on prefrontal-related cognitive functions, in principle LLLT should

be able to modulate other brain functions. For example, **stimulating different** brain regions should affect different functions related to sensory and motor systems.

STACKING THERAPIES FOR BRAIN OPTIMIZATION







istribution of this mate Trevor Berry, DC, DA

STACKING THERAPIES FOR BRAIN OPTIMIZATION



WATCH DR. BERRY ON THE DANICA PATRICK PODCAST: PRETTY INTENSE FEBRUARY 9TH, AND THE "LASER LIGHT SHOW"



MASTER BRAIN SETTING



WHEN IN DOUBT, DEFAULT TO THE MASTER BRAIN PROTOCOL FOR ANY NEUROLOGICAL DISORDER INCLUDING DEMENTIAS LIKE ALZHEIMER'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 3-3-3-2-2 up Kton Rate or a strighting of the material Group or in CR EVRL without permission of Trevor Berry, DC, DACNB is prohibited.

without permission of Trever Berry, DC, DACNE is prohibited MASTER BRAIN SETTING

BASIC BRAIN PROTOCOLS REVIEW FOR HANDS ON:

TRANSCRANIAL BASIC 101 SETTING SUGGESTIONS-



ERCHONIA

OLD FX 635 1-40-10 *10 MINUTES RED* SAGITTAL MOHAWK 1 FRONT 10 BRAIN STEM or TURN TO CROSS TEMPORAL AT 5 MINUTES (NEW FX 1-40 PFC, 40-60 APICAL, 10-10 BASE OF SKULL) 6 MINUTES VIOLET/RED FX 405 (3&3) (1-40 FOR APICAL DIODE)

XLR8 1-10-40-60: 3 MINUTES PFC, 3 MINUTES APICAL, 3 MINUTES STEM/BASE OF SKULL, 2 MINUTES SCM/VAGAL, 2 MINUTES STOMACH

EVRL OR GVL 1-10-40-60: 2 MINUTES PFC, 2 MINUTES APICAL, 2 MINUTES STEM/BASE OF SKULL, 2 MINUTES SCM VAGAL, 2 MINUTES STOMACH

PL TOUCH 1-10-40-60 STAND-2 MIN EACH SIDE CROSS-CEREBELLAR FRONTAL, 2 MIN VAGAL/PARIETAL 421 WHEN IN DOUBT ANY NEUROLOGICAL APPLICATION USE 1-10-40-60

DR. BERRYS MASTER LASER SELF CARE



STACK WITH MEDITATION GRATITUDE THEN PURE MEDITATION

USE TEXTURED PURSUITS TO PROMOTE MIDLINE STABILITY PARIETAL LOBE AND VERMAL CEREBELLUM ARE BIG DRIVERS OF PURSUITS VERTICAL PURSUITS WILL HELP DRIVE POSTURAL CURVES



REGIONAL BRAIN OVERVIEW



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While an injury may occur in a specific area, it is important to understand that the brain functions as a whole by interrelating its component parts, Duplication, ci

	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			
femporal Speech (understanding obe language) Memory Hearing Sequencing Organization		Difficulty understanding language and speaking (aphasia) Difficulty recognizing faces Difficulty identifying / naming objects Problems with short- and long-term memo 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			

without permission of Trever Berry, DC, DACNB is BINAURAL BEATS



A recent study published in *Frontiers in Human Neuroscience* has revealed promising findings regarding the ability of inaudible binaural beats to profoundly affect human brain function and induce states of relaxation.

The results indicated that binaural beats significantly impacted the participants' brain wave **activity**, demonstrating the potential for developing musical products that leverage the inaudible sound frequencies to affect human neural rhythms and states of consciousness.

In the example, a binaural beat of 10 Hz would be produced based on the differing frequencies of 440 Hz and 430 Hz.

Specifically, some research has suggested that binaural beats can stimulate brain wave activity to alter states of consciousness to promote relaxation, sleep, or concentration.

The chosen frequencies for the binaural beats were Theta at 4 Hz and Alpha at 8 Hz, which are associated with deep meditative states. The resulting track, titled "Relax

1," served as the auditory stimulation for the study.

Front. Hum. Neurosci., 05 May 2023 Sec. Sensory Neuroscience Volume 17 - 2023 [https://doi.org/10.3389/fnhum.2023.1138650 This article is part of the Research Topic Methods and Protocols in Sensory Neuroscience View all 3 Articles TRIFECTA FINISH: ERCHONIA TRANSCRANIAL LLLT FREQUENCY MATCHED BINAURAL BEATS OR BRAIN TAP OXYGEN CONCENTRATOR

Case report: binaural beats music assessment experiment

Elizabeth Krasnoff1°

OVERTRAINING



If You're a Triathlete (at Any Level), These RED-S Guidelines Should Scare You

The International Olympic Committee just came out with an update on Relative Energy Deficiency in Sport (shortened to REDs or RED-S) - and endurance athletes are firmly in their crosshairs.

The 2023 statement has been expanded to incorporate new data since their last one in 2018: If you thought that RED-S was something that only skinny, elite, women need to worry about, think again. Athletes of all genders and abilities need to pay attention to both what they are eating and how much to avoid injury, illness, and a whole lot of lousy workouts (or worse). Warning – be prepared to get hit with some truth bombs.

When we do not take in enough calories to cover what we burn through exercise, there isn't enough energy left over to cover our basic biological processes.

RELATIVE ENERGY DEFICIENCY IN SPORT (REDs or RED-S)

LOW ENERGY AVAILABILITY

Impaired reproductive functionMental health issuesReduced immunityImpaired bone healthImpaired growth & developmentImpaired gastrointestinal functionReduced skeletal muscle functionImpaired energy metabolism, regulationImpaired cardiovascular functionImpaired hematological functionSleep disturbancesUrinary incontinenceImpaired neurocognitive functionImpaired glucose & lipid metabolism

RED-S & SPORT PERFORMANCE

Decreased athlete availability Decreased power performance Decreased endurance performance Decreased muscle strength Decreased training response Decreased recovery Decreased cognitive performance/skill

OVERTRAINING



2023 updates on RED-S

The following is a summary of the most important points of the IOC's 2023 statement. Importantly, these guidelines no longer

apply to just elite and professional athletes. Any person who exercises at a high level, (ie: triathlon training of any distance) without adequate refueling can end up with RED-S.

PSYCHOLOGICAL INDICATORS OF LEA/RED-S

Mood disturbances/fluctuations

Cognitive dietary restraint

Drive for thinness

Reduced sleep quality

Perfectionistic tendencies

Depressive symptoms and affective disorders

Primary or secondary exercise dependence/addiction

Anxiety related to injury and/or recovery

Sport-specific issues, such as difficulty coping with weight

SLEEP





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without permission of Trever Berry, DC. DACNB is prohibited. 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 <u>30% increase in dementia risk</u> 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

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 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 WASTES 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 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)
- 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
- MAGNESIUMS FOR SLEEP #1 LYSINATE, #2 L THREONATE
- FASTING? DYSGLYCEMIA, PREGNENOLONE
- INFLAMMATION SOURCES AND MRA

MATTRESS RECOMMENDATIONS CONSUMER REPORTS 2022



INNERSPRING:

1.AVOCADO GREEN (BEST OVERALL RATED)

2. DENVER MATTRESS DOCTORS CHOICE PLUSH 3. CASPER ORIGINAL HYBRID

FOAM:

1.ESSENTIA STRATAMI 2.CASPER ORIGINAL 3.SLEEP ON LATEX PURE GREEN FIRM

ADJUSTABLE AIR 1.SLEEP NUMBER 360 P6 SMART 2.SAATVA SOLAIRE 3.PERSONAL COMFORT H10 BED

ANOTHER CONSIDERATION: THE SS500 COMFORT PREMIUM BASE "SUPERIOR SLEEP EXPERIENCE" COOL GEL ADJUSTABLE BED

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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

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Front Hum Neurosci. 2018; 12: 387. Published online 2018 Sep 24. doi: <u>10.3389/fnhum.2018.00387</u>

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. ²ublished online 2019 Dec 10. doi: <u>10.4103/jfmpc.jfmpc_575_19</u>

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 Kumar 1 Conclusion:

It was concluded that 432 Hz music has some significant calming effect as reflected by increased

Nature

alpha activities

. 2006 Nov 30;444(7 Boosting sl Lisa Marshall 1. Hall Here we show that i (0.75 Hz) during enhances th healthy hum endogenous cortica **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)

potentials wave sleep, ries in e sleep,

aps.

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 distributio#96f this material in whole or in part

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iplication, citation of distribution of this material in whole or in p without percession of texenseny, DC, DACNB is prohibited. 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 <u>promotes sleep</u> 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

June 8, 2016

GVL CONSIDERATION: GREEN BEAM 2 MIÑ[®] APPLICATION RETINAL BEFORE BEDTIME

SOME OF DR. BERRY'S FAVORITE DOCUMENTARIES



-FED UP (SUGAR INDUSTRY)

-FOOD INC. (FOOD INDUSTRY AND MONSANTO)

-KISS THE GROUND (A FAIR REPRESENTATION ABOUT THE ENVIRONMENT)

-FANTASTIC FUNGI (HOW IMPORTANT FUNGI ARE TO US AND THE PLANET)

-HOW TO CHANGE YOUR MIND (MICHAEL POLLAN - BOOK OR NETFLIX DOCUMENTARY SERIES ON PSYCHEDELICS)

-CRIME OF THE CENTURY (BIG PHARMA AND THE OPIOID CRISIS)

-AMERICAN ADDICT (BIG PHARMA AND OUR ADDICTION TO PRESCRIPTIONS)

-PRESCRIPTION THUGS (BIG PHARMA AND OUR ADDICTION TO PRESCRIPTIONS)

-INNER WORLDS OUTER WORLD'S (AN IN DEPTH EXPLORATION INTO SPIRITUALITY AND SCIENTIFIC PRINCIPLES) -ANYTHING FROM TERRANCE MCKENNA OR PAUL STAMETS

PODCASTS: JOE ROGAN EXPERIENCE, DANICA PATRICK - PRETTY INTENSE, HUBERMAN LAB (ANDREW HUBERMAN)

THE LASER LIGHT SHOW! EXCELLENT PODCAST SERIES INCLUDING GREAT TALKS ON ERCHONIA LASERS Duplication, citation or distributio#96 fthis material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



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

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COST IDEAS PER TREATMENT: XLR8 \$40/TREATMENT (MIN \$20) EVRL \$50/TREATMENT (MIN \$30) GVL \$60/TREATMENT (MIN \$40) FX 635 >\$85/TREATMENT FX 405 \$100/TREATMENT (\$500 PREPAY CASH - \$85/VISIT) ZERONA \$150+/VISIT 8 VISIT TREATMENT BASE = \$1200. PREPAY CASH = \$1000 (NOT INCLUDING OTHER SUPPLEMENTS/LABS)





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 439

ALZHEIMER'S OVERVIEW

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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 <u>WWW.AZCHIRONEURO.COM</u> <u>AZCHIRONEURO@GMAIL.COM</u>

(480) 756-2600

TELEDOC AND COMPLEX CASE CONSULTATIONS INTENSIVES TO SET UP BASELINÉ



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 ADVANCED NEUROLOGICAL SEMINARS GO TO ERCHONIA.COM AND GET REGISTERED FOR ONE OF OUR EVENTS IN 2023 AND WATCH FOR AFN TRAINING!



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APPLIED FUNCTIONAL NEUROLOGY



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THREE PRIMARY TRACTS: 1. NEURODEGENERATIVE CONDITIONS 2. TRAUMATIC BRAIN INJURY/STROKE 3. NEURODEVELOPMENTAL

KEEPING YOUR BRAIN YOUNG



The Top 5 Ways to Keep Your Brain Young

1. STIMULATE THE BRAIN

APPS (SEE NEXT PAGE), CROSSWORDS, PUZZLES, MAZES, GAMES, ART AND CRAFTS, INSTRUMENT/MUSIC, CONTINUING EDUCATION. LEARNING NEW HOBBIES OR SKILLS

2. MOVE!

ANY MOVEMENT IS BETTER THAN NONE. 3 MINUTE HIIT TRAINING, COMPLEX MOVEMENT PATTERNS LIKE TAI CHI OR YOGA, SOME ENDURANCE IS GOOD (WATCH FOR THE EXERCISE PARADOX - TARGET 23-37 MINUTES), DANCE APP IDEAS: 7 MINUTE WORKOUT, MAP MY RUN. USE SMART WATCHES FOR FITNESS AND BIOMETRICS

3. CALORIC RESTRICTION

FEWER CALORIES IN GENERAL IS BENEFICIAL TO THE BRAIN. 24 HOUR WATER FASTS ARE A GREAT HABIT (1 EVERY 2-3 WEEKS). 16:8 RULE OF EATING (NOT ALWAYS INDICATED). GRAZING ON GOOD FATS, PROTEINS AND VEGGIES (ORGANIC) IS JUST AS IMPORTANT AS FASTING. FASTING FROM THE SAD DIET IS IMPORTANT BUT REGULAR FUEL CONSUMPTION OF HEALTHY FATS, PROTEINS AND FIBER IS JUST AS IMPORTANT. 3+ DAY WATER FAST RESETS - BONE BROTH RETURN. APP: 21-DAY HERO

4. BUILD AND MAINTAIN SOCIAL NETWORKS

"THE NUN STUDY". DON'T BECOME A RECLUSE. KEEP WORKING OR VOLUNTEER. "MEETUP" IS A GOOD RESOURCE TO MEET LIKE MINDED PEOPLE, SHARING HOBBIES AND MORE. BUMBLE BFF IS AN APP FOR MEETING PEOPLE AND BUILDING A SOCIAL NETWORK

5. KEEP ANXIETY AND STRESS TO A MINIMUM

SHORT BURSTS OF STRESS ARE OK, OR EVEN BENEFICIAL (THINK HIIT TRAINING AND FASTING) BUT CHRONIC STRESS IS NOT! GET OUT OF BAD RELATIONSHIPS, JOBS ETC. KEEP YOUR SCHEDULE BALANCED AND MANAGEABLE. "TALKSPACE" IS A GOOD REMOTE THERAPY SERVICE AND LOW COST ALTERNATIVE TO IN OFFICE THERAPISTS. ANXIETY MANAGEMENT APPS INCLUDE "ROOTD" WHICH EVEN HAS A PANIC BUTTON FEATURE FOR IMMEDIATE CBT. "WYSA" AND "PERSONAL ZEN" ARE OTHER APP IDEAS. BREATHING EXERCISES 4:4:8 OR 4:7:8. GROUNDING AND MEDITATION (LASER COMBO!)

SCHEDULE THESE INTO POURI OAVEN 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)

CONSIDER A SECOND LANGUAGE APP:

- **BABBEL**
- DUOLINGO
- ROSETTA STONE
- MONDLY

https://www.focusbuilderapp.com/shop

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Contact <u>NeurdSolutions@gmail.com</u> to get a Focus Builder App for Free Promo Code for iOS only (Not for Android).

Available in the App Store only.

RECOMMENDED CONTINUING EDUCATION FOR NEUROLOGY OF EYE MOVEMENTS:

- 1. NEUROLOGY OF EYE MOVEMENTS SECTION UNDER DOCTOR'S LOUNGE AT <u>WWW.AZCHIRONEURO.COM</u>. FREE FOR SEMINAR ATTENDEES THAT PURCHASE AN ERCHONIA LASER AT ONE OF HIS EVENTS
- 2. FOCUS BUILDER SEMINARS BY DR. NOEL AND DR. WHITAKER

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- 3. COMING IN 2024, ADVANCED TRAINING MODULES THROUGH AFN!
- 4. CARRICK INSTITUTE TBUNGOUEES/NEUROLOGY OF EYEMOVEMENTS BY LEIGH AND ZEE without permission of Trevor Berry, DC, DACNB is prohibited.

VIBRANT LABS ORDERING





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Now offering three unique options to enhance your shopping experience! We prioritize your convenience and aim to make your purchasing process as smooth as possible.



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We provide you with the flexibility to make immediate purchases without a full upfront payment, easing financial strain and allowing you to manage your budget effectively.



ABILITY TO USE HSA/FSA ACCOUNTS

Now you can maximize the benefits of your healthcare savings account, making it easier for you to access the products you need while saving money on eligible expenses.

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Enjoy a seamless and secure checkout experience. With just a few taps, you can conveniently complete your purchase using your saved payment information, ensuring a smooth and hassle-free transaction process

*Patients must have either signed in or verified their phone number on the

VIBRANT WELLPROZ PRICES INCLUDE RESULTS CONSULTATION WITH DR. BERRY

NEW BOOK SUGGESTIONS





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without permission of Trever Berry, DC, DACNB is prohibit PARKINSON'S DISEASE



Parkinson's disease is 50% more common than previously reported, researchers say

For the study, researchers examined Parkinson's incidence from 2012 healthcare data in North America. The data included cohorts of 6.7 million person-years of adults aged 45 and older and 9.3 million person-years of adults aged 65 and older.

The researchers found that 60,0000–95,000 people were diagnosed with PD in North America in 2012, significantly more than previous estimates of <u>40,000–</u>60,000

Trusted Source

Advanced age was strongly linked to an increased rate of PD, and **males were consistently more likely to develop the condition than females.**

Open Access

8, Article number: 170 (2022)

Published: 15 December 2022

Incidence of Parkinson disease in North America

 A. W. Willis, E. Roberts, J. C. Beck, B. Fiske, W. Ross, R. Savica, S. K. Van Den Eeden, C. M. Tanner & C. Marras on behalf of the Parkinson's Foundation P4 Group npj Parkinson's Disease volume
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BIOMARKERS OF PARKINSON'S DISEASE



Blood, CSF Marker Detects Parkinsonian Disorders — Enzyme also identified preclinical Lewy body disease

CSF levels of DOPA decarboxylase (DDC) -- an enzyme that converts levodopa into dopamine -- accurately identified patients with Lewy body disease

"This study shows for the first time that DDC is elevated in both cerebrospinal fluid and blood in patients with Parkinsonian disorders, including Parkinson's disease, Lewy body dementia, progressive supranuclear palsy, and multiple system atrophy," Hansson told *MedPage Today*. "We even found that the levels were increased before symptom onset and could predict subsequent development of clinical disease."

DDC, also known as aromatic L-amino acid decarboxylase, identified preclinical Lewy body disease in unimpaired individuals who had a positive alpha-synuclein seed amplification assay (AUC 0.81), the researchers said. It predicted progression to clinical Lewy body disease over a 3-year period in preclinical cases (HR 3.7 per standard deviation change, 95% CI 1.1–12.7, *P*=0.035).

DDC levels were increased in atypical Parkinsonian disorders but not in other neurodegenerative disorders like Alzheimer's disease, frontotemporal dementia, or vascular dementia.

Open Access

Published: 18 September 2023

DOPA decarboxylase is an emerging biomarker for Parkinsonian disorders including preclinical Lewy body disease

• Joana B. Pereira,

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CASE STUDY: USING LASERS DIAGNOSTICALLY PATIENT HAD BEEN GIVEN PARKINSON'S DIAGNOSIS...





- -PATIENT IS AN INTEGRATIVE MEDICAL DOCTOR WHO FLEW IN FROM CALIFORNIA 5/22/22 - WAS GIVEN THE DIAGNOSIS OF A
- "PARKINSON'S LIKE BALANCE DYSFUNCTION" AND RX SINEMENT. THIS CARBI-LEVODOPA RX SEEMED TO HELP GAIT AND BALANCE INITIALLY BUT HAS BECOME MARKEDLY WORSE IN THE PAST YEAR!
- -HE HAD MILD CONSTIPATION ORIGINALLY BUT WITH SINEMENT HE HAS TO TAKE 9 LAXATIVES!
- -HE FAILED THE PROPRIOCEPTIVE SECTION ON CDP
- -VNG SHOWED HORIZONTAL SQUARE WAVE JERKS, LEAKY NI ON LATERAL GAZE HOLDING AND SEVERE FATIGUE ON ALL TESTS. SACCADES (BOTH VERTICAL AND HORIZONTAL) WERE SLOW, LATENT AND HYPOMETRIC
- -PATIENT IS A VEGAN WITH MEGALOBLASTIC ANEMIA PATTERN AND SEVERE METABOLIC FATIGUE ON TESTING. IS ADDING METHYLATED B'S AND OMEGA 3 FATTY ACIDS

CASE STUDY: USING LASERS DIAGNOSTICALLY PATIENT HAD BEEN GIVEN PARKINSON'S DIAGNOSIS...



DIAGNOSTIC TREATMENT (MASTER BRAIN) SETTING EVRL (1-10-40-60 FOR ALL AREAS):

- 1. CEREBELLUM/BASE OF SKULL 4 MINUTES. STACKED WITH CARTESIAN CROSS "NO-NO" GAZE STABILIZATION EXERCISES, GLUT ISOMETRICS AND LEFT HAND COMPLEX MOVEMENT PATTERN IN BETWEEN CARTESIAN AND 4:4:8 BREATHING
- 2. PRE-FRONTAL LASER 4 MINUTES WITH GAIT VISUALIZATION THEN FAST FINGER TO NOSE
- 3. VAGAL NERVE STIMULATION PROTOCOL (2 MIN LASER SCM WHILE PERCUSSING GUT THE 2 MINUTES OVER GUT)
- 4. LOWER EXTREMITY STACKING LASER FROM ANKLE UP THE LEG WITH HIGH SPEED PERCUSSOR (2 MINUTES EACH LIMB)

PATIENT IS PURCHASING AN EVRL AND PERCUSSOR FOR PERSONAL USE (AND WANTS TO IMPLEMENT IT INTO HIS PRACTICE) Duplication, citation or distribution of this materia without permission of Trevor Berry, DC, DACK





*Low Catecholamines - I Mental Speed, Alertness, Concentration, Cognition, Distracted, Caffeine Helps Mental Function

PARKINSON'S ETIOLOGY



"The study's findings suggest that exposure to **trichloroethylene** in water may increase the risk of Parkinson's disease. Millions worldwide have been and continue to be exposed to this ubiquitous environmental contaminant,"

Interestingly, TCE was also found in decaffeinated coffee. However, it has been banned by the food and pharmaceutical industries since the 1970s. Despite being removed from dry cleaning processes in the mid-1950s, <u>TCE is still used in metal</u> <u>cleaning, degreasing, and as an extraction solvent in the textile</u> <u>manufacturing industry.</u>

May 15, 2023

ERCHONIA

Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune

Samuel M. Goldman, MD, MPH^{1,2}; Frances M. Weaver, PhD^{3,4}; Kevin T. Stroupe, PhD^{3,4}; <u>et al</u> Lishan Cao, MS³; Beverly Gonzalez, PhD⁵; Kalea Colletta, DO⁵; Ethan G. Brown, MD^{2,5}; Caroline M. Tanner, MD, PhD^{2,5} Author Affiliations <u>Article Information</u>

JAMA Neurol. Published online May Ruplication, citation or distributio4796f this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



Parkinson's Breakthrough: Scientists Have Identified a Key Molecule

Adenosine, a neurotransmitter, has been found to act as a brake on dopamine, another neurotransmitter involved in motor

control, by researchers at Oregon Health & Science University. The findings, which were published in the journal Nature, reveal that adenosine and dopamine operate in a push-pull dynamic in the brain.

"There are two neuronal circuits: one that helps promote action and the other that inhibits action," said senior author Haining Zhong,

Ph.D., a scientist with the OHSU Vollum Institute. **"Dopamine promotes the first circuit to** enable movement, and adenosine is the 'brake' that promotes the second circuit and brings balance to the system."

Notably, adenosine is also well known as the receptor that caffeine acts upon.

"Coffee acts in our brain through the same receptors," Mao said. "Drinking coffee lifts the brake imposed by adenosine."

While drinking caffeine each day does not appear to help improve sleepiness among people with Parkinson's disease, it may have a benefit in controlling movement, according to new research published in the August 1, 2012, online issue of Neurology®, the medical journal of the American Academy of Neurology.

"Studies have shown that people who use caffeine are less likely to develop Parkinson's disease, but this is one of the first studies in humans to show that caffeine can help with movement symptoms for people who already have the disease," said study author Ronald Postuma, MD, MSc, with McGill University in Montreal and the Research Institute of the McGill University Health Center. Postuma is also a member of the American Academy of Neurology.

References: "Locomotion activates PKA through dopamine and adenosine in striatal neurons" by Lei Ma, Julian Day-Cooney, Omar Jáidar Benavides, Michael A. Muniak, Maozhen Qin, Jun B. Ding, Tia⁵⁷yi Mao and Haining Zhong, **9 November 2022,** *Nature*.

PARKINSON'S DISEASE



Study: Two New Early Signs of Parkinson's Disease Identified

The findings may help health care providers diagnose and begin treating patients earlier.

Hearing loss and <u>epilepsy</u> have been identified as two new, early signs of <u>Parkinson's disease</u>, according to new research published in <u>JAMA</u> <u>Neurology</u>.

THE PRIOR PD TRIAD: - CONSTIPATION -ASYMMETRIC SHOULDER PAIN/STIFFNESS NOT RESOLVING FROM STANDARD CARE - LOSS OF SMELL

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- Researchers have found a distinct genetic signature in the immune cells of people with Parkinson's disease.
- The genes in question are associated with oxidative stress, inflammation, and other immune-related changes.

"The evidence that [Parkinson's disease] may partially be an autoimmune disease is just beginning to accumulate," said <u>James Beck, Ph.D.</u>, chief scientific officer of the Parkinson's Foundation in the United States.

Inflammation is associated with Parkinson's, and it may be a result of autoimmune processes, he told *Medical News Today*.

Memory T cells are immune cells that remember the specific molecular features of past infections or autoimmune reactions.

"But all of our work points toward T cells having a role in the disease," she adds.However, whether autoimmunity actually causes the disease has yet to be proven.

Open Access Published: 21 March 2022

Transcriptional analysis of peripheral memory T cells reveals Parkinson's disease-specific gene signatures

Rekha Dhanwani, João Rodrigues Lima-Junior, Ashu Sethi, John Pham, Gregory Williams, April Frazier, Yagian Xu, Amy W. Amara, David G. Standaert, Jennifer G. Goldman, Irene Litvan, Roy N. Alcalay, Bjoern Peters, David Sulzer, Cecilia S. Lindestam Arlehama & Alessandro Sette
 npj Parkinson's Disease
 volume

volume 8, Article number: 30 (2022)

PARKINSON'S DISEASE



Eating berries may protect against Parkinson's disease

Men and women who regularly eat berries may reduce their risk of developing Parkinson's disease, according to a new study from Harvard School of Public Health (HSPH). The researchers, led by Xiang Gao, research scientist in HSPH's Department of Nutrition, believe the

high flavonoid content in berries, apples and oranges help ward off the disease. Study participants who consumed the most flavonoids were 40% less likely to develop Parkinson's.

Can three or more servings of berries a week help lower risks of Parkinson's disease?

It is truly possible, according to the new studies published in Neurology.²

Men and women that consume berries, three or more times a week are nearly 27% less likely to develop Parkinson's disease, than those who have less than two servings per month.

Exactly how berries can help lower risk is still not known, but they are rich in antioxidants, known as **flavonoids**, who could protect brain cells from damages. Flavonoids are usually

Front. Aging Neurosci., 03 April 2020 | https://doi.org/10.3389/fnagi.2020.00089

Mitochondrial Dysfunction and Parkinson's Disease —Near-Infrared Photobiomodulation as a Potential Therapeutic Strategy

Aaron Song Chuan Foo1,2, Tuck Wah Soong1, Tseng Tsai Yeo2 and Kah-Leong Lim3,4*

As the main driver of energy production in eukaryotes, mitochondria are invariably implicated in disorders of cellular bioenergetics. Given that dopaminergic neurons affected in Parkinson's disease (PD) are particularly susceptible to energy fluctuations by their high basal energy demand, it is not surprising to note that mitochondrial dysfunction has emerged as a compelling candidate underlying PD. A recent approach towards forestalling dopaminergic neurodegeneration in PD involves nearinfrared (NIR) photobiomodulation (PBM), which is thought to

enhance mitochondrial function of stimulated cells through augmenting the activity of cytochrome C oxidase.

LOW LEVEL LASER FOR ALZHEIMER'S AND PARKINSON'S DISEASE: GOOD REVIEW PAPER



ERCHONIA

Turning On Lights to Stop Neurodegeneration: The Potential of Near Infrared Light Therapy in Alzheimer's and Parkinson's Disease

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.

PARKINSON'S DISEASE AND LLLT



Mol Neurodegener. 2009 Jun 17;4:26. doi: 10.1186/1750-1326-4-26.

Reduced axonal transport in Parkinson's disease cybrid neurites is restored by light therapy.

Trimmer PA1, Schwartz KM, Borland MK, De Taboada L, Streeter J, Oron U.

CONCLUSION:

The results from this study support our proposal that axonal transport is reduced in sporadic PD and that a single, brief treatment with near-infrared light can restore axonal transport to control levels. These results are the first demonstration that LLLT can increase axonal transport in model human dopaminergic neuronal cells and they suggest that LLLT could be developed as a novel treatment to improve neuronal function in patients with PD.

<u>J Comp Neurol.</u> 2010 Jan 1;518(1):25-40. doi: 10.1002/cne.22207.

Neuroprotection of midbrain dopaminergic cells in MPTP-treated mice after nearinfrared light treatment.

Shaw VE1, Spane S, Ashkan K, Benabid AL, Stone J, Baker GE, Mitrofanis J. "In summary, our results indicate that NIr light treatment offers neuroprotection against MPTP toxicity for dopaminergic cells in the SNc". **START TREATING YOUR BRAIN** <u>BEFORE</u> **SYMPTOMS START!**

Exp Neurol. 2009 Aug;218(2):320-5. doi: 10.1016/j.expneurol.2009.03.016. Epub 2009 Mar 26. **The cybrid model of sporadic Parkinson's disease.** Trimmer PA1, Bennett JP Jr.

Most PD (approximately 90%) is sporadic (sPD), and is associated with mitochondrial deficiencies Our recent studies show that near-infrared laser light therapy normalizes mitochondrial movement and can stimulate respiration in sBD cybrid neurons, and mitochondrial gene therapy can restore respiration and stimulate mitochondrial ETC gene and protein expression.

PARKINSON'S DISEASE AND LLLT



- > 24 hour water fast 1x/week (minimum every 2 weeks), FIX GUT/LEAKY GUT
- ► ADDRESS DOPAMINE NEUROCHEMISTRY/CO-FACTORS/MICRONUTRIENTS
- LLLT GI AND SCM vagal setting (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 Mastoid vestibular stimulation (4-8, 50-150) 5 minutes. CO-ACTIVATE
- LLLT MASTER BRAIN SETTING 1-10-40-60 (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).
- Initial treatment 3x/week 4 weeks, monitor progress and phase down.
- BIAS TREATMENTS TO FX 405 OR EVRL BUT STACK WITH GVL WHEN AVAILABLE

PARKINSON'S DISEASE AND LLLT



<u>Front Hum Neurosci.</u> **2019 Apr 3**;13:118. doi: 10.3389/fnhum.2019.00118. eCollection 2019. Abnormal Phase Coupling in Parkinson's Disease and Normalization Effects of Subthreshold Vestibular Stimulation.

Lee S1,2, Liu A2,3, Wang ZJ1,4, McKeown MJ2,5.



Resting EEG was collected from 16 PD subjects and 18 age-matched, healthy controls (HC) in four conditions: *sham* (no stimulation), EVS1 (4-8 Hz multisine), EVS2 (50-100 Hz multisine) and EVS3 (100-150 Hz multisine) APICAL DIODE SCAN: 4-8 HZ, BOTH MASTOID DIODES SCAN: 50-150 HZ



*NOTE: Several electrophysiology studies using local field potential (LFP) recordings demonstrated that, in the dopamine-deficient state, the neuronal synchronization in the basal ganglia (BG) is exaggerated at frequencies in the beta range (13–30 Hz) (Brown and Williams, 2005; Eusebio et al., 2009; Litvak et al., 2012; Oswal et al., 2013). These beta oscillations are also highly synchronized with sensorimotor areas (Brown et al., 2001; Marsden et al., 2001; Cassidy et al., 2002; Williams et al., 2002) as well as muscle activity of upper limbs during movement (Marsden et al., 2001). This excessive beta synchronization is considered to be, in part, responsible for the Parkinsonian symptoms and thus reducing the abnormal synchronization with deep brain stimulation (DBS) has

TRAUMATIC BRAIN INJURY





ACUTE PHASE TBI



Summary: Acute Phase TBI Management (<4 Weeks)



24 HOUR WATER FAST ON DAYS 7, 14, 21 AND 28 CONSIDER ATP IGNITE AM CREATINE + COFACTORS AM:

- **1 OMEGA MONOPURE CURCUMIN EC**
- **1 S-ACETYL GLUTATHIONE**
- 1 ACTIV NUTRIENTS WITHOUT COPPER AND IRON
- **1 ACTIV NUTRIENTS PERFORMANCE**
- **1 MONOPURE PRM**
- 1 ALAMAX CR

PM:

- **1 MELATONIN CR**
- 1 NAC
- 1 K2-D3
- **1 HEMP MONOPURE**
- **1 OPTIMAG NEURO**
- **1 RESVERATIN PLUS**

* 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 pa

- 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



RON B.



81 YEAR OLD MALE

MOTOR VEHICLE ACCIDENT

CERVICAL SPINE FRACTURE RESULTING IN A 9 LEVEL CERVICAL TO THORACIC FUSION ON 11/8/23 (2 DAYS POST MVA) RESULTING IN NEAR FULL UPPER AND LOWER EXTREMITY USE

AND COMPLETE BOWEL/BLADDER CONTROL LOSS

HAD BEEN DOING HOSPITAL AND IN HOME OT/PT SINCE SURGERY

FIRST VISIT AT MY OFFICE 1/30/24

COULD NOT DO CDP BALANCE TESTING

VNG SHOWED HORIZONTAL SQUARE WAVE JERKS IN ALL GAZE HOLDING QUADRANTS, LATENCIES AND HYPOMETRIA OF ALL SACCADE DIRECTIONS, AND SACCADIC INTRUSIONS OF ALL PURSUITS

TREATMENT:

INITIAL TREATMENTS FOCUSED ON VAGAL QUAD PROTOCOL, LE REBUILDER, GVL C SPINE STACK WITH EYELIGHTS/HEMISTIM, RPS MINISTIM WITH OCULOMOTOR PRELOAD INTO MOVEMENT PATTERNS (IE MEDIAN NERVE INTO BALL SQUEEZE, ULNAR INTO HAND BALL FLARE, GLUT ISOMETRICS ETC). SEATED VIBE PLATE 40 HZ GLUT ISO AND SEATED CROSS-CRAWL. NSI IM AND PROSACCADES. FX 405 FINISH TCT MASTER WITH CONCENTRATOR AND 40 HZ BINAURAL BEATS. PROGRESSED INTO GAIT TRAINING STACKING (ASSISTED AND UNASSISTED) SEE VIDEOS...

BY 3/14/24 (6 WEEKS OF CARE IN MIDST OF 12 VISIT TREATMENT TRIAL DRIVING UP FROM TUCSON) HE HAD FULL BOWEL CONTROL!

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RON B.





RON VAGAL NERVE STIMULATION FOR BOWEL AND BLADDER CONTROL POST SPINAL CORD INJURY



ERCHONIA



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VIDEOS BELOW ARE FROM 4/2/24 VISIT. HE WALKED 50 FEET IN HOUSE UNASSISTED BY 4/16/24 VISIT! 473

RON END OF TREATMENT FX 405





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RON CAN NOW WALK 60+ FEET





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SENSORY MISMATCH



DO THE EYES, INNER EAR AND PROPRIOCEPTIVE SYSTEMS SAY THE SAME THING THE THE VESTIBULAR NUCLEI AND CORTEX (PIVC RIGHT PARIETAL LOBE)?

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Modified CTSIB

ALANCE	Modified CTSIB (Main Results)	Name: Sample Profile ID#: XXXXXX Facility:	BALANCE	Ma (0	odified CTSIE COP Details)	3	Name: Sample Profile ID#: XXXXXX Facility:
e modified Clinical Test of stural control. The first trial i the BTrackS Balance Plate a "perturbed", foam surface prinception, vision and vest	Sensory Integration and Balance (CTSIB) is the "Standard" condition where balance is t 's firm surface. The second (i.e. eyes closed o), and fourth (eyes closed on a foam surface inular information are respective) used for ha	avaluates sensory contributions to ested with eyes open and two feet on a firm surface), third (eyes open) trials to give an indication of how lance	Modified CTSIB results users may have use, ho trace. These metrics and	are typically determin wever, for additional visuals are provided	ned on the basis of COP metrics and/or below.	Center of Pressure enlarged visualizat	(COP) path length. Some tions of the raw COP data
			Standard (Eyes Open/Firm)	Proprioc (Eyes Close	eption w/Firm)	Vision (Eyes Open/Foam)	Vestibular (Eyes Closed/Foam)
(Eyes Open/Firm)	(Eyes Closed/Firm) VISION (Eyes Closed/Firm) (Eyes Open/Foarm) (Eyes Closed/Foam)	e <u>à 22</u>	4 0		A4	
Path Length (cm)	40 44 Path Length (cm)	n) Path Length (cm)	Ang = -	.1cm .4cm 3deg	ML = 1.5cm AP = 4.1cm Ang = -2deg	ML = 3.6cm AP = 4.2cm Ang = 19deg	ML = 6.8cm AP = 7.7cm Ang = 35deg
0 Percentile=18%	0 100 0 Percentile=11	100 0 100 % Percentile=12%					
18 Path Length (cm)	22 Path Length (cm) Path Length (cm	A A B Path Length (cm)	HL = 0 AP = 2 Ang =	.8cm .2cm 3deg	ML = 1.5cm AP = 2.9cm Ang = 8deg	ML = 1.9cm AP = 5.2cm Ang = 13deg	48 ML = cm AP = cm Ang = deg
Percentile=36% hen compared to healthy adults of four conditions (Standard, Proprio	Percentile=56% Percentile=53% Percentile=53\% Parcentile=53\% Parcen	100 Percentile=84% S showed bottom quartile performance ine mCTSIB test results show bottom	A 95% confidence ellipse is f smallest ellipse that fits 95% The ML and AP metrics give quantifies the ellipse rotation	t to the raw data from ear of the Center of Pressure the COP range in the me to the left (negative) or rig	ch test condition in the gr (COP) test data within i dial/lateral and anterior/p (ht (positive) of vertical. N	aphs above. The ellipse t. Three metrics describ osterior directions. The / IL/AP metrics assume a	s are determined as the ing the data are also provided. Angle (i.e. Ang) metric Ilignment along to the cardinal
artile performance in zero conditio	ons.	is an improvement of 93cm.	axes of the BTrackS Balance	Plate.			
e composite mCTSIB results wer	re 208cm at Baseline and 115cm at Post-Baseline. This						
e composite mCTSIB results wer	re 208cm at Baseline and 115cm at Post-Baseline. This			Ba	aseline Resu	lts	
composite mCTSIB results wer	Baseline Results		DATE	Ba STD (ML,AP,ANG)	Seline Resu PRO (ML, AP, ANG)	VIS (ML,AP,ANG) VEST (ML AP, ANG)
e composite mCTSIB results wer DATE STD	Baseline Results PRO % VIS % VES % COMP %	NOTE	DATE 2/1/2018 15:47	Ba STD (ML,AP,ANG) 1.1, 2.4, -3	PRO (ML,AP,ANG) 1.5, 4.1, -2	VIS (ML,AP,ANG 3.6, 4.2, 19) VEST (ML AP,ANG) 6.8, 7.7, 35
DATE STD 2/1/2018 15:47 22	Baseline Results % PRO % VIS % VES % COMP % 18 40 5 44 11 102 12 208 8	NOTE Initial Visit	DATE 2/1/2018 15:47	Ba STD (ML,AP,ANG) 1.1, 2.4, -3	PRO (ML,AP,ANG) 1.5, 4.1, -2	VIS (ML,AP,ANG 3.6, 4.2, 19) VEST (ML AP,ANG) 6.8, 7.7, 35
DATE STD 2/1/2018 15:47 22	Baseline Results % PRO % VIS % VIS % COMP % 18 40 5 44 11 102 12 208 8	NOTE Initial Visit	DATE 2/1/2018 15:47	Ba STD (ML,AP,ANG) 1.1, 2.4, -3 Post	Baseline Resu PRO (ML,AP,ANG) 1.5, 4.1, -2 Baseline Re	Its VIS (ML,AP,ANG 3.6, 4.2, 19 sults) VEST (ML AP, ANG) 6.8, 7.7, 35
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DATE STD 2/1/2018 15:47 22 DATE STD	Baseline Results % PRO % VIS % VES % COMP % 18 40 5 44 11 102 12 208 8 Post-Baseline Results % PRO % VIS % VES % COMP % 18 40 5 44 11 102 12 208 8 Post-Baseline Results % VES % COMP %	NOTE Initial Visit	DATE 2/1/2018 15:47 DATE 2/8/2018 15:20 3/2/2018 16:24	Ba STD (ML,AP,ANG) 1.1,2.4,-3 Post STD (ML,AP,ANG) 1.2,22,2 1.1,19,-2	Aseline Resu PRO (ML,AP,ANG) 1.5, 4.1, -2 -Baseline Re PRO (ML,AP,ANG) 1.5, 3.8, 4 1.3, 3.6, 2	ts VIS (ML,AP,ANG 3.6,4.2,19 sults VIS (ML,AP,ANG 3.4,4.1,16 2.9,3.6,11) VEST (ML AP,ANG) 6.8,7.7,35) VEST (ML AP,ANG) 6.2,7.2,30 5.8,68,25
DATE STD 2/1/2018 15:47 22 DATE STD 2/1/2018 2/1/2018 15:47 22 DATE STD 2/1/2018 2/1/2018 15:40 23 2/1/2018 16:20 23	Baseline Results 50 PRO 95 VIS 96 VES 96 OOMP 90 18 40 5 44 11 102 12 206 8 Post-Baseline Results 90 PRO 95 VIS 96 VES 96 OOMP 90 18 40 5 44 11 102 12 206 8 Post-Baseline Results 14 39 7 44 11 72 28 28 64 I22	NOTE Initial Visit	DATE 2/1/2018 15.47 DATE 2/8/2018 15 20 3/2/2018 16 24 4/1/2018 14 27	Ba STD (ML, AP, ANG) 1.1, 2.4, -3 Post STD (ML, AP, ANG) 1.2, 2.2, 2 1.1, 1.9, -2 0.9, 1.7, 0	Aseline Resu PRO (ML,AP,ANG) 1.5, 4.1, -2 -Baseline Re PRO (ML,AP,ANG) 1.5, 3.8, 4 1.3, 3.6, 2 1.1, 3.1, -2	Its VIS (ML,AP,ANG 3.6, 4.2, 19 sults VIS (ML,AP,ANG 3.4, 4.1, 16 2.9, 3.6, 11 2.1, 3.6, 10	 VEST (ML AP,ANG) 6.8, 7.7, 35 VEST (ML AP,ANG) 6.2, 7.2, 30 5.8, 6.8, 25 4.2, 6.6, 21
DATE STD 2/1/2018 15.47 22 DATE STD 28/2018 2/8/2018 15.47 22 UATE STD 28/2018 2/8/2018 15.20 23 2/1/2018 16.24 20	Baseline Results %	NOTE http://vst NOTE WK.1.Follow-up WK.4.Follow-up WK.4.Follow-up	DATE 2/1/2018 15.47 2/8/2018 15.20 3/2/2018 16.24 4/1/2018 14.27 4/2/8/2018 15.37	Ba STD (ML,AP,ANG) 1.1,2.4,-3 Post STD (ML,AP,ANG) 1.2,22,2 1.1,1.9,-2 0.9,17,0 0.7,1.6,1	aseline Resul PRO (ML AP ANG) 1.5, 4.1, -2 -Baseline Re PRO (ML AP ANG) 1.5, 3.8, 4 1.3, 3.6, 2 1.1, 3.1, -2 0.9, 2.7, 1	Its VIS (ML, AP, ANG 3.6, 4.2, 19 Suits VIS (ML, AP, ANG 3.4, 4.1, 18 2.9, 3.6, 10 2.1, 36, 10 1.9, 3.3, 10	 VEST (ML AP, ANG) 6.8, 7.7, 35 VEST (ML AP, ANG) 6.2, 7.2, 30 6.8, 6.8, 25 4.2, 6.6, 21 3.3, 5.6, 19
DATE STD 2/1/2018 15.47 22 DATE STD 2/8/2018 15.40 28 3/2/2018 16.44 20 4/1/2018 14.27 18 4/1/2018 14.27 18	PRO 96 PIXIS 66 VES 16 60 97 18 40 5 44 11 102 12 208 8 Post-Baseline Results 90 PRO 90 VIS 16 VES 16 COMP 90 18 40 5 44 11 102 12 208 8 Post-Baseline Results 90 PRO 90 VIS 96 COMP 96 14 39 7 44 11 78 28 184 12 20 36 10 15 70 14 166 22 26 31 15 49 49 60 62 145 37 3 28 31 50 43 25 14 37 3 28 34 35 14 12 25	NOTE Initial Visit W4.1 Follow-up W4.4 Follow-up W4.8 Follow-up W4.9 Follow-up	DATE 2/1/2018 15.47 28/2018 15.20 3/2/2018 15/20 3/2/2018 16/24 4/1/2018 16/24 4/2/2018 15/37 6/2/2018 15/41	Ba STD (ML,AP,ANG) 1.1, 2.4, -3 Post STD (ML,AP,ANG) 1.2, 2.2, 2 1.1, 1.9, -2 0.9, 1.7, 0 0.7, 1.6, 1 0.8, 1.3, 2	Baseline Result PRO (ML,AP,ANG) 1.5, 4.1, -2 Baseline Re PRO (ML,AP,ANG) 1.5, 3.8, 4 1.3, 3.6, 2 1.1, 3.1, -2 0.9, 2.7, 1 0.8, 2.5, 1	Its VIS (ML, AP, ANG 3.6, 4.2, 19 Sults VIS (ML, AP, ANG 3.4, 4.1, 10 2.9, 3.6, 11 2.1, 3.6, 10 1.9, 3.3, 10 1.6, 3.1, 9	 VEST (ML AP, ANG) 68, 7.7, 35 VEST (ML, AP, ANG) 62, 7.2, 30 58, 68, 825 42, 66, 21 33, 58, 19 32, 53, 16
DATE STD 2/1/2018 15.47 22 DATE SID 2/1/2018 15.47 22 2/1/2018 15.47 23 3/2/2018 15.20 23 3/2/2018 15.24 20 4/2/2019 15.27 10 6/2/2018 15.47 11	PRO % VIS % VES % COMP % 18 40 5 44 11 102 12 206 8 Post-Baseline Results % PRO % VIS % VES % COMP % 18 40 5 44 11 102 12 206 8 Post-Baseline Results % PRO % VIS % VES % COMP % 18 39 7 KIS % VES % COMP % 26 36 10 40 15 70 141 166 22 30 23 15 49 60 21 43 30 43 55 71 132 52 61 52 76 119 66 31 25 61 52 61 52 76 132 </td <td>NOTE Initial Visit Wit 1 Follow-up Wit 4 Follow-up Wit 12 Follow-up Wit 12 Follow-up Wit 12 Follow-up</td> <td>DATE 2/1/2018 15:47 2/8/2018 15:20 3/2/2018 15:20 3/2/2018 15:27 4/1/2018 15:37 6/2/2018 15:41 8/1/2018 15:41</td> <td>Ba STD (ML,AP,ANG) 1.1, 2.4, -3 Post STD (ML,AP,ANG) 1.2, 2.2, 2 1.1, 1.9, -2 0.9, 1.7, 0 0.7, 1.6, 1 0.8, 1.3, 2 0.8, 2.2, 3</td> <td>aseline Resu PRO (ML_AP_ANG) 1.5, 4.1, -2 Baseline Re PRO (ML_AP_ANG) 1.5, 3.8, 4 1.3, 3.8, 2 1.1, 3.1, -2 0.9, 2.7, 1 0.8, 2.5, 1 1.5, 2.9, 8</td> <td>Its VIS (ML, AP, ANG 3.6, 4.2, 19 Sults VIS (ML, AP, ANG 3.4, 4.1, 16 2.9, 3.6, 11 2.1, 3.6, 10 1.9, 3.3, 10 1.9, 3.3, 10 1.9, 5.2, 13</td> <td>) VEST (ML AP_ANG) 6.8,7.7,35) VEST (ML,AP_ANG) 6.2,7.2,30 5.8,6.8,25 4.2,6.6,21 3.3,5.8,19 3.2,5.3,16 </td>	NOTE Initial Visit Wit 1 Follow-up Wit 4 Follow-up Wit 12 Follow-up Wit 12 Follow-up Wit 12 Follow-up	DATE 2/1/2018 15:47 2/8/2018 15:20 3/2/2018 15:20 3/2/2018 15:27 4/1/2018 15:37 6/2/2018 15:41 8/1/2018 15:41	Ba STD (ML,AP,ANG) 1.1, 2.4, -3 Post STD (ML,AP,ANG) 1.2, 2.2, 2 1.1, 1.9, -2 0.9, 1.7, 0 0.7, 1.6, 1 0.8, 1.3, 2 0.8, 2.2, 3	aseline Resu PRO (ML_AP_ANG) 1.5, 4.1, -2 Baseline Re PRO (ML_AP_ANG) 1.5, 3.8, 4 1.3, 3.8, 2 1.1, 3.1, -2 0.9, 2.7, 1 0.8, 2.5, 1 1.5, 2.9, 8	Its VIS (ML, AP, ANG 3.6, 4.2, 19 Sults VIS (ML, AP, ANG 3.4, 4.1, 16 2.9, 3.6, 11 2.1, 3.6, 10 1.9, 3.3, 10 1.9, 3.3, 10 1.9, 5.2, 13) VEST (ML AP_ANG) 6.8,7.7,35) VEST (ML,AP_ANG) 6.2,7.2,30 5.8,6.8,25 4.2,6.6,21 3.3,5.8,19 3.2,5.3,16

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.

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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 CEM: DO NOT USE DEPTUBLED SUBJECTS DURING ACUTE PHASE SPINAL OPTHODEDIC
- 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)

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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)
- -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
- -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





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.xCitations: 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-ShuPiaoBogdan A.StoicaJunfangWuBorisSabirzhanovZaoruiZhaoRainierCabatbatDavid J.LoaneAlan 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,

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



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 i	s stopped at 2 minutes,	53% of POTS pati	ents 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 i	s stopped at 5 minutes,	27% of POTS pati	ents will be missed!	
tanding at 6 minutes	88/62 Pulse Pressure: 26	P-132	Palpitations	
e in heart rate	. as well as a v	erv notable	Palpitations	
in pulse pres	sure (the differ	rence betwe	Eingling in face increased, tingling all environments, muted sounds, legs gave in acting out, had to be assisted to	
HR was 68 when su	pine then jumped to 13:	2 (+64 BPM) just b	efore near syncope.	
		11 6 . 10/ 6		

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DYSAUTONOMIA/POTS



THE ENERGY FAILURE MODEL OF NEURODEGENERATION IS ROBUST IN THE LITERATURE. UTILIZING YOUR ERCHONIA WAVELENGTHS FOR AUTONOMIC/BLOOD PRESSURE REGULATION:

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VAGAL NERVE STIMULATION



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.485

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.



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

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.

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DYSAUTONOMIA

IV response.



GARY M. MAYO CLINIC TILT TABLE ANS TESTING

POTS HR ORTHOSTATIC VS BLOOD PRESSURE

Duplication, ci

the distal leg and normal at all	other sites.					
DEEP MANEUVER RESPONSES	BREATHING & VALSALVA					
Result Normal Cutoff						
Deep Breathing Heart 7.9 > 9	Rate Range (bpm)					
Valsalva Maneuver Valsa 1.38 > 1.36	lva Ratio					
Comments on Deep Breathing: Hear breathing were reduced.	t rate responses to deep					
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

BLOOD PRESSURE AND HEART RATE RESPONSES TO TILT BP (mmHg) Heart Rate (BPM) 166/98 Supine Tilt 1 min Tilt 3 min 124/91 127/92 71 82 Tilt 5 min Tilt 5 min Tilt 7 min Tilt 10 min Tilt -1 min 119/90 85 124/87 105/85 149/92 85 86

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)



DYSAUTONOMIA

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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



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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 12 SprizettionResitation 2 and RetributionResitation and Retribution Research without permission of Trevor Berry, DC, DACNB is prohibited.



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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

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<u>H</u>P AXIS





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THYROID





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!

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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.
- **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.



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 I Article ID 126720 I 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 https://www.caused.by.cat.

THYROID



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

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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.

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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 tim lasts, If you have a very much stimula

Additional note: 1 contacted by pat and an ER visit witi laser use in the mc

USE

GVI

BROCK PROTOCOL HYPOTHYROID 20-59-822-3884 HYPERTHYROID 3-20-59-160

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s. Khave been yraid storm it that type of

ERCHONIA

REPRODUCTIVE SYSTEM





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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: <u>10.5978/islsm.12-OR-05</u>

PMCID: PMC3944482 PMID: 24610987 Personal Overview of the Application of LLLT in Severely Infertile Japanese Females

Toshio Ohshiro, MD PhD

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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.

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Biomedicine (Taipei). 2018 Jun; 8(2): 7. Published online 2018 May 28. doi: <u>10.1051/bmdcn/2018080207</u>

PMCID: PMC5992952 PMID: 29806585 male infertility

Effectiveness of low level laser therapy for treating male infertility

Sergey Vladimirovich Moskvin1,* and Oleg Ivanovich Apolikhin2

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, PREGENOLONE 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 Duplication, 5: MINUTES: IRREFORM TAker APRILS or in part without permission of Trevor Berry, DC, DACNB is prohibited.



RCHONIA

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 11,

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 attretic follicles compared to CTX alone. LLLT increased the number of primordial follicles compared to 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.

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REPRODUCTIVE SYSTEM



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

Gonzalo Oubiña 1,

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without permission of Trever 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

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-LOW PROGESTERONE

- DYSGLYCEMIA
- -ANTI-PHOSPHOLIPID ANTIBODIES -> LUPUS AI
- -LOW THYROID/AUTOIMMUNE THYROID
- -SMOKING, ALCOHOL OR DRUG USE
- -CONGENITAL DEFECT/CHROMOSOMAL DISORDER

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- -CERVIX INSTABILITY/UTERINE FIBROIDS
- INFECTIOUS DISEASE (IE. CMV OR STD'S)



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ERECTILE DYSFUNCTION PROTOCOL



- THE CANARY IN THE COAL MINE BLOOD FLOW AND 02 DELIVERY COMPROMISE (CV DISEASE, DIABETES, ANEMIA ETC)
- CHOLESTEROL IS NEEDED!

CIRCULATION SETTING (DAN MURPHY) 20-42-43-53 ABOVE PUBIC BONE SHOCKWAVE THERAPY. IE THE PHOENIX

- PROCREATION VS SURVIVAL SHOCKWAVE T - 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

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



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 COMMON MEDICATIONS THAT CAN CAUSE ED: develop NARCOTICS (RX OR RECREATIONAL) In cultur owth **ANTI-DEPRESSANTS** and redu ANTI-HISTAMINES/H2 BLOCKERS The find afil as **BLOOD PRESSURE MEDICATIONS** a possib PARKINSON'S MEDICATIONS **CANCER/HORMONE MEDICATIONS** A team of res **NSAIDS** ic gs that developed a AND MANY MORE! might be effective against AD. NIT S National Institute of Aging (NIA) supported the study. Results appearie ditation Note the study of the study. Results appearie ditation Note the study of the study of

LLLT AND PROSTATE CANCER



Prostate FLO'

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 515

Jaber Zafari 1, Fatemeh Javani Jouni 2

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 art

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 i 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 34, 3a Ha Lee 5, Dong Hoon Sin 12, Mong Kim 12, Jag Hong Nor 12 without permission of Trevor Berry, DC, DACNB is prohibited.

ADRENAL SUPPORT



CORTISOL



ERCHONIA

HIGH CORTISOL LEVEL SYMPTOMS	LOW CORTISOL LEVEL SYMPTOMS
High blood pressure	Sugar & salt craving
Fatigue	 Decreased sex drive
Erectile dysfunction	Anxiety
 Reduced sex drive 	Irritability
Weakened immune response	Bone & muscle loss
Weight Gain	Weight gain
Hyperglycemia	Depressed Mood
Insomnia	Insomnia
 Poor concentration/memory 	Fatigue
Source Melatonin CR	

TYPICAL ADRENAL FATIGUE PROGRES

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ePV:10101-01|101011|00100|

90 Tablets Dietary Supplement

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15

30

ADRENAL SUPPORT



ADRENAL SUPPORT



ERCHONIA



B ACTIV

OTHER CLINICAL CONSIDERATIONS: VIBE MANAGE CHRONIC STRESSORS SUCH AS CHRONIC PATHOGENS, LIFESTYLE STRESS AND CHANGES SLEEP DISORDERS SUPPORT ENTIRE HP AXIS? PREGENOLONE STEAL/CHOLESTEROL/SEX HORMONES Duplication, citation or distributio®26f this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA LLLT: FREQUENCIES 10-21-33-59 60 SECONDS EACH ADRENAL GLAND (LOWER RIB CAGE AREA) AFTER UPREGULATING T/L SPINE AREA FOR 20 SECONDS + 2 MIN VAGAL STIM

DON'T FORGET BRAIN 1-10-1-10

VIBRANT LAB FOCUS: SALIVA ASI AND DRIED URINE TEST



DEPRESSION





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DEPRESSION AND BRAIN INFLAMMATION



<u>Front Neurosci.</u> 2019; 13: 213. Published online 2019 Mar 15. doi: <u>10.3389/fnins.2019.00213</u>

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... indry. interpresent study, we characterize intergent activation in oral storegoins when affective brain dysfunctions such as depressive-like behaviors typically develop. Mice manifested <u>chronic mechanical hypersensitivity</u> <u>immediately after CCI and developed depressive-like behaviors 8 weeks post-injury.</u> <u>Concurrently, significant increases of soma size and microglial cell number were</u> <u>observed in the medial prefrontal cortex (mPFC), hippocampus, and amygdala 8 weeks</u> <u>post-injury.</u> Transcripts of CD11b, and TNF-a, 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.**

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DEPRESSION AND THE 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** ["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 damphetamine, 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 ["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. G925 David J. Nutt, Philip J. Cowen, Gitte Knudsen and Eugenii A. Rabiner, 28 October 2022, *Biological Psychiatry*. DOI: 10.1016/i.bioosych.2022.10.012

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 S-HT₃ receptor antagonists (e.g. ondansetron) Fenfluramine Ergotamine/methysergide Hallucinogens



MANUFACTURED IN THE RAPHE NUCLEI LOCATED IN THE BRAINSTEM

ERCHONIA

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

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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

Erancisco Bomo-Nava

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

without permission of Trevor Berry, DC. DACNB is prohibite 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.

<u>Neurophotonics.</u> 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.**

<u>Cassano P</u>1, <u>Petrie SR</u>2, <u>Hamblin MR</u>3, <u>Henderson TA</u>4, <u>Iosifescu DV</u>5. <u>Neuroscience</u>. 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. <u>Barrett DW1</u>, <u>Gonzalez-Lima F</u>.

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



JAffect 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



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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 t⁵³ ugment 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

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*Serotonin Synthesis begins in the gut. Enterochromaffin Cells and certain Microbiome Strains Bifido Infantis, Breve and Longum. Lactobacillus Helveticus and Acidophilus *Low Serotonin - No Joy at All,
Pain,
Sleep, Loss of Enthusiasm and Enjoyment, Sad, Paranoia, Inner Rage

DEPRESSION, HISTAMINES AND INFLAMMATION



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."

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CLINICAL CONSIDERATIONS FOR REDUCING HISTAMINES



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



FOODS THAT PROMOTE

DAO PRODUCTION:

ORGAN MEATS

ESPECIALLY KIDNEY

GREEN PEA SPROUTS

FATS: OLIVE OIL

OMEGA 3'S

Histamine-rich foods are:

There are also a number of foods that trigger histamine release in the body, such as:

- alcohol and other fermented beverages
- fermented foods and dairy products, such as yogurt and sauerkraut
- dried fruits
- eggplant
- <u>spinach</u>
- processed or smoked meats
- <u>shellfish</u>
- aged cheese

Foods that block DAO production include:

- <u>alcohol</u>
- black tea
- mate tea
- green tea
- energy drinks

 nuts, specifically walnuts, cashews and peanuts
 food dyes and other additives
 HISTAMINES IN THE CNS AND INTRACELLULAR ARE BROKEN DOWN BY HISTAMINE N-METHYLTRANSFERASE ENZYME WHICH REQUIRES PROPER METHYLATION OF

COPPER, SELENIUM AND ZINC

B12/FOLATE, VITAMINS A&D, MINERALS MOLYBDENUM,

<u>alcohol</u>

<u>bananas</u>

tomatoes

beans

<u>papaya</u>

chocolate

citrus fruits

wheat germ



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.

LASERS AND ALLERGIES

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

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Psilocybin Increases Brain Connectivity in People With Depression After Use

FeaturedNeurosciencePsychology April 11, 2022

Summary: People with depression who responded to psilocybin therapy showed an increase in brain connectivity for up to three weeks following treatment. The increased brain connectivity was correlated with selfreported improvements in depression symptoms.

"Increased global integration in the brain after psilocybin therapy for depression" by Robin Carhart-Harris et al. Nature Medicine 539

DMT THERAPIES



Study Reveals DMT's Effects on the Human Brain in Unprecedented Detail

The cap measured electrical signals from the volunteers' scalps over 31 different sites. At the same time, the fMRI scan detected the movement of oxygenated blood inside their brains as a proxy for brain activity. This allows the team to create a detailed image of their participants' brain activity. Their trip lasted around 20 minutes in total. Over that period, there were dramatic changes in their brains.

Brain activity is usually segregated into discrete networks. After DMT injection, those networks appeared to break down, meaning that overall brain connectivity increased. This aligns with a <u>previous study</u> of the brain response to psilocybin published by senior author Robin Carhart-Harris. The most significant changes were detected in brain areas linked to high-level cognitive functions like imagination.



That has all changed with the release of the new paper. "This work is exciting as it provides the most advanced human neuroimaging view of the psychedelic state to date," said study first author <u>Dr. Chris Timmermann</u>, from the Centre for Psychedelic Research at Imperial College London.



NUTRITION CONSIDERATIONS: ORTHOMOLECULAR



MUSHROOM PRODUCTS STAMETS LINE "HOST DEFENSE" IS MYCELIUM BASED MYCELIUM BASED IS GROWN WITH BROWN RICE SO EVERY CAP OF THEIRS HAS OVER 50% BROWN RICE MOST IMPORTANT ACTIVE INGREDIENT OF MUSHROOMS IS BETA GLUCANS HOST DEFENSE HAS LESS THAN 20% BETA GLUCANS

MOST COMPANIES DON'T PASS SOURCE INSPECTION OF QUALITY AND DOSE OF BETA GLUCANS

ORTHOMOLECULAR IS A BLEND OF 6 MUSHROOMS AND PASS 3RD PARTY TESTING OF BETA GLUCANS/PURITY

FOR BRAIN BASED CONDITIONS, CORDYCEPS AND LION'S MANE ARE CONSIDERED THE BEST STRAIN OF MUSHROOM

CORDYCEPS ALSO KNOW FOR EXERCISE PERFORMANCE SUCH AS IMPROVED VO2 MAX AND IS A POWERFUL GLUTATHIONE RECYCLER

WATCH TH1 DOMINANT CONDITIONS LIKE HASHIMOTO'S AND MUSHROOMS SINCE THEY ARE A POWERFUL TH1 STIMULATOR

ONE OF BETA GLUCAN'S PRIMARY FUNCTIONS IS TO TAG PATHOGENS AND BAD CELLS FOR COMPLIMENT SYSTEMS TO RECOGNIZE, ATTACK AND KILL THEM

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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)

ANXIETY AND ADHD





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Without permission of Trever Berry, DC, DAGNB is prohibited. ANXIETY TREATMENT IDEAS



behav Brain Funct . 2009 Dec 8;5:46. doi: 10.1186/1744-9081-5-46.

Psychological benefits 2 and 4 weeks after a single treatment with near infrared light to the forehead: a pilot study of 10 patients with major depression and anxiety

Fredric Schiffer 1, Andrea L Johnston, Caitlin Ravichandran, Ann Polcari, Martin H Teicher, Robert H Webb, Michael R Hamblin

We gave 10 patients, (5 M/5 F) with major depression, including 9 with anxiety, 7 with a past history of substance abuse (6 with an opiate abuse and 1 with an alcohol abuse history), and 3 with post traumatic stress disorder, a baseline standard diagnostic interview, a Hamilton Depression Rating Scale (HAM-D), a Hamilton Anxiety Rating Scale (HAM-A), and a Positive and Negative Affect Scale (PANAS). We then gave four 4-minute treatments in a random order: NIR to left forehead at F3, to right forehead at F4, and placebo treatments (light off) at the same sites.

At the end of the study (2 & 4 week follow-up), six of the 10 patients experienced a remission of their depression, and seven of the 10 patients experienced a remission of their anxiety. There were no observable side effects

ANXIETY TREATMENT IDEAS



Anxiousness is associated with decreased alpha waves, increased beta waves, and can be affected by low delta and

theta waves. Anxiousness and feelings of panic can be caused by more than fear and insecurities. They can be chemically driven states from an imbalanced and poorly regulated brain. It's a delicate balance where every wave plays their part.



ERCHONIA HAND HELD LASER "ANXIETY PROTOCOL"

1–10–1–10 PREFRONTAL CORTEX MINIMUM 5 MINUTES (UP TO 6 VIOLET/10 RED)

PLUS VAGAL NERVE STIMULATION PROTOCOL

2 MINUTES SCM/2 MINUTES OVER THE GUT CONSIDER BIAS TO RIGHT PREFRONTAL CORTEX

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 Larson, Jennifer "What Are Alpha Brain Waves and Without ipermission? Of Flore Yor Berry in DCcceDACNB: is prohibited w.healthline.com/health/alpha-brain-waves>

GABA, GLUTAMATE AND ACETYLCHOLINE



STRESS WILL MOBILIZE FATTY ACID AMIDE HYDROLASE (FAAH) WHICH IMPEDES THE CONVERSION OF GLUTAMATE TO GABA...CBD (ENDOCANNABINOIDS) HELP REVERSE THIS!!!



548

The REAL Krebs cycle:



Learn the Krebs cycle

Forget the Krebs cycle



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UNDER THE ASSUMPTION THAT LIFESTYLE MODIFICATIONS AND DIET HAVE BEEN ADDRESSED (GOOD FATS/PROTEINS, EXERCISE, ELIMINATE STRESSORS) CONSIDER THE FOLLOWING:

- -OMEGA 6:3 RATIO TARGET 4:1 OR ABOVE 8%->10+% ON MICRONUTRIENT PANEL (VIBRANT)
- -CANNABINOIDS (IE. FORTIFY ELITE) TARGET 25 MG UP TO BID
- -MINIMUM 3 GRAMS OF MAXSIMIL TECHNOLOGY VS ETHYL ESTER FISH OIL PER DAY. CONSIDER 1 OMEGAMONOPURE 1300 AM AND ONE HEMP MONOPURE PM
- -INTRACELLULAR MAGNESIUM SUPPORT (ALSO MEASURED ON VIBRANTS MICRONUTRIENT PANEL) WITH MAGNESIUM L-THREONATE (TRADEMARK NAME MAGTEIN). 1 GRAM OF XYMOGEN OPTIMAG NEURO DAILY (MIN. 500 MG BID) -XYMOGEN'S RELAXMAX FOR ACUTE EPISODES (DOSE CAN VARY SINCE IT'S IN POWDER FORM. CAN BE USED WITH KIDS
- -ADDRESS ANY UNDERLYING INFLAMMATION SOURCES (SAD/DIET, PATHOGENS, METALS OR OTHER ENVIRONMENTAL FACTORS)
- -STABILIZE ADRENAL CURVES (VIBRANTS 24 HOUR SALIVA TEST) WITH ADRENALIV AM, CORTISOLV OR ASHWAGANDHA 35 PM OR DURING ANY CORTISOL SPIKES. IF PATIENT CAN'T TOLERATE GLANDULARS USE ADRENAL ESSENCE
- -XYMOGEN'S NEW PROBIOMAX SERENITY
- -THIS PROBIOTIC LINE LPC37 IS CONSIDERED A PSYCHOBIOTIC AND HAS BEEN DEMONSTRATED TO BE EFFECTIVE FOR SOCIAL ISOLATION, ANXIETY AND PERCEPTION OF STRESS AND STESSORS!
- -IT CONTAINS 17.5 BILLION CFU'S WHERE AS MOST COMPANIES USE 1-2 BILLION
- -XYMOGENS TOP SELLING PRODUCTS ARE THEIR PROBIOTICS. THEY HAVE 2 YEAR STRESS TESTS, ACID/BILE/PEPSIN/ PANCREATIN TOLERANCE AND TOP ADHERENCE
- -THE SERENITY PRODUCT HAS A 3 WEEK ADHERENCE WHERE AS MANY IF NOT MOST STRAINS HAVE A FEW DAYS
- THEREFORE THIS PRODUCT COULD BE TAKEN INTERMITTENTLY ONCE DESIRED CLINICAL RESULTS ACHIEVED
- -CONSIDER RUNNING STRESS INDEX SCALES PRE TREATMENT IMPLEMENTATION AND RUN PROGRAMS FOR MINIMUM 8
- WEEKS



ATTENTION/ADHD



The effects of TeaCrine[®] and caffeine on endurance and cognitive performance during a simulated match in high-level soccer players

 Marissa L. Bello, Alan J. Walker, Bridget A. McFadden, David J. Sanders and Shawn M. Arent<u>Email author</u> Journal of the International Society of Sports Nutrition201916:20

Background

Theacrine (1,3,7,9-tetramethyluric-acid) is a pure alkaloid with a similar structure to caffeine and acts comparably as an adenosine receptor antagonist. *Early studies have shown non-habituating effects*,

including increases in energy and focus in response to Teacrine[®], the compound containing pure theacrine. The purpose of this study was to determine and

compare the effects of Teacrine® and caffeine on cognitive performance and time-to-exhaustion during a simulated soccer game in high-level male and female athletes.

Conclusions

The 27–38% improvements in TTE (TIME TO EXHAUSTION) reflect increased performance capacity that may have important implications for overtime scenarios. These findings suggest TeaCrine® favorably impacts endurance and the combination with caffeine provides greater benefits on cognitive function than either supplement independently.

CLINICAL PEARL DOSE COGNIQUIL PRIOR TO A BRAIN BASED REHAB VISIT AT YOUR OFFICE, IMPORTANT TESTS/ STUDYING OR SPORTING EVENTS!



* COGNIQUIL CONTAINS B12, MAGTEIN (L-THREONATE) AND TEACRINE

ERCHONIA LASER PREFRONATAL/NASAL Duplication, citation or distribution of this material in whate targin part MASTER BRAIN PROTOCOL period or Berry, DC, DACNB is promotived. **ADHD**



An analysis of data from five large-scale studies found somewhat lower white matter fractional anisotropy in certain regions of the brain in persons diagnosed with attention deficit hyperactivity disorder (ADHD). Fractional anisotropy is generally considered an indicator of connectivity in the brain. Similar microstructural anomalies were not present for anxiety, mood, or externalizing problems. The study was published in *Biological Psychiatry*.

analysis showed that both the level of ADHD traits and ADHD diagnosis were associated with "altered microstructure of the inferior longitudinal fasciculi (ILF) and the left uncinate fasciculus". The overall size of these differences was small. "White matter tract

microstructural anomalies were not as prominently associated with problems related to mood, anxiety or other externalizing problems," the study authors conclude.

* THE ILF IS MAINLY FOR SEMANTIC PROCESSING CONNECTING THE OCCIPITAL AND TEMPORAL LOBES. EX. VISUAL PROCESSING AND LANGUAGE COMPREHENSION. CONSIDER 1-10-1-10 TO PREFRONTAL CORTEX (AND CEREBELLUM) WHILE PRACTICING HAND WRITING. THE LEFT UNCINATE IS A FRONTAL - ANTERIOR TEMPORAL CONNECTION. A MAJOR FUNCTION IS RECOGNITION OF FAMILIAR/FAMOUS FACES. FLASH IMAGES OF KNOWN PEOPLE AND HAVE THE PATIENT CALL OUT THE NAME OF THE IMAGE WHILE LASERING PFC

A mega-analytic study of white matter microstructural differences across five cohorts of youth with attention deficit hyperactivity disorder <u>Gustavo Sudre</u>

Published:September 25, 2022DOI:https://doi.org/10.1016/j.biopsych.2022.09.021

ADHD ADVANCED FN THERAPIES



-OVERLAP SACCADES (VS GAP). FOCUS BUILDER APP

-GO NO GO EXERCISES

-ANTI-SACCADES

-ISOMETRICS

-LEFT SIDE INTERACTIVE METRONOME

-DEEP BREATHING 4:4:8

-MIRROR CURSIVE HANDWRITING

-FACIAL RECOGNITION

-1111 LASER SETTING or 1-10-1-10 PFC (5+ MINUTES), CEREBELLUM (2MIN) + VAGAL STIM PROTOCOL (SCM/GUT 2 EACH) without permission of Trevor Berry, DC, DACNB is prohibited.



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The Food and Drug Administration (FDA) recently greenlit EndeavourRx to treat symptoms of ADHD, via prescription, specifically in kids 8- to 12-years-old.

EndeavorRx is an "immersive video game experience" that's quite similar to many games that children already play for, your know, fun. On an iPhone, iPad, or Android device, the user plays the game while receiving a variety of sensory and attentional cues via movement of their character through a multi-level sensory experience, Dr. Kahn says. The game developer says that the system measures the areas that the child struggles with while they're playing. The software then tailors future gaming sessions to address those specific areas of attention or focus.

Unlike recreational games, the therapeutic gameplay in EndeavorRx is only to be played on a prescriptive schedule (25 minutes, five times a week). The system will not allow access to more time on the game.

553



NEURODEVELOPMENTAL



Conner Bor Group expert • 37m • 😁

Here's a fun one for you guys. I have been working with this kid diagnosed with seizures. 6 months after he started coming in, he had a clean EEG and parents decided to take him off of all his seizure meds. They ended up purchasing an XLR8 to take the place of the meds and here is his second EEG 6 months after the first completely clean! Lasers are cool.

Dr Bor!! I just wanted to say we got done with set s 24 hour EEG this morning and the doctor called me a few minutes ago to say his entire EEG was absolutely unremarkable. His EEG was so clean and perfect this time and I wanted to let you know as soon as possible. I really don't have the words to thank you enough for everything you have done. I truly truly appreciate you and your work. I have no idea where we would be had we not started seeing you last

year.

Thank you 🤎



CLINICAL PEARL: ALWAYS INTEGRATE PRIMITIVE REFLEXES ESPECIALLY IN NEURODEVELOPMENTAL DISORDERS! (SEE BACK OF NOTES FOR PRIMITIVE REFLEXES. PART OF AFN TRAINING)

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AUTISM





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MITOCHONDRIAL DAMAGE, TYLENOL AND AUTISM

They collected umbilical cord blood from 996

acetaminophen and two of its byproducts in

diagnosed with ADHD only, 6.6% with ASD

each sample. By the time the children were an

TYLENOL (ACETAMINOPHEN)

LAWSUIT INJURIES:

MANUFACTURERS: Johnson & Johnson

Disorder (ADHD)

Autism Spectrum Disorder (ASD)

LAWSUIT STATUS: Accepting New Clients

Attention-Deficit Hyperactivity

AUTISM ADHD LAWSUIT

births and measured the amount of

average of 8.9 years, 25.8% had been

only and 4.2% with ADHD and ASD

PEOPLE EFFECTED:

ASD (Autism Spectrum

(Acetaminophen) during pregnancy AND/OR pa of children who meet that

criteria

Disorder) or ADHD (Attentio

Deficit Hyperactivity Disorder)

ther took Tylenol

duals diagnosed with

YLENO

TYLENOL

FDA Warnin Not Yet Issu



COVID-19 COVID.gov | NIH Research information | Español | NIH staff guidance (NIH On

NEWS RELEASES

Media Advisory Wednesday, October 30, 2019

NIH-funded study suggests acetaminophen exposure in pregnancy linked to higher risk of ADHD, autism

◎ ≥ f y +

What

posure to acetaminophen in the womb may increase a child's risk for attention deficit/hyperactivity disorder and autism spectrum sorder, suggests a study funded by the National Institutes of Health and the Agency for Health Care Research and Quality. The study as conducted by Xatonic Wang, MLD, of the Johns Hopkins University Bloomberg School of Public Health, Baltmore, and colleagues. appears in JAMA Psychiatry.

ention deficit/hyperactivity disorder (ADHD) is marked by a pattern of hyperactivity and impulsive behavior. Autism spectr order (ASD) is a complex developmental disorder that affects how a person behaves, interacts with others and learns.

Breaschers analyzed data from the Botton Birth Calora, a long-term study of factors influencing pregnancy and child development. They collected unbilical cord blood from 996 births and messured the amount of acetaninophen and two of its byproducts in each sample. By the time is children were an average of SJ years, 25.8% had been diagnosed with XHD oxy6, 65% with X60 mly and 42% with XHD and X50. The researchers classified the amount of acetaninophen and its byproducts in the sample into third, from were to highest. Compared to the lowest third, the middle third of exposure was associated with about 22.50 times the risk for XHD. The highest third of exposure was associated with 2.85 times the risk. Smilarly, ASD risk-was higher for those in the middle third (2.14 times) and highest that (3.52 times).

The authors conclude that their results support earlier studies linking acetaminophen exposure in the womb with ADHD and ASD and anderscore the need for additional research. The U.S. Food and Drug Administration urges careful consideration# before using any pain-reliening medication during pregramcy.

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.

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 of ADH 3P43trifs your of the Tylenol ADH developing in babies of pregnancy and your child developed that permission to the Tylenol during pregnancy and your child developed that the to the to the tylenol during pregnancy and your child developed that the total during pregnancy and your child developed the total during pregnancy and your child during the total during pregnancy and your child during pregnancy and your child during the total during total during the total during total duri

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Su R



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-

ace 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 FLOS ONE

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.



HHS Public Access

Author manuscript JNeurochem. Author manuscript; available in PMC 2016 April 21.

hed in final edited form as: *urochem.* 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

Mai

bstract Mitchondria play important roles in generation of free radicals, ATP formation, and in apoptosis. We studied the levels of mitchondrial electron transport chain (ETC) complexes, that is, complexes 1, III, IIV, and V, in brain tissues amples from the cerebellum and the frontal, parietal, complexes 1, III, IIV, and V, in brain tissues amples from the cerebellum and the frontal, parietal, superstanding the second seco

Keywords

autism; electron transport chain complexes; energy; mitochondria; oxidative stress

Address correspondence and reprint requests to Abha Chauhan, NYS Institute for Basic Research in Develop Forest Hill Road, Staten Island, NY 10314, USA, abha.chauhan@opwdd.ny.gov. ental Disabilities 105





Biological Psychology Volume 175, November 2022, 108448

Right anterior theta connectivity predicts autistic social traits in typically developing children

Author links open overlay pan

Aron T.HillabJodieVan Der ElstaFelicity J.BigelowaJarrad A.G.LumaPeter G.Enticotta 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.

AUTISM



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 <u>elevated left-over-right</u> 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: <u>10.1371/journal.pone.0175870</u>

PMCID: PMC5414938 PMID: <u>28467487</u>

Is functional brain connectivity atypical in autism? A systematic review of EEG and MEG studies

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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)



pen 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^{uplication, citation or distribution of the material in whole or in part}



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

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...



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.

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, Ruteliantion reitation in here without permission of Trevor Berry, DC, DACNB is prohibited.

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.

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. In 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 Duplication, citation or distribution of this material in whole or in part changes were made in subjects inform partness or intervorment protocol, back a symptoms.

THE ADVANCED LASER NEUROLOGY **PROTOCOL FOR AUTISM**





TREATMENT TIME: 30 **SECONDS** PER YEAR OF AGE



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!** ion or distributio for this material in v



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

569

network predicted more self-reported diffi STACK WITH GO-NO GO EXERCISES: everyday life.

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

Blavlock, 1.* Jiri Patocka, 2 and Otakar Strunecky

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 wopsenimetic vicitors in the inflammation. without permission of Trevor Berry, DC, DACNB is prohibited.

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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: <u>10.1016/j.jtemb.2017.11.012</u>

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.





Child Care Health Dev

AUTISM

. 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 phyperactivity is material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

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AUTISM



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) 573

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

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:

- o gut contractions
- ^o the number of neurons in the small intestine
- ^o 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.



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: <u>32532137</u>

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 (<u>Table S3</u>). 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 [<u>186,187</u>]. 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

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

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

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)

IMMUNOLOGY AND PATHOGENS





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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 Duplication, citation or dis**phenicity 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



ERCHONIA

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



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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









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IMMUNE FUNCTION AND PATHOGENS: COMMON SETTINGS

 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

PATHOGENS AND ALZHEIMER'S



ERCHONIA

rent

ocks

CONSIDER TRANSCRANIAL LASER APPLICATIONS USING THE MASTER BRAIN AND IMMUNE PROTOCOLS ON ALL OF YOUR PATIENTS AND FAMILY MEMBERS AT REGULAR INTERVALS TO PROTECT THE BRAIN AND BARRIER SYSTEMS, SUPPORT PROPER IMMUNE FUNCTION AND REGULATION, DECREASE INFLAMMATION AND SO ON.

EVEN IF THEY ARE COMING IN FOR KNEE PAIN!

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IMMUNE FUNCTION AND PATHOGENS

orpus 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///HSM-Biard herpes.azosterDNave:only/virostatic effect

without permission of Trevor Berry, DC. DACNB is prohibited. **MULTIPLE SCLEROSIS**





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MULTIPLE SCLEROSIS



Multiple sclerosis linked to infection in adolescence – new study

September 7, 2021 4.57am EDT

We found that most infections before age 11 were not associated with a later MS diagnosis. In contrast, infections diagnosed in a hospital (indicating they are relatively severe) between ages 11 and 19 were consistently associated with a raised risk of developing MS.

There was a minimum of five years between infection and MS diagnosis – and usually longer – indicating that the disease progresses slowly until there is sufficient damage to the brain for symptoms of multiple sclerosis to develop.

Not all types of infection were associated with subsequent MS, but a striking finding is that infections of the central nervous system (the brain and spinal cord) increased MS risk most notably.

Respiratory infections in adolescence were also associated with MS, increasing the risk by 51%.

Hospital-diagnosed infections before age 20 and risk of a subsequent multiple sclerosis diagnosis Yin Xu, Kelsi A Smith, Ayako Hiyoshi, Fredrik Piehl, Tomas Olsson, Scott Montgomery Brain, Volume 144, Issue 8, August 2021 Pages 2390 at ftfbution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited. without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA

NEUROLOG

Epstein-Barr Virus Found to Trigger Multiple Sclerosis

SUMMARY POINTS:

SCIENCE: JAN 13 2022

- 2.8 MILLION PEOPLE WORLDWIDE SUFFER FROM M.S.
- 95% OF AMERICANS HAVE ANTIBODIES TO EPSTEIN-BARR VIRUS (EBV)
- THE RESEARCHERS TESTED 10 MILLION MILITARY PERSONAL NEGATIVE FOR EBV WHEN JOINING THE MILITARY. OF THOSE, THEY WERE ABLE TO ANALYZE APPROPRIATE SAMPLES OF 801 PEOPLE THAT DEVELOPED M.S. AND 1566 CONTROLS. ONLY 1 OF THE 801 THAT DEVELOPED M.S. DID NOT CONTRACT EBV!!!
- THIS IS A 32 FOLD CORRELATION TO EBV AND M.S.!

- FOR CONTEXT, SMOKING STUDIES HAVE A CORRELATION OF

Epidemiologist Alberto Ascherio, senior author of the new study, says, "The bottom line is almost: if you're not infected with EBV, you don't get MS. It's rare to get such black[®]and-white results."

Positive		Moderate					2		
(IgG + IgA)	lgM	(IgG + IgA)	lgM		Neg	auve		590	
	Blood Brain Barrier Disruption Anti-s100b Peripheral Neuropathy	Demyelination Antigens Anti-Tubulin Blood Brain Barrier Disruption Anti-S100b Anti-Glucose regulated protein 78 Brain Autoimmunity Anti-Amyloid beta (25-35)	Demyelination Antigens Anti-Tubulin Blood Brain Barrier Disruption	Demyelination Antigens Anti-Myelin proteolipid protein	Anti-Myelin basic protein	Demyelinat Anti-Myelin oligodendrocyte glycoprotein Blood Brain Ba	ion Antigens Anti-Neurofascin rrier Disruption	Anti-MAG	NEURAL ZOOMER
	Anti-GM1 Neuromuscular disorders Anti-Voltage gated potassium		Neuromuscular disorders Anti- Acetylcholine receptors	Anti-Glial fibrillary acidic Anti-Microglia protein Optical and Autonomic nervous system disorder				PLUS	
	channels		Brain Autoimmunity Anti-RAGE	Anti-Neuron specific enolase	Anti-Aquaporin4 Peripheral	Anti-CV2			
THE CON	NZ+ CAN E	BE USED A	NS A R A	Anti-GM2	Anti-Hu Neuromuscu	Anti-Ri Ilar disorders	Anti- Amphiphysin		
PREDICTI BEST	VE MODEL USED AS	. CLINICA AN UPSTR	LLY, IT IS EAM	Anti-Muscle specific kinase	Anti-Voltage gated calcium channels Brain Aut	Anti-Titin oimmunity			
INFLAMMATION OF END STAGE INFLAMMATION AND AUTOIMMUNE TRIGGERS. IF I SEE POSITIVE FINDINGS I START TO LOOK FOR IMMUNE TRIGGERS. THE MOST				Anti-HSV1 Anti-Amyloid beta (1-42) Anti- Hydroxytryptami ne Anti-NMDA receptor	Anti-Cerebellum Anti-Tau Anti-Alpha- synuclein Brain Infl	Anti-Purkinje cell Anti-Glutamate Anti-α1 and β2 adrenergic receptors	Anti-Yo Anti-Dopamine Anti-Endothelin A receptor		
Common And Pat Trigger Ane Disruptio Brain F Brain H	N BEING FO HOGENS O RING MOLE O OFTEN PE ON OF THE BARRIERS! AS THE LU	DOD SENS DR OTHER ECULAR M RECEDED E GUT AND ! NORMAL IXURY OF \$	ITIVITIES TOXINS IIMICRY BY BLOOD- LY THE SITTING	Anti-AMPA receptor Anti-Dipeptidyl aminopeptidase like protein ó Anti-Leucine- rich glioma- inactivated protein 1 (Anti- LGI1)	Anti-Dopamine receptor 1 Anti-Glycine receptor Anti-Ma	Anti-Dopamine receptor 2 Anti-Neurexin 3	Anti-GABA receptors Anti-Contactin- Associated Protein-like 2 Antibodies		
BEHIND OF IMM	A PROTEC	TIVE VAUL CAL REAC	To FREE TWITY ssion	Anti-HSV2 or distribution of of Trevor Berr	Anti-EBV f this matterial in y, DC, DACNE	Anti-CMV n whole or in pa s is prohibited.	Anti-HHV 6 art		

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Researchers Find A Web Of Factors Behind Multiple Sclerosis

January 21, 20198:35 AM ET BRET STETKA

"We now know that MS is not infectious in the true sense of the word. It is not contagious in the way, say, the flu is. But infection does likely play a role in MS.

As may be the case in Alzheimer's disease, it's looking more and more as though MS strikes when infectious, genetic and immune factors gang up to eventually impair the function of neurons in the brain and spinal cord."

it is for the most part accepted that microbes play some role in MS given that dozens of microbes, including the Epstein-Barr virus, have been tied to MS. COMMON => EBV AND HHV

The immune siege appears to be a result of something called "molecular mimicry." Normally the body's immune system attacks foreign invaders like viruses and bacteria. If a molecule that's part of the body happens to closely resemble a portion of an intruding microbe, then both molecules can be targeted.

GDP-I-fucose synthase is a CD4+ T cell-speci c autoantigen in DRB3*02:02 patients with multiple

sclerosis Planas, Raquel; Santos, Radleigh; Tomas-Ojer, Paula; Cruciani, Carolina; Lutterotti, Andreas;

RESULTS:

Before treatment, EBV and HSV1 were observed in 29.9% and in 3.8% of periodontal patients respectively, while coinfection with both viruses was detected in 1.7% of cases. Periodontal Nd:YAG laser treatment ("Periodontal Biological Laser-Assisted

Therapy", PERIOBLAST) produced statistical significant benefits, especially in EBV periodontal infection: 78.2% of EBV

positive patients became EBV-negative following treatment.



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.

Author in Depart Abstract Recurr introdu therap influen patient randon 2 wk. y

Schind

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

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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 unspirity since treatment, it might be considered as an alternative to established therapeutiergemeens in This instruction-DACNB is prohibited.

IMMUNE FUNCTION AND PATHOGENS



CONSIDER FASTING AND FOOD ELIMINATION. TARGET DAIRY AND GLUTEN FIRST. LEAKY GUT REPAIR PROTOCOL. VITAMIN D 70-100. WORK UP TO A 3 DAY WATER FAST...

Cell Rep. 2016 Jun 7; 15(10): 2136–2146. Published online 2016 May 26. doi: 10.1016/j.celrep.2016.05.009

Diet mimicking fasting promotes regeneration and reduces autoimmunity and multiple sclerosis symptoms

In Young Choi,1,† Laura Piccio,2,† Patra Childress,3 Bryan Bollman,2 Arko Ghosh,4 Sebastian Brandhorst,1 Jorge Suarez,1 Andreas Michalsen,5 Anne H. Cross,2 Todd E. Morgan,1 Min Wei,1 Friedemann Paul,6,7 Markus Bock,6,7,* and Valter D. Longo1,4,8,9,* Summary

Dietary interventions have not been effective in the treatment of multiple sclerosis (MS). Here we show that periodic 3 day cycles of a fasting mimicking diet (FMD) are effective in ameliorating demyelination and symptoms in a murine experimental autoimmune encephalomyelitis (EAE) model. The FMD reduced clinical severity in all mice, and completely reversed symptoms

in 20% of the animals. These improvements were associated with *increased corticosterone levels and Treg cell number*, reduced levels of pro-inflammatory cytokines, TH1 and TH17 cells, and antigen presenting cells (APCs).

Moreover, the FMD promoted oligodendrocyte precursor cell regeneration and remyelination in axons in response to both EAE and cuprizone MS models, supporting its effects on both suppression of autoimmunity and remyelination. We also report preliminary data suggesting that a FMD or a chronic ketogenic diet are safe, feasible and potentially effective in the treatment of relapsing remitting multiple sclerosis (RRMS) patients (NCT01538355).

Autoimmunity. 2016;49(2):132-42. doi: 10.3109/08916934.2015.1124425. Epub 2015 Dec 24.

Low-level laser therapy ameliorates disease progression in a mouse model of multiple sclerosis. t V1, Fidelis GS2, Barbosa RI3, Silveira PC2, de Pinho RA2, Dutra RC1,2,3. Goncalves ED1.2, Souza PS2, Lieberk Author information Abstract

Furthermore, these beneficial effects of LLLT seem to be associated with the down-regulation of NO levels in the CNS, although the treatment with LLLT failed to inhibit lipid peroxidation and restore antioxidant defense during EAE. Finally, histological analysis showed that LLLT blocked neuroinflammation through a reduction

of inflammatory cells in the CNS, especially lymphocytes, as well as preventing demyelination in the spinal cord after EAE induction. Together, our

results suggest the use of LLLT as a therapeutic application during autoimmune neuroinflammatory responses, such as MS.

MULTIPLE SCLEROSIS



J Photochem Photobiol B. 2018 Oct 3;189:55-65. doi: 10.1016/j.jphotobiol.2018.09.024. [Epub ahead of print] Low-level laser therapy modulates demyelination in mice.

Duarte KCN1, Soares TT1, Magri AMP1, Garcia LA1, Le Sueur-Maluf L1, Renno ACM2, Monteiro de Castro G3.

Author information

Abstract

There are no effective therapies for remyelination. Low-level laser therapy (LLLT) has been found advantageous in neurogenesis promotion, cell death prevention, and modulation of inflammation in central and peripheral nervous system models. The purpose of this study was to analyse LLLT effects on cuprizone-induced demyelination. Mice were randomly distributed into three groups: Control Laser (CTL), Cuprizone (CPZ), and Cuprizone Laser (CPZL). Mice from CPZ and CPZL groups were exposed to a 0.2% cuprizone oral diet for four complete weeks. Six sessions of transcranial laser irradiation were applied on three consecutive days, during the third and fourth weeks, with parameters of 36 J/cm2, 50 mW, 0.028 cm2 spot area, continuous wave, 1 J, 20 s, 1.78 W/cm2 in a single point equidistant between the eyes and ears of CTL and CPZL mice. Motor coordination was assessed by the rotarod test. Twenty-four hours after the last laser session, all animals were euthanized, and brains were extracted. Serum was obtained for lactate dehydrogenase toxicity testing. Histomorphological analyses consisted of Luxol Fast Blue staining and immunohistochemistry. The results showed that laser-treated animals presented motor performance improvement, attenuation of demyelination, increased number of oligodendrocyte precursor cells, modulated microglial and astrocytes activation, and a milder toxicity by cuprizone. Although further studies are required, it is suggested that LLLT represents a feasible therapy for demyelinating diseases.

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Abstract

Introduction: Multiple sclerosis (MS) is an autoimmune disease. Inflammatory cells, cytokines and chemokines play a major role in the pathogenesis of the disease. Low-level laser therapy (LLLT) as a photobiostimulation approach could affect a wide range of cellular responses. LLLT inhibits the inflammatory signaling pathway, improves cell viability, inhibits apoptosis, modulates immune responses and induces the production of growth factors. **Methods:** In this review, we discuss the effect of LLLT on cellular responses and its application in the treatment of MS. Such keywords as "low-level laser therapy", "photobiomodulation" and "multiple sclerosis" were used to find studies related to laser

therapy in MS in Google scholar, PubMed and Medline databases. **Results: LLLT reduced the** inflammatory immune cells and mediators. It also enhanced the regeneration of neurons. Conclusion: Investigations showed that besides current treatment strategies, LLLT could be a promising therapeutic approach for the treatment of MS.

J Lasers Med Sci

2021 Dec 28;12:e88. doi: 10.34172/jlms.2021.88. eCollection 2021.

The Role of Low-Level Laser Therapy in the Treatment of Multiple Sclerosis: A Review Study

Nikoo Hossein-Khannazer 1, Mandana Kazem Arki 2, Aliasghar Keramatinia 3 4, Mostafa Rezaei-Tavirani 5 Affiliations expand

PMID: 35155173 PMCID: PMC8837843 DOI: 10.34172/jlms.2021.88
Free PMC article

MULTIPLE SCLEROSIS



Researchers at the Netherlands Institute for Neuroscience have discovered that the energy management of inhibitory brain cells is different than that of excitatory cells in our brain. Why is that the case and what is the link with multiple sclerosis?

Myelin does appear to be important in PV cells. Previous studies on tissue from MS patients showed that PV cells die when myelin is lost. Apart from conduction, myelin also plays an important role in nourishing the cell.

In PV cells, mitochondria behave in an opposite way from what was previously known in the literature for other types of cells. But why exactly does this happen in these cells?

Researcher Koen Kole says, "We suspect that it has to do with the fact that PV cells have an incredibly high energy demand due to their high level of activity. In addition, their axons are very thin compared to those of other cell types, which could further increase their energy consumption. **PV cells may therefore be more dependent on external nutrients from the myelin.**"

SUMMARY: PARVALBUMIN (PV) CELLS ARE INHIBITORY IN THE CORTEX AND HAVE HIGH ENERGY DEMANDS THAT ARE MET BY MITOCHONDRIA IN THEIR MYELIN SHEATH. THIS NEW DISCOVERY SHOWS THAT PROTECTING THE MITOCHONDRIA IN THE PV MYELIN IS CRITICAL IN MULTIPLE SCLEROSIS AND DEMYELINATING DISEASES!

Published: 09 December 2022

Parvalbumin basket cell myelination accumulates axonal mitochondria to internodes



without permission of Trevor Berry DC DACNB is prohibited LOOK AT ANTIMYELIN ANTIBODIES (NEURAL **ZOOMER) AS AN EARLY PREDICTOR OF MS**

> FIBER => SHORT CHAIN FATTY ACIDS => MORE TREG **CELLS WHICH LEADS TO BETTER ORAL AND CENTRAL TOLERANCE. SCFA'S LIKE BUTYRATE AND** PROPRIONATE

KETOGENIC DIETS HAVE SHOWN PROMISE (IMMUNE NEUTRAL, HIGH FAT AND NO INSULIN SURGE)

This diet allows you to eat healthy saturated fats (fish, meats, nuts, seeds, avocado). The Wahls dietary protocol is heavily plant based, and encourages a transition to a Paleo diet (no grains, with a focus on eating heaps of veggies, berries, algae, meats and fish).

The excluded foods include; gluten in all forms, dairy, refined sugars + any foods that you react to (allergies and food intolerances)

MULTIPLE SCLEROSIS



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- High powered magnetic resonance spectroscopic imaging can detect early, subtle metabolic changes associated wit multiple sclerosis (MS).
- Early diagnosis of MS may help neurologists treat the disease more effectively.
- In a new study, researchers found changes in white matter and cortical gray matter that appears "normal" in standard magnetic resonance imaging.
- JOURNAL OF RADIOLOGY

JAN 04, 2022

Original Research Free Access Neuroradiology

Extensive Brain Pathologic Alterations Detected with 7.0-T MR Spectroscopic Imaging Associated with **Disability in Multiple Sclerosis**

Eva Heckova, Assunta Dal-Bianco, Bernhard Strasser, Gilbert J. Hangel, Alexandra Lipka, Stapislav Motvka, Lukas Hingerl, Paulus S. Rommer, ..., See all authors Dublication, Citation of distribution of distribution and the second state of the seco without permission of Trevor Berry, DC, DACNB is prohibited.

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

LLLT AND IMMUNE PROPERTIES



QUESTION: HOW DOES LOW LEVEL LASER KNOW HOW TO TARGET CERTAIN CELLS **OVER OTHERS IN DISEASED STATES?**

599

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 PEARLISS 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 THII "ATTACK", RIGHT BRAIN THINK TH2 AND "RESOLVE" without permission of Trevor Berry, DC, DACNB is prohibited.

SILVER HYDROSOL



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Or go to the link: https://sovereignsilver.com/discount/DRB20





EXERCISE RESOURCE FOR AUTOIMMUNE PATIENTS



GETAUTOIMMUNESTRONG.COM

For people struggling with autoimmune disease, fibromyalgia, and other chronic pain issues, exercise is a double edged sword. Movement is essential, but too much movement can stimulate a flare-up of the worst kind.

Exercise CAN help us manage our symptoms and flare-ups. Chronic pain conditions are exacerbated by inflammation, and, if done properly, exercise can help to reduce inflammation and pain caused by inflammation.

Additionally, the right exercises can teach our bodies adapt to a pain response, which can help us get through the pain and exhaustion of our daily activities. Our bodies need to learn to be comfortable being uncomfortable in a safe, protected way, so that when we are exposed to discomfort in our daily lives, our bodies are prepared to handle it.

This is the philosophy behind Autoimmune Strong- to provide a small degree of strength and challenge with incremental increases over time, so that our bodies can be prepared for the unpredictable nature of life.

EXERCISES, FOOD AND LIFESTYLE PLANS, AND SUPPORT COMMUNITY

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UPDATE ON "MO"





MAUREEN H. SUFFERED A RIGHT MIDDLE **CEREBRAL ARTERY STROKE IN 2015. SHE** HAD MADE EXCELLENT PROGRESS THROUGH HER TEAM OF THERAPISTS. FUNCTIONAL NEUROLOGY, ERCHONIA LASERS AND EVEN PEPTIDE THERAPIES WITH LASER. ALTHOUGH SHE WAS ABLE TO RETURN TO CERTAIN ACTIVITIES OF DAILY LIVING, SHE HAD PLATEAUED WITH HER MOTOR RECOVERY. WE DISCUSSED STEM CELLS AS THE FINAL FRONTIER. SHE IS A VERY EDUCATED PATIENT AND EVEN HAS A FULL WEBSITE AND BLOG DEDICATED TO THOSE INJURED BY CVA'S. CHECK IT OUT AT STROKE RECOVERY SOLUTIONS. SHE SHARED HER JOURNEY THROUGH THE STEM CELL PROCESS AND I THINK IT IS A GREAT LESSON FOR THOSE **CONSIDERING THIS FORM OF REGENERATIVE MEDICINE...**

STROKERECOVERYSOLUTIONS.COM A GREAT RESOURCE FOR STROKE PATIENTS AND FAMILIES

STEM CELL THERAPY FOR MCA STROKE



STEM THERAPY REPORT **Advanced Biomedicine Clinic** Cancun, Mexico November 14-17, 2022

CLINIC SCHEDULE The Process

MONDAY, Nov. 14th Appointment: 10:10 am to 11:30 am

CBC, Medical History, etc. + ...

(1st injection of 2) of Leukocytes (colorless cell that circulates in the blood and body fluids and is involved in counteracting foreign substances and disease; a white (blood) cell. There are several types, all amoeboid cells with a nucleus, including lymphocytes, granulocytes, monocytes, and macrophages.)

Purpose: To stimulate the production of white blood cells to ensure maximum the introduction & efficacy of the new stem cells that will be introduced Wednesday.

TUESDAY, Nov. 15th Appointment: 10:10 am to 10:30 am

2nd Injection of Leukocytes

WEDNESDAY, Nov. 16th Appointment: 7:30 am to 11:00 am IN HOSPITAL

Prep & injection of Twilight Sedation (I tell you, their Twilight is MUCH stronger than America's, thank goodness! WOW!) Retrieval of bone marrow from (my BIG!) right hip! 😂

"Washing" of bone marrow to prepare the stem cells for injection in two ways: intrathecally (via the spine) & intravenously (IV). Back to the hotel, where I slept all afternoon! $z^{z}z^{z}z^{z}z^{z}$

THURSDAY, Nov 17th Appointment: 10:10 am to 11:30 am

2nd IV of stem cells & final written report discussion.

When we entered the clinic today, one of the aword octars (the quist is the open was BEAMING register or in part 1. He was shocked at how well everything were derived by the permission of Trevor Berry, DC, DACNB is prohibited.

STEM CELL THERAPY FOR MCA STROKE



2nd IV of stem cells & final written report discussion.

- When we entered the clinic today, one of the two doctors (the quiet, shy one) was BEAMING, because:
- 1. He was shocked at how well everything went; &
- He could NOT believe the massive increase of Leukocytes- from 9.5 to 38 IN TWO DAYS!!! He said, "We only see such increases in the KIDS we treat!" U They treat tons of kids for autism & various other diseases. (We got the impression his surprise was due to my "advanced age," or something silly like that!)
- 3. As you can imagine, we were thrilled with all the good news!

of STEM CELLS INJECTED: 492 MILLION (They initially thought they'd be able to retrieve 250,000-300,000 million stem cells from my a) 'aged' body; b);the facts that it was a massive stroke vs mild or medium; c) that I was 7 yrs post-stroke!

Little did they know...!) 🗃

- So, what's next?
- The 1st 3 weeks post- therapy are critical due to the process of "homing." That means these babies are assimilating into the bloodstream & brain.

During that **3-week period**, the following requirements are necessary: 1. Add Vitamin E supplement to my vitamin program.

- 2. No alcohol.
- 3. No inflammatory foods/drinks.
- 4. Sleep lots of it! Fatigue & 'flu-like' symptoms are common.
- 5. If aches or pains occur, acetaminophen only, nothing else.
- 6. No sunburn.
- 7. Minimal sugar zero is preferred.
- 8. Lots of water. Already do!
- 9. No gluten.
- 10. Exercise

607

STEM CELL THERAPY FOR MCA STROKE



Is full recovery possible? Of course! Along with rigorous stroke exercises & lots of visualizations WITH INTENSE FEELINGS as though I'm already healed! I'd be grateful for your participation in this endeavor, as always - both during our monthly sessions & beyond. Since y'all are the most powerful people on this planet, I KNOW we'll be successful! "It takes a village!"

The doctors anticipate 3-4 months for full recovery.

Here we go! 😃

GENERAL INFORMATION

A Patented Procedure: This procedure was researched, created and patented by Dr. Rudolph Gonzales. He does not reside/work in Cancun. He's in CA:

Executive Director Stem Cell Therapeutics at Caribou Biosciences, University of California, San Diego, CA

Meanwhile, in America ... (I'll try not to be too sarcastic!)

The "stem cell" procedure "approved by the FDA allows for 60,000 - 80,000 stem cells injected, via IV ONLY. "Intrathecally" is not allowed, but it makes all the difference in the world! The cost in America ranges from \$10,000 -\$20,000. Mine cost \$22,000, and was properly administered. Sadly, patients may not know enough & think 'all stem cell therapies are the same.' Not.

Another money maker for doctors, hospitals & big pharma.

Glad I did my research!

Enough said.

Hope this helps with your understanding. If you have any questions, please ask!

HAPPY THANKSGIVING!

I'm thankful more than ever this year; & next year, I plan to be COOKING the turkey, & all the trimmings! MAUREEN H.

AS SOON AS MAUREEN RETURNED TO PHOENIX WE BEGAN IMMEDIATE ERCHONIA LASER PROTOCOLS TO PROMOTE LOCALIZATION, DIFFERENTIATION AND CONNECTIONS

UPDATE ON MO





FOR THE FIRST TIME SINCE HER STROKE, SHE IS NOW ABLE TO HAVE FULL HIP FLEXION WITHOUT ASSISTANCE. PROXIMAL LARGE MUSCLES FIRST THEN THERAPY WILL WORK HER WAY DOWNOTO FINE MOTOR



DECEMBER 2019⁰¹⁰ "BUDDY" PRIOR TO INITIAL EVALUATION

IDEXX Telemedicine Consultants

Radiology Report (5159980-15/Radiographs Only - Stat) Completed 12/20/19 02:17 PM

Patient Name: Buddy Berry (265A)
Requesting Doctor: Shareen Masaih, DVM Species: Canine Age: SY: 9Mo
Gender: Unincom Breed: Unincom Weight: 7.00 Kg
DPESPUTING VAID Non-monitor Methods

HISTORY: Buddy is suffering from not bearing weight in both hind limb for last two weeks... need assistant to go outside...still controlling defecution/ unnation.apatile slow down this moming.examining both hid limb reveal motor, body all time.

THORAX and ABDOMEN/THORAC

FINDINGS:

The categorization of the second sec

A mild annot of heterogenous soft liseux and gas couchy resembling overni ingetta is seen within the shanktimitie gracity material ingenesita. In all format of horm liseux within the cosh, hord of the small instantial loops, distended byond normal limits, and have normal shape, and pattern. The liver, spleen, left kidney urinary bladder, addominal sorosal data are normal. The registric data and the state of the small relation of the liver and gastrointestinal structures. There lines of enthespotyles paralleling the physical scar on both femoral necks.

CONCLUSIONS: — There is evidence of multilocal intervertebral disc disease, and the superimposition of the mineral opacities with the intravertebral foramen is concerning for extrusion into the carral, dorsaf to 15-14, though this could also be lateral to the signal cost. Mineratization in situ date and correlate will will minigons of signal cord compression, and advanced imagi Unfortunate), pian firms cannot determine any degree of spinal cord or spinal nerve root compression associated with bory changes.

ny changes. The thorax and abdominal cavifies are considered within normal limits. 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!







BUDDY OCTOBER 10, 2020





HAIR PROTOCOL





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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

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HAIR PROTOCOL



Cutis. 2019 Jul;104(1):17-24.

Nonsurgical hair restoration treatment.

Nazarian RS1, Farberg AS1, Hashim PW1, Goldenb 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.

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 Be

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: <u>10.31491/apt.2020.03.014</u>

The potential of GHK as an anti-aging peptide

Yan Dou,a Amanda Lee,a Lida Zhu,a John Morton,a and Warren Ladigesa,*



NUTRAFOL

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)

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HAIR PROTOCOL



HAIR RESTORATION PROGRAM **EXAMPLE:**

12 MONTH PROGRAM \$9500 **INCLUDES 12 MONTHLY SCALP** PRP WITH MICRONEEDLE

ALL PEPTIDES

2X/MONTH SCALP LOW LEVEL LASER TREATMENTS

NUTRITION SUPPLEMENTS

HAIR PROTOCOL



ERCHONIA

STEP

STEP 2. Take close photographs of all treatment area

STEP 3.

Patient blood draw for PRP preparation

STEP 4

Clean the scalp / hair with sterile or non-sterile cleanser ie; soap and water, hibicleanse

STEP 5.

Numb area to be treated with BLT Cream (optional ring block, in addition to BLT)

STEP 6

Teach patient how to perform Sub Q injection of 25ml Thymosin Beta-4 Patient will continue these injections at home per at-home protocol instruction

STEP 7.

Be sure scalp is sufficiently anesthetized and wipe off any excess BLT cream

STEP 8

Prepare PRP for injection - Saving 1-3mL PRP for micro-needling and all remaining PPP 1. Buffer PRP with bicarb

- 0.3ml bicarb for 7ml PRP 0.5ml bicarb for 10ml PRP 2. Activate PRP with Calcium Chloride
- 0.3ml Calcium for 7ml PRP 0.5ml Calcium Chloride for 10ml PRP STEP 9.

Inject activated and buffered PRP into scalp treatment area.

STEP 10.

Microneedle entire treatment area with saved 1-3mL of PRP until pin point bleeding occurs. If necessary, use remaining PPP for additional gliding lubricant and ease.

STEP 11

IMMEDIATELY spray PTD-DBM and Valproic Acid over entire treatment area following micro-needling

STEP 12.

Schedule 6 and 12-week follow-up appointments and give patient treatment calendar.

Safety And Effective nets to Peptidie to His mark of Piscoco Pischeria Four balanair Regeneration without permissione of the second second



SAMPLE DERMAROLLER



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HAIR PROTOCOL



Procedue Day														
	31		1		2		3		4		5		6	
PROCEDURE TB4 .25ml DTP-DBM + VPA Topicals		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		
	7		8		9		10		11		12		13	
TE PTD-DBM + V Topicals	TB4 .25ml PTD-DBM + VPA Topicals 2 pumps each night		oicals ch nightly	TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		
	14		15		16		17		18		19		20	
2X/DAY: .5mm DERMA ROLLER PTD-DBM + VPA Topicals		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		
	21		22		23		24		25		26		27	
2X/DAY: .5mm DERMA ROLLER TB4 .25ml PTD-DBM + VPA Topicals		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		TB4 .25ml ZN/GHK Topicals 2 pumps each nightly		
	28		29		30		31		1					
2X/DAY: .5mm DERMA ROLLER TB4 .25ml DTP-DBM + VPA Topicals		TB4 .25ml ZN/GHK Topicals ZN/Gł 2 pumps each nightly 2 pum		ZN/GHK Top 2 pumps eac	√GHK Topicals ZI pumps each nightly 2		ZN/GHK Topicals 2 pumps each nightly		get rid					
			Schedule 6 & 12 week follow-up appointment		619									

RETINAL/VISUAL APPLICATIONS





WARNING: DO NOT USE HIGHER POWERED DEVICES FOR THESE APPLICATIONS!

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* 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

RETINAL/VISUAL APPLICATIONS



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

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RETINAL/VISUAL®® APPLICATIONS



Open Access

Published: 24 November 2021

Weeklong improved colour contrasts sensitivity after single 670 nm exposures associated with enhanced mitochondrial function

• <u>Harpreet Shinhmar, Chris Hogg, Magella Neveu</u> & <u>Glen Jeffery</u> <u>Scientific Reports</u>

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.

RETINAL/VISUAL APPLICATIONS



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 noninvasive, 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: <u>10.3238/arztebl.m2021.0034</u>

8/arztebl.m2021.0034 PMCID: PMC8188418 PMID: <u>33785126</u>

Letters to the Editor
Low-Level Laser Therapy

Tomislav Ivandic,

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RETINAL/VISUAL APPLICATIONS



PMCID: PMC7738953

Int J Med Sci. 2021; 18(1): 109–119. Published online 2021 Jan 1. doi: <u>10.7150/ijms.52980</u>

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.

RETINAL/VISUAL APPLICATIONS



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)

2022 Mar 4:12(1):3575. doi: 10.1038/s41598-022-07427-6.

Sci Rep

Effect of low-level light therapy in patients with dry eye: a prospective, randomized, observer-masked trial

Yuli Park 1, Hoon Kim 1, Sehwan Kinuplication critation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



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

eves. 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

RETINAL/VISUAL APPLICATIONS

ERCHONIA

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

627



DEGENERATION **STUDY!**

DECREASED CORTICAL ATROPHY PRESERVATION OF THE HIPPOCAMPUS **IMPROVED SLEEP**

DECREASED BRAIN INFLAMMATION MARKERS

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REVIEW: PRIMARY MECHANISMS OF NEURODEGENERATION



1.MITOCHONDRIAL DYSFUNCTION

2.NEUROINFLAMMATION AND AUTOIMMUNITY

3.OXIDATIVE STRESS LASERS, LIFESTYLE AND NUTRITION

629

REVIEW



- 1. CAN YOU NAME THE MASTER BRAIN FREQUENCIES
- 2. CAN YOU DESCRIBE THE MASTER BRAIN PROTOCOL FOR EACH OR YOUR DEVICES
- 3. CAN YOU DESCRIBE THE BASE VAGAL PROTOCOL AND VAGAL STACKING AND THEIR IMPORTANCE TO BARRIER SYSTEMS
- 4. CAN YOU DESCRIBE THE PRIMARY PAIN TREATMENT METHODS
- 5. (SET IT AND FORGET IT, AND DOWNSTREAM)
- 5. CAN YOU NAME 3 ESSENTIAL/FOUNDATIONAL NUTRIENTS TO PROTECT YOUR BRAIN
- 6. CAN YOU NAME 3 LIFESTYLE/DIET/EXERCISE RECOMMENDATIONS TO PROTECT YOUR BRAIN
- 7. CAN YOU DESCRIBE THE OPTIMAL DOSE AND WAVELENGTH PARAMETERS THAT YOU CAN SAFELY USE ON THE BRAIN
- 8. DON'T FORGET TO REVIEW YOUR NOTES X3, REFERENCE THE PROTOCOL SECTIONS AND NEVER STOP LEARNING
- 9. ARE YOU GOING TO TAKE THE TIME AND RESOURCES TO PROTECT YOURSELF AND YOUR LOVED ONES

"INVEST IN YOURSELF AND YOUR LOVED ONES.

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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 3-3-3-2-2 XLR8, 2-2-2-2 GVL OR EVRL

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

WHAT IS IT WORTH TO YOU...





MASTER TREATMENT ALGORITHM



- 1. BRAIN BALANCE BASED ON BEDSIDE NEURO EXAM OR OTHER OBJECTIVE MEASURES
- 2. MASTER BRAIN PROTOCOL FOR EACH DEVICE
- \star
- 3. "DOWN STREAM" CLEARANCE (CONSIDER ALWAYS DOING THIS TO GET BRAIN LASER EXPOSURE)
- 4. STACKING MODALITIES WITH LOCAL LASER EXPOSURE AND CO-ACTIVATION
- 5. "SET IT AND FORGET IT" (MAY BE ALL THAT IS NECESSARY)
- 6. FUNCTIONAL MEDICINE, INTEGRATIVE NEUROLOGY AND OTHER SYSTEMIC CONSIDERATIONS Without permission of Trevor Berry, DC, DACNB is prohibited.

SUMMARY SECTION



SUMMARY SECTION OF "PROTOCOLS" PAGES NOTE: PROTOCOLS ARE MEANT TO BE REFERENCE GUIDES AND MAY NOT COVER THE ENTIRETY OF THE CONDITION MANAGEMENT OR SUPPORT. NEVER CLAIM YOU ARE TREATING OR CURING ANY DISEASE



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SEMINAR SUGGESTIONS



-TRY NOT TO ATTEMPT TO FOLLOW THE VERBIAGE OF EACH SLIDE LINE BY LINE

-USE THE SLIDES FOR FUTURE REFERENCE AND REVIEW X3

-DR. BERRY PURPOSELY CREATES HEAVY CONTENT SLIDES AND COMPLEX MATERIAL

FOR MULTIPLE REASONS INCLUDING GOING BACK TO REVIEW AND REFERENCE IN

MORE DETAIL, BDNF AND COGNITIVE RESERVE AND RESEARCH REFERENCES

-PROTOCOL SECTION AND SUPPORTING/OLDER MATERIAL AT THE END OF THE SLIDES

-TRY TO ASSOCIATE CONDITIONS WITH CURRENT PATIENTS, FAMILY MEMBERS OR

YOURSELF

-START MONDAY WITH BASIC DIAGNOSTICS AND CLINICAL APPLICATIONS WITH EVERY PATIENT ON MONDAY AND BUILD FROM THERE

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)

MASTER BRAIN PROTOCOL



1. MOST COMMONLY USED FREQUENCIES (IN ORDER): 40 - 10 - 1 - 60 EX. OPTIMIZE: 10-40-10-40 MASTER BRAIN: 1-10-40-60 REST-DIGEST: 1-10-1-10 VAGUS/PEAK ALPHA: 10-10-10-10

2. BASE PREVENTION AND TREATMENT BRAIN PROTOCOLS:

FX 405 6 MINUTES WITH 1-40 TO PREFRONTAL CORTEX, 10-40 APICAL/PARIETAL, 10-40 BRAINSTEM CEREBELLUM IT'S OK TO TURN IT FROM SAGITTAL TO CORONAL PLANE AT 1/2 WAY. IF POSITIONED USE THE 1-10 TO THE RIGHT HEMISPHERE

FX 635 EXACT SAME SET UP AND FREQUENCIES AS FX 405 BUT DO A 10 MINUTE PROTOCOL (AGAIN, IT'S OK TO TURN THE DIODES FROM SAGITTAL TO CORONAL AT THE HALF WAY 5 MINUTE MARK)

FINISH EITHER FX DEVICE WITH THE "GUT SHOT". SCM/NECK, LUNG/UPPER GI, LOWER GI MINUM 4 MIN UP TO 10 MIN

HAND HELDS (USE ANY OF THE ABOVE COMMONLY USED FREQUENCIES):

GVL 2 MINUTES BILATERAL PREFRONTAL CORTEX, 2 MINUTES APICAL/PARIETAL, 2 MINUTES BRAINSTEM/CEREBELLUM

EVRL DO THE SAME POSITIONS AND GVL BUT 3 MINUTES PER REGION

XLR8 DO THE SAME POSITIONS AS OTHER HAND HELDS BUT DO 4 MINUTES PER REGION

FINISH ALL HAND HELDS WITH MINIMUM 2 MIN + 2 MIN VAGAL PROTOCOL (SCM/NECK AND OVER GUT) CAN DO UP TO 10 MINUTES (IE. WAND THE DEVICE OR 5 & 5 MIN NECK AND GUT)



THE MASTER PROTOCOL NEUROMUSCULOSKELETAL



1. ASSESS THE BALANCE OF THE CENTRAL NERVOUS SYSTEM. A COMPREHENSIVE DIAGNOSTIC AND BEDSIDE WORK-UP IS ALWAYS RECOMMENDED. 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 BEFER 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 PRFRONTAL 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

SOME OF DR. BERRY'S FAVORITE FREQUENCY SETTINGS



MASTER BRAIN 1-10-40-60 (70% OF MY PATIENTS, INCLUDING UPSTREAM/DOWNSTREAM PAIN)

BRAIN OPTIMIZATION 10-40-10-40 VAGUS 10-10-10-10

REST DIGEST 1-10-1-10

DELTA 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 RERUCHAS LASER A-Z PROTOCOL BOOK!

COMMON PATHOGEN AND IMMUNE FREQUENCIES

Community 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

SOLFEGGIO FREQUENCIES



What are Solfeggio Frequencies?

ALSO READ ABOUT ROYAL RIFE

The Solfeggio Frequencies are a series of 6 electromagnetic musical tones that the Gregorian Monks were said to use when they chanted in meditation. Rediscovered in 1974 by Dr. Joseph Puleo, the Solfeggio Frequencies are said to deeply penetrate the conscious and subconscious mind, stimulating inner healing. Dr. Puleo was intuitively led to rediscover these healing frequencies in the Book of Numbers (a book in the Hebrew Bible), using a numerological technique to decipher the six repeating codes he found. The result was the rediscovery of the Solfeggio Frequencies.

Physicist, inventor, and electrical engineer Nikola Tesla once said, "If you only knew the magnificence of the 3, 6 and 9, then you would hold a key to the universe." Interestingly, these three numbers form the root vibration of the six Solfeggio Frequencies.

- UT 396 Hz transforming grief into joy and guilt into for giveness
- RE 417 Hz clears negativity and removes subconscious blockages
- MI 528 Hz stimulates love, restores equilibrium, repairs DNA
- FA 639 Hz strengthens relationships, family, and community unity
- SOL 741 Hz physically cleanses the body from all types of toxins
- LA 852 Hz a waite anscitation of the proving the

MITOCHONDRIA AND NEURODEGENERATION



SUMMARY OF MITOCHONDRIAL ETC COMPLEXES, COMMON NEURODEGENERATIVE DISEASES AND ERCHONIA WAVELENGTHS:

ALPHA SYNUCLEIN DISEASES INCLUDING PARKINSON'S, LEWY BODY DEMENTIA AND MULTIPLE SYSTEM ATROPHY (MSA): ETC COMPLEX 1 = VIOLET

TAU HYPERPHOSPHORYLATION DISEASES INCLUDING ALZHEIMER'S, FRONTOTEMPORAL DEMENTIA, PICK DISEASE, PROGRESSIVE SUPRANUCLEAR PALSY (PSP) AND CORTICOBASAL ATROPHY ETC COMPLEX 1 = VIOLET

> MHTT IN HUNTINGTON'S CHOREA ETC COMPLEX 2 = VIOLET



mSOD1 IN AMYOTROPHIC LATERAL SCLEROSIS (ALS) AND ALZHEIMER'S ETC COMPLEX 3 = GREEN

ALZHEIMER'S DISEASE: ETC'S 1,3 & 4 (VIOLET-GREEN-RED) STACK ALL 3 ERCHONIA WAVELENGTHS

PARKINSON'S DISEASE: ETC'S 1 & 4 (VIOLET-RED) FX 405 OR EVRL

HUNTINGTON'S DISEASE: ETC'S 2, 3 & 4 (VIOLET-GREEN-RED) STACK ALL 3 ERCHONIA WAVELENGTHS

ALS: ALL 4 COMPLEXES SO USE ALL 3 ERCHONIA DIODES AGING: ETC'S 1,2 & 4 (VIOLET-RED) FX 405 OR EVRL

DEFAULT TO MASTER BRAIN PROTOCOLS FOR EACH CONDITION USING 1-10-40-60

VAGAL NERVE STIMULATION: MASTER PROTOCOL



VAGAL NERVE STIMULATION USING ERCHONIA LASERS:

CONSIDER "VAGUS" SETTING 10-10-10-OR "MASTER BRAIN" 1-10-40-60 OR "REST-DIGEST" 1-10-1-10

MINIMUM 2 MINUTES OVER THE SCM AND 2 MINUTES OVER THE GUT STACK WITH YOUR PERCUSSOR

UP TO 10 MINUTES TOTAL CONSIDER PREFRONTAL CORTEX THEN BASE OF SKULL/MEDULLA THEN OVER THE EAR/TRAGUS REGION THEN SCM THEN OVER THE GUT RULE OF 2'S



RIGHT VERSUS LEFT? BOWEL SOUNDS/ MOVEMENTS, LUBRICATION/ED ASSESS - BILATERAL BP, PULSE OX/BPI, PALATAL PARESIS, CAPILLARY REFILL ETC CLINICAL CONSIDERATIONS -CONTRALATERAL CORTICAL +,



THE VAGAL NERVE STACK:

- 1. ERCHONIA LLLT TO THE SCM AND GUT WITH ANY OF THE 10 HZ SETTINGS. MINIMUM 2 MINUTES EACH (DID 10 MINUTES IN HRV STUDY)
- 2. PERCUSSOR GI. 4 LAPS AROUND THE GUT IN A CLOCKWISE FASHION. FINISH OVER THE UMBILICUS
- 3. HOOLEST PRIME TO SCM (#3 SETTING)
- 4. REZZIMAX TO HEAD/THYMUS/VAGUS
- 5. BOX BREATHING/RELAXATION MUSIC

Management of a "leaky gut"

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- Identify the sources of inflammation and reactivity and remove them (Wheat and Gut Zoomer, Food Sensitivity Panel, Stress, SAD diet, Antibiotics, Chemical Stressors, AND MOST IMPORTANT; INFECTIOUS AGENTS LIKE BACTERIA, VIRUSES, FUNGI, PARASITES ETC.)
- PHASE 1: Gut repair, liver detox and inflammation reduction. Month 1 Xymogen 6 DAY DETOX KIT. Contains Opticleanse GHI (for GI function and repair, Liver Detox and Inflammation cytokine regulation), Drainage (homeopathic for colon, kidney and liver detox), ColonX (like a colonic without the hassle) and ProbioMax DF (30 Billion CFU including HOWARU Bifido Lactis HN019). Consider 1 -> 3 day fast with water or continue Opticleanse GHI,1 scoop 2-3 x/day. HistDAO 1-4 daily depending on histamine sensitivity. Vitamin A, LIQUID D 7K/DAY. Omega MonoPure 1300 EC (For the duration of care). S-Acetyl Glutathione/NAC (1each daily for duration of care). Probiomax Ig26 DF (2+ Caps/day) once daily formula complete. Optimagneuro 1 scoop or less daily. Oncoplex (2 caps daily). *NEW GI BALANCE, 2 SCOOPS/DAY INITIAL 14 DAYS THEN 1 SCOOP FOR REMAINDER OF 3 MONTHS
- May need other management such as adrenal support/stress reduction, sleep help like Melatonin CR
- CONSIDER MEDPAX FOR PATIENT COMPLIANCE!
- Continue for 3 months minimum. Retest Barrier Systems via Neural and Wheat Zoomers. Retest food sensitivities.
- MUST ADDRESS BRAIN-GUT AXIS! VAGAL STIM. STRESS REDUCTION.



FASTING: INTERMITTENT #16:8", T DAY, 3 DAY+



A Look Inside Vibrant GZ 3.0

- Part 1: Commensal Bacteria (DNA)
- Part 2: Pathogens (DNA)
- Part 3: Functional Markers (Mass Spec)
- An immense amount of data summarized into 3 very concise, actionable pages

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VibrantWellness
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THE 5 R'S OF GUT HEALTH 1. REMOVE

1. REMOVE: ELIMINATE OFFENDING FOODS, ALLERGENS AND UNNECESSARY OTC MEDICATIONS COMMONLY USED NUTRITIONAL SUPPORT

BERBEMYCIN: BROAD SPECTRUM KILL AGENT

Amount Per Serving %Daily Value Oregon Grape (Mahonia aquifolium)(roots)

Grapefruit 5:1 Extract (Citrus × paradisi)(seed)

Berberine HCI

Uva Ursi Extract (Arctostaphylos uva-ursi)(leaves)(16% arbutin)

PROBIOMAX IG 26 DF: PASSIVE IG THERAPY PLUS SPORE BASED PROBIOTIC

FIXES LEAKY GUT AND INOCULATED AGAINST THE 29 MOST COMMON PATHOGENS INCLUDING C. DIFF! AVIAN GRAS Y IMMUNOGLOBULIN THERAPY IS MORE IMMUNE NEUTRAL TOWARDS HUMAN IMMUNOLOGY

> CELLCORE BIOSCIENCES FOUNDATIONAL PROGRAMS OTHER TARGET SPECIFIC PROTOCOLS LIKE CANDICIDAL LAURICIDIN/MONOLAURIN



RCHONIA

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390 mg **

350 mg

250 mg



5 R'S 2. REPLACE: FORTIFY NORMAL DIGESTIVE FUNCTION



ERCHONI

RUN VIBRANT WHEAT AND GUT 3.0 ZOOMER, FOOD SENSITIVITY AND MICRONUTRIENT PANELS



BASIC GUT HEALTH XYMOGEN MEDPAX: 1 PROBIOMAX IG 26 DF 1 XYMOZYME HCL 1 HISTDAO





HistoDAO[™] is a patented enzyme formula containing diamine oxidase (DAO) — the main enzyme responsible for the degradation of ingested histamine. This enzyme has been clinically tested and found to break down food-derived histamine in the digestive tract. DAO is not absorbed and does not have systemic activity. HistDAO does not manage or address antibodyrelated or IgE-related food allergies.* ERCHONIA VAGAL PROTOCOL SCM AND INTESTINAL REGION 10 HZ SETTING (10-10-10-10) 2+ MINUTES EACH REGION EVERY VISIT PERCUSSOR GI/VAGAL STIM



ProbioMaxe IG 26 DF features clinically validated ingredients to support microbiome wellness and overall immune health. LactoSpore® (*Bacillus coagulans* MTCC5856) is a unique strain of shelf-stable L (+) lactic acid-producing bacteria with a naturally protective spore coating. IG 26 DF (IgY Max™), hyperimmunized egg powder, provides immunoglobulins and immune cofactors to support the body's natural defenses by limiting non-beneficial microbial adhesion.*

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5 R'S OF GUT HEALTH 3. REPAIR

		eCommunity Educatio
Amount Per Serving	Supports Gastrointestinal Health*	
%DV Calories	Supports Mucous Membrane Integrity*	
40	Promotes Healthy Bowel Function*	
	*GI BALANCE CAPS DO NOT CONTAIN	
Total Carbohydrate	GLUTAMINE IF THERE ARE CONCERNS	17
7 α	ABOUT CONTRAINDICATIONS (COLON	XYMAREN
3%†	CANCER, GLUTAMATE ETC)	GI Balance
Dietary Fiber	Croop Viuifruit (Actividio dellaiseo)/fruitVC2 500 ALL estinidio)24	Southing GI Tract Support*
2 g	Green Kiwiiruit (<i>Actinidia deliciosa</i>)(Iruit)(62,500 AU actinidin)S I	
7% Total Sugars	400 mg	150 ^{Vegetarian} Dietary Supplement
	Marshmallow (<i>Althaea officinalis</i>)(root)	THE PROPERTIES
1 g **		
Zinc (as zinc I-carnosine)	400 mg **	5 CAPS/DAY
32.25 mg 293%	Licorice Extract (<i>Glycyrrhiza glabra</i>)(root)(3.5% glabridin)S2	30 DAYS
	300 mg	
	** Ouercetin (as quercetin dihydrate)	
4 g **		
Slippery Elm (<i>Ulmus fulva</i>)(bark)	200 mg	a l
2.0	Prune (<i>Prunus domestica</i>)(fruit)	XYNABEN
2 Y **	200 mg	GI Balance Powder
N-Acetyl-D-Glucosamine	200 Ing **	Southing GI Tract Support* #PV:011001-03(011010)00101
2 g	Chamomile Extract (<i>Matricaria recutita</i>)(flowering head)(1% apigenin-7-glucoside)	NULLA CHAIFLAVOR
** Aloe Vera 200:1 Extract (<i>Aloe barbadensis</i>)(leaf gel)	200 mg	EXCLUSIVE PROFESSIONAL FORMULAS
	Cat's Claw (<i>Uncaria tomentosa</i>)(bark)	
600 mg **		2 SCOODS/DAV
Porcine Gastric Mucin	200 mg tion_citation or distributic652h this material in whole or in part	2 JUUPS/DAT
	Okra (Hibiscus esculentus)(00d)	14 DAYS

4. REINOCULATE (CONSIDER ROTATION)



CONSIDER SOLUTIONS FOR LONG TERM GUT HEALTH:

1. ALWAYS ASSESS AUTONOMIC FUNCTION AND THE BRAIN GUT AXIS (HRV, BP, CAROTID RESPONSE). A HEALTHY BRAIN AND VAGAL OUTPUT IS ESSENTIAL FOR PROPER DIGESTIVE FUNCTION AND TONE! VAGAL NERVE PROTOCOLS.

5. REBALANCE

2. EDUCATE PATIENT OF THE HAZARDS OF A STRESSFUL LIFESTYLE (JOBS, RELATIONSHIPS, EVEN RELAXED EATING. TAKE TIME TO ENJOY MEALS. DON'T EAT SCARED! USE FOOD SENSITIVE AND ALLERGY TESTING AS THE GOLD STANDARD FOR ELIMINATION. WHEN IN DOUBT, TRY GLUTEN FREE, DAIRY FREE AND SOY FREE.

3. CONSIDER A FULL DETOX PROGRAM. 1-3 DAY WATER FASTS ARE AN EFFECTIVE REPAIR AND RESET IDEA. XYMOGEN'S DETOX RENEWAL MICRO-KIT IS AN EASY TO FOLLOW PROGRAM.

4. WATCH FOR CO-MORBIDITIES AND PATHOLOGY. MOLD AND OTHER PATHOGENS MAY BE IN PLAY. THE PATIENT MAY NEED A REFERRAL.

5. PROPER BOWEL MOVEMENTS ARE A MUST TO ELIMINATE WASTE AND TOXINS. MAGNESIUM AND FIBER SUPPORT (XYMOGEN'S OPTIFIBER LEAN) MAY BE INDICATED.



ERCHONIA

ERCHONIA



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

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.



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



XymoZyme[•]HCl

Disputes Supplement

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



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



ADIPOSE REDUCTION



BASIC 8 TREATMENT PLATFORM: 2X/WEEK FOR 3 WEEKS 1X/WEEK FOR 2 WEEKS FOLLOW ERCHONIA'S TREATMENT PLAN (LYMPHATICS, WALK, H20 ETC) PRE-SCREEN MAF

> OTHER CONSIDERATIONS: EXTENDED TX PLANS BASED ON SIZE AND GOALS REFERRAL VISITS "MEMBERSHIP MODEL"

ZERONA Z6 (OTC)





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 657



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SET IT AND FORGET IT



STANDARD BASE POSITION LOW BACK PAIN. MAX 20 MINUTES. 10 MINUTES AVERAGE



ÉRCHONIA

CURRENT BACK PAIN TREATMENT MODEL

OVERVIEW OF (LOW) BACK PAIN MANAGEMENT:

- 1. IS IT PERIPHERAL SENSITIZATION
- 2. UTILIZE ERCHONIA LASER DEVICES AS PART OF THE INITIAL TREATMENT TRIAL AND DIAGNOSTICS
- 3. APPLY PROLOGEL IF AVAILABLE
- 4. MAKE SURE THEY ARE PROPERLY OFFLOADING PARASPINAL MUSCLES AND PELVIC INSTABILITY WITH HIP EXTENSOR AND EXTERNAL ROTATION ACTIVATION (THE "GLUT CLAMSHELL LASER" PROTOCOL. MAKE SURE IT IS GLUT ISOLATED AND NOT RECRUITING PARASPINALS, QUADS, HAMSTRINGS...
- 5. ALWAYS GIVE AT LEAST 6 VISITS FOR LASER TREATMENT TRIALS, 12 FOR COMPLEX CONDITIONS LIKE CRPS
- 6. ASSUMING NO CONTRAINDICATIONS, REDUCE ANY BIOMECHANICAL FAULTS/SUBLUXATION SOURCES WITH CHIROPRACTIC CARE
- 7. ADDRESS THE PATIENTS INDIVIDUAL NEEDS AND GOALS
- 8. BASED ON THAT, RX HOME THERAPIES OF ACTIVITY THAT THE PATIENT WANTS TO PARTAKE IN
- 9. SEND THEM HOME WITH THEIR ACUTE/INITIAL TREATMENT PLAN KIT AND MUTUAL RECOMMENDATIONS. THE KIT SHOULD INCLUDE AN ERCHONIA HAND HELD LASER (RENTAL PROGRAM), RESISTANCE LAP BAND, BOTTLE OF PROLOGEL (RED LIDOCAINE VERSION IN SEVERE ACUTE CASES)
- 10. DAILY GLUT ISOMETRIC HOLDS SIDE LYING. WORK UP TO 20 MINUTES (10 MINUTES EACH SIDE, ONE MINUTE ON, ONE MINUTE OFF)
- 11.ONCE FINISHED GLUT ISO COMBO HAVE THE PATIENT LASER THE VAGUS NERVE AND PREFRONTAL CORTEX WHILE THEY VISUALIZE PERFORMING TASKS THAT THEY WANT TO DO WITHOUT PAIN, AND MEDITATIVE CBT FOR HAPPY INTROSPECTION
- 12. ENCOURAGE THEM TO GET POSITIVE REINFORCEMENT OF BASIC ADL'S (IE. IF IT USED TO HURT GETTING UP OFF OF THEIR FAVORITE CHAIR, HAVE THEM TRY IT AFTER DOING THEIR HOME LASER PROTOCOLS ABOVE)
- 13. AS THEY ESTABLISH POSITIVE REINFORCEMENT AND SAFTEY OF BASIC ADL'S, ENCOURAGE THEM TO PARTAKE IN PHYSICAL ACTIVITIES THEY ENJOY SUCH AS WALKING, HIKING, YOGA...WHATEVER THEIR FAVORITES ARE!

DYNAMIC LOW BACK PROTOCOL





661

ACUTE LOW BACK HOME PLAN





EVRL

XLR8

\$300/WK

2. BOTTLE OF PROLOGEL IF NO CONTRAINDICATIONS TO LIDOCAINE **USE RED FOR ACUTE PAIN**



3. GIVE PATIENT RESISTANCE APPROPRIATE THERABAND FOR GLUT CLAMSHELLS

SUGGESTED PACKAGE PRICE FOR 1 WEEK RENTAL WITH XLR8, 1 BOTTLE OF PROLOGEL AND 1 BAND

\$400/WK \$500/WK Duplication, citation or distribution of this material in whole or without permission of Trevor Berry, DC, DACNB is prohibited.

GVL

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

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's Clinical Affairs Manager stated, "The latest 510(k) statement once again proves Erchonia's commitment to the science of low-level laser therapy through blinded and controlled clinical trials to obtain FDA 510(k) marketing statements. As the World Leader in Low Level Laser Technology, we have always believed we



THE MASTER PROTOCOL **NEUROMUSCULOSKELETAL**



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
- 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 THE CITY OVER A FOUNDON IS HERE OF RECOMPART without permission of Trevor Berry, DC, DACNB is prohibited.



PAGE 1

- 1. UPREGULATE THE VAGUS NERVE FOR 2 MINUTES BRAIN SETTING (1-10-40-60).
- 2. TRANSCRANIAL LASER BASE BRAIN SETTING 1-10-40-60 TO THE CORTEX OF THE INVOLVED SIDE (IE RIGHT MIDDLE CEREBRAL ARTERY OR INTERNAL CAPSULE INFARCT LASER RIGHT CORTEX). LASER POSITION CAN BE BIASED TOWARDS DESIRED REHAB (IE WHEN DOING LEFT HAND MOTOR REHAB LASER THE RIGHT FRONTAL LOBE). ALSO LASER THE CONTRALATERAL CEREBELLUM WITH BASE BRAIN SETTING (IE RIGHT CORTICAL = LEFT CEREBELLUM). WITH THE FX AND PLTOUCH THIS CAN BE DONE AT THE SAME TIME. PARIETAL LOBE IF PAIN BASED.
- 3. HAVE PATIENT PRE-VISUALIZE THE DESIRED MOVEMENT OF REHAB WITH THE EYES CLOSED. (IN PAIN SYNDROMS VISUAL MOVEMENT WITHOUT PAIN)
- 4. ACTIVATE THE FRONTAL LOBE WITH SACCADES (IE TO FIRE RIGHT FRONTAL LOBE USE LEFTWARD SACCADES. ANGLE UP AND AWAY FOR MORE LATERALIZATION).
- 5. MIRROR OVER THE TARGET LIMB SO THAT THE PATIENT HAS THE VISUAL PERSPECTIVE THAT IT IS THE INVOLVED LIMB BUT WILL ACTUALLY BE THE CONTRALATERAL "GOOD" LIMB.
- 6. HAVE THE PATIENT ACTIVELY MOVE THE GOOD LIMB IN WHATEVER DESIRED PATTERN YOU ARE TARGETING (IE ELBOW FLEXION). THE MIRROR IS PLACED IN A WAY THAT THE PATIENT IS LOOKING AT THE LIMB MOVING IN THE MIRROR AND THE BRAIN THINKS THE INJURED LIMB IS MOVING BUT IT'S ACTUALLY THE NON-INJURED LIMB. (NOTE: THIS CAN BE DONE FOR OTHER CONDITIONS LIKE CRPS, FROZEN SHOULDER, PHANTOM LIMB PAIN ETCOME IN whole or in part CONT...





MIRROR THERAPIES





Du



MIRROR THERAPIES





le or in part ohibited.

PAGE 2

7. REPEAT THE GOOD LIMB MOVEMENTS IN THE MIRROR WHILE THE PROVIDER OR FAMILY MEMBER MOVES THE INJURED LIMB BEHIND THE MIRROR FOR THE PATIENT PASSIVELY. LASER THE DISTAL TISSUE IN PAIN/POST-OP ETC. 8. SENSORY CO-ACTIVATION SHOULD BE APPLIED TO THE BAD SIDE DURING THIS PROCEDURE. TENS, VIBRATION MASSAGE ETC CAN BE USED ON THE INJURED SIDE AT THE SAME TIME AS THE MOTOR/MIRROR REHAB.

9. IF THE PATIENT IS ABLE TO DEVELP NEUROPLASTICITY TO THE POINT OF GETTING THEIR OWN ACTIVE MOVEMENT ON THE INJURED SIDE YOU CAN START TO ADD ACTIVE THERAPY BEHIND THE MIRROR INSTEAD OF PASSIVE.

10. ONCE THEY ARE ABLE TO DO ACTIVE CARE, REMOVE THE MIRROR AND TRANSITION INTO "CROSS-CRAWL PATTERNS" (IE IF WORKING ON LEFT ELBOW FLEXION BEHIND THE MIRROR AND THE PATIENT IS ABLE TO START ACTIVATING THE LEFT ELBOW INTO FLEXION, REMOVE THE MIRROR AND HAVE THEM OBSERVE THE LEFT ELBOW INTO FLEXION WHILE THE RIGHT ELBOW GOES INTO EXTENSION. THIS CAN BE BUILT UP INTO FULL CROSS-CRAWL MARCHING). 11. OTHER CO-ACTIVATION MAY BE APPLIED SUCH AS HEMI-STIM VEP'S, OTHER SPECIAL SENSES (OLFACTION, AUDITORY ETC).

669

12. THIS MIRROR TECHNIQUE CAN BE DONE FOR ANY LIMB.



ERCHONIA

ERCHONIA

LASERS A - Z MANUAL JEROME RERUCHA

BASE MUSCULOSKELETAL/PAIN DEFAULT UPREGULATION

9-16-42-53

TENDONITIS 1-21-250-2720

NEUROGENIC INFLAMMATION/FASCIITIS 9-16-33-36

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

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WOUNDS 3-16-24-111





FX LE PROTOCOL BASIC



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



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PERIPHERAL NEUROPATHY ETIOLOGIES



COMMON DIFFERENTIALS FOR PERIPHERAL NEUROPATHY:

-DIABETIC NEUROPATHY. COMMON LABS: HBAIC, FASTING GLUCOSE, C-PEPTIDE

-B VITAMIN/METHYL DONOR DEPLETION COMMON LABS: CBC WITH DIFF, HOMOCYSTEINE, MICRONUTRIENT PANEL, MMA ELEVATION (LOW B12), GENETICS LIKE MTHFR (VIBRANT NUTRIPRO)

-STATINS

-GLUTEN SENSITIVITY/TRANSGLUTAMINASE 6 COMMON LABS: WHEAT ZOOMER (VIBRANT)

673

-CHEMOTHERAPY INDUCED

-VITAMIN B6 TOXICITY

-EMF EXPOSURE



RESTLESS LEG SYNDROME (WILLIS-EKBOM DISEASE, RLS) CLINICAL CONSIDERATIONS



-RULE OUT ANEMIA PATTERS ESPECIALLY IRON DEFICIENCY: #1 ENVIRONMENTAL TRIGGER (RUN CBC AND FULL IRON PANEL) -CONSIDER OTHER MICRONUTRIENT DEPLETION ESPECIALLY MAGNESIUM (RUN MICRONUTRIENT PANEL) -WATCH PROTON PUMP INHIBITORS/H2 BLOCKERS/ACID BLOCKERS (CAUSE CO-FACTOR DEPLETION AND OTHER POSSIBLE MECHANISMS) SEE STUDY BELOW. SUMMARY: THESE MEDS DOUBLE THE RISK OF RLS! -WATCH DOPAMINE (ARE THEY ON A MEDICATION DEPLETING DOPAMINE, PARKINSONIAN RELATED ETC)

> NUTRITION CONSIDERATIONS FOR RLS: IRON GLYCINATE (IF LAB OR FUNCTIONALLY LOW SERUM IRON) OPTIMAG NEURO (MAGTEIN, MALATE, GLYCINATE) OMEGA 3 FATTY ACIDS 3/DAY A GOOD MULTIVITAMIN (EX, VIT C HELPS IRON ABSORPTION) CALMING AGENTS LIKE VALERIAN, CHAMOMILE, EPSOM BATHS L-THEANINE (IMPROVES CALMING & SLEEP PATTERNS) DOPAMINE AGONISTS AS INDICATED CBD (FORTIFY ELITE 1-2 PM 25-50 MG) OR HEMP MONOPURE

TREATMENT CONSIDERATIONS FOR RLS: IN OFFICE - MASTER BRAIN SETTING FULL BRAIN/VAGAL PROTOCOL (1-10-40-60) FX PFC/VAGAL/CB 10 MIN 635, 6 MIN 405 STACK WITH GLUT ISOMETRICS (TBAND CLAMSHELL 30 SECOND HOLDS X3-4) WHILE DOING MULTIPLE SETS OF ANTI-SACCADES SWITCH LASER TO FULL LOWER EXTREMITY EXPOSURE (SEE LAST SLIDE) 5 MINUTES EACH LUMBOSACRAL PLEXUS-KNEE/BIFURCATION-TARSAL TUNNEL/PLANTAR

Association of proton pump inhibitor and histamine H₂-receptor antagonists with restless legs

syndrome

Eric J Earley, Maria Didriksen, Bryan R Spencer, Joseph E Kiss, Christian Erikstrup, Ole B Pedersen, Erik Sørensen, Kristoffer S Burgdorf, Steven H Kleinman, Alan E Mast ... Show more Sleep, Volume 44, Issue 4, April 2021

PERIPHERAL NERVES



Bioengineering (Basel). 2018 Jun 9;5(2). pii: E44. doi: 10.3390/bioengineering5020044.

Photobiomodulation Therapy (PBMT) in Peripheral Nerve Regeneration: A Systematic Review. Rosso MPO1, Buchaim DV2,3, Kawano N4, Furlanette G5, Pomini KT6, Buchaim RL7,8.

Author information

Abstract

Photobiomodulation therapy (PBMT) has been investigated because of its intimate relationship with tissue recovery processes, such as on peripheral nerve damage. Based on the wide range of benefits that the PBMT has shown and its clinical relevance, the aim of this research was to carry out a systematic review of the last 10 years, ascertaining the influence of the PBMT in the regeneration of injured peripheral nerves. The search was performed in the PubMed/MEDLINE database with the combination of the keywords: low-level laser therapy AND nerve regeneration. Initially, 54 articles were obtained, 26 articles of which were chosen for the study according to the

inclusion criteria. In the qualitative aspect, it was observed that PBMT was able to accelerate the process of nerve regeneration, presenting an increase in the number of myelinated fibers and a better lamellar organization of myelin sheath, besides improvement of electrophysiological function, immunoreactivity, high functionality rate, decrease of inflammation, pain, and the facilitation of neural regeneration, release of growth factors, increase of vascular network and collagen. It was concluded that PBMT has beneficial effects on the recovery of nerve lesions, especially when related to a faster regeneration and functional improvement, despite the variety of parameters.

CLINICAL CONSIDERATIONS: 2X/WEEK 2-2-2-2 SETTING 10 MINUTES 8-12 VISITS (4-6 WEEKS) YOU SHOULD OBTAIN SUBJECTIVE AND OBJECTIVE CHANGES DEPENDING ON SEVERITY OF INJURY AND HOW OLD THE INJURY IS. CONSIDER NERVE ROOT UPREGULATION AND AT TARGET



GREEN WAVELENGTH



INJURED NERVES

The continuation of this work with different wavelengths, showed that green 540 nm is more effective than 632 nm.

ADD THE GREEN WAVELENGTH OF THE GVL TO YOUR **PERIPHERAL NERVE APPLICATIONS!** CONSIDER 2-2-2-2 HZ FREQUENCY SETTING 677



REVIEW

New trend in neuroscience: Low-power laser effect on peripheral and central nervous system (basic science, preclinical and clinical studies)

Semion Rochkind and Georges E. Ouaknine

eery. Tel-Aviv Sou The source of th erapy p oped in the la

Keywords: Low-power laser irradiation, peripheral nerve and spinal cord injury, spinal c brain transplantation, tethered spinal cord

Correspondence and reprint requests to: Dr Semion Rochkind, Department of Neurosurgery, Ichilov Hospital, Tel-Aviv Sourasky Medical Center, 6 Weizman Street, Tel-Aviv 64239, Israel. Accepted for publication July 1991.

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HEADACHES

SUPRATROCHLEAR NERVE





© 2021 All Rights Reserved | Please do not copy or redistribute without written consent of the presenters. ERCHONIA **HEADACHES 2 MINUTES ERCHONIA INFRATROCHLEAR** (GVL) HAND HELD **NERVE AND TRIGEMINAL** 1-10-40-60 **GANGLIA** Supraorbital nerve Supratrochlear nerve TRIGEMINAL GANGLION 100 Supraorbital nerve Supratrochlear nerve Occipital artery Infraorbital Infraorbital nerve nerve Greate occipital nerve Lesser Mental occipital nerve nerve Sternocleidomastoid Great-Mental nerve auricular muscle Supraclavicular nerve nerve CONSIDER MASTICATION RELEASE TECHNIQUES DURING THIS TIME (INTERNAL AND EXTERNAL) © 2021 All Rights Reserved | Please do not copy or redistribute without written consent of the presenters.

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CHEMO BRAIN

CHEMO BRAIN:

ÉRCHONIA

ERCHONIA TRANSCRANIAL LASER 1-10-1-10 (2-10 MINUTES DEPENDING ON THEIR METABOLIC CAPACITY AND FATIGUE RATIOS)

NUTRTION SUPPORT

LOW SUGAR DIET. PROMOTE KETOSIS (MCT OILS) HIGH DOSE OMEGA 3 FATTY ACIDS MINMUM 3 GRAMS PER DAY (XYMOGEN HEMP MONOPURE IN PM, AND DHA MONOPURE AM RECOMMENDED) S-ACETYL GLUTATHIONE AND NAC AM AND PM OTHERS: VINPOCETINE, GINKGO BILOBA, BACOPA MONNIERI, PHOSPHATIDYLSERINE (ALL ARE IN XYMOGEN'S MEMORALL!)

NAD+ through NAD+ precursors, such as NMN (Nicotinamide mononucleotide) or Nicotinamide riboside

Methylene blue 40-50 mg with 250 ml 5% dextrose over 45 minutes *ALWAYS WATCH CONTRAINDICATIONS AND INTERACTIONS OF SUPPLEMENTS WITH CHEMOTHERAPY DRUGS, A GOOD REFERENCE IS "NATURALMEDICINES.COM" without permission of Trevor Berry, DC, DACNB is prohibited.



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

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

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 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

DR. BERRY'S MEDPAX 2024





ERCHONIA LLLT BRAIN/VAGAL, PAIN AREAS, IMMUNE BOOST ETC





- -1 OMEGAMONOPURE COQ10
- -1 ACTIV NUTRIENTS PERFORMANCE
- -1 ACTIV NUTRIENTS (MULTI WITHOUT COPPER AND IRON)
- -1 S-ACETYL GLUTATHIONE

-1 OPTIMAG NEURO CAP (MG NORMALLY IN PM FOR MOST) -1 HISTDAO

PM:

- -1 NAC
- **-1 CORTISOLV**
- -1 K2-D3 5000
- -1 ONCOPLEX WITH MYROSINASE
- -1 PROBIOMAX IG 26 DF (ROTATE)
- -1 HEMP MONOPURE

= L-SERINE AND RESVERATROL

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NON-ALCOHOLIC FATTY LIVER DISEASE

Fatty Liver Affects 80% of obese Women and 30% general population Over 40 — Here's How to Prevent and Reverse It

1. EAT EGGS AND OTHER HIGH CHOLINE FOOD SOURCES

University of North Carolina study, 80 percent of women with fatty liver who upped their choline intake had a 100 percent reversal of the condition, often within 30 days. **500 mg DAILY. *XYMOGEN PHOSPHALINE = 2.7 GRAMS!**

ERCHONIA LOW LEVEL LASER THERAPY: 12 VISIT TREATMENT TRIAL OVER 3 MONTHS 5 MINUTES WAND OR FX SCAN OVER LIVER/RUQ (ERCHONIA CLINICAL TRIAL IS 1X/WEEK FOR 10 MINUTES WITH EVRL) FREQUENCIES 53-537-55-751

> PRE AND POST CHECK LIVER ENZYMES (**ALT**/AST/GGT) OTHER CONSIDERATIONS: HS-CRP, HBA1C, LIPID PANEL, ESR

CURCUPLEX-95, NRF2 ACTIVATOR

4. EXERCISE

Just 30 minutes of activity daily helps the liver burn 39 percent of its stored fat, say British scientists. STOP ALCOHOL/SUGAR INTAKE!

Brition Solentisto

Eur Endocrinol, 2020 Oct; 16(2): 152–155. Published online 2020 Oct 6. doi: <u>10.17925/EE.2020.16.2.152</u>

685

The Prevalence and Predictors of Non-alcoholic Fatty Liver Disease in Morbidly Obese Women – A Cross-sectional Study from Southern India

ACUTE PHASE TBI NUTRITIONAL SUPPORT



ERCHOM

PhosphaLine

Summary: Acute Phase TBI Management (<4 Weeks)



24 HOUR WATER FAST ON DAYS 7, 14, 21 AND 28 CONSIDER ATP IGNITE AM CREATINE + COFACTORS AM:

- **1 OMEGA MONOPURE CURCUMIN EC**
- **1 S-ACETYL GLUTATHIONE**
- 1 ACTIV NUTRIENTS WITHOUT COPPER AND IRON
- **1 ACTIV NUTRIENTS PERFORMANCE**
- **1 MONOPURE PRM**
- 1 ALAMAX CR
- PM:
- **1 MELATONIN CR**
- 1 NAC
- 1 K2-D3
- **1 HEMP MONOPURE**
- **1 OPTIMAG NEURO**
- **1 RESVERATIN PLUS**

* 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 itaboo Nor Reput Addit of Symptoms PERSIST! without permission of Trevor Berry, DC, DACNB is prohibited. Duplication, citation or distribution of this material in whole or in part

ACUTE PHASE TBI of Trevor Ber 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)

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 INFE LONG CTATION



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SUMMARY WF 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)
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- 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:

USE

- 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

BASIC THYROID LASER PROTOCOL COURTESY OF KIRK GAIR



Step 1: Loser 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 the patient starts to feel anxious or any unusual symptoms, stop the process, and note how long lasts. If you have a patient who is very ramped up immunologically or neurologically, they may not be able to top de very much stimulation initially, so use your clinical judgement as to what is appropriate for your patient.

Additional note: I STONGLY caution not to use a high powered laser, such as a class IV with these protocols, Mave been contacted by patients who received treatment with Class IV lasers over the thyraid who had it trigger a thyraid storm and an ER visit with serious flare ups of their symptoms. Additionally, I have not seen any studies that support that type of laser use in the manner described in this webinar!

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ERCHONIA

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, PREGENOLONE 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

ERECTILE DYSFUNCTION PROTOCOL



ERCHONIA

- THE CANARY IN THE COAL MINE BLOOD FLOW AND 02 DELIVERY COMPROMISE (CV DISEASE, DIABETES, ANEMIA ETC)
- CHOLESTEROL IS NEEDED! - PROCREATION VS SURVIVAL

CIRCULATION SETTING (DAN MURPHY) 20-42-43-53 ABOVE PUBIC BONE SHOCKWAVE THERAPY. IE THE PHOENIX

- 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 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 LLLT AND PROSTATE CANCER



Prostate FLO'

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 693

Jaber Zafari 1, Fatemeh Javani Jouni 2

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**

Duplication, Auto Duplication, Automatication, Automaticatio, Automaticatio,

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 Sun 1 2, Kidong Kim 1 2, Jae Hong No 1 2



ADRENAL SUPPORT

MedPax

edit name

Qty: 1

Qtv: 1

PM Packet

Cortisolv® Capsules

MinRex® Capsules

NRF2 ACTIVATOR

MONOPURE COQ10

ADRENAL

SUPPORT

PM

ト



ERCHONIA LLLT: FREQUENCIES 10-21-33-59 60 SECONDS EACH ADRENAL GLAND (LOWER RIB CAGE AREA) AFTER UPREGULATING T/L SPINE AREA FOR 20 SECONDS + 2 MIN VAGAL STIM

DON'T FORGET BRAIN 1-10-1-10

VIBRANT LAB FOCUS: SALIVA ASI AND DRIED URINE TEST



OTHER CLINICAL CONSIDERATIONS: MANAGE CHRONIC STRESSORS SUCH AS CHRONIC PATHOGENS, LIFESTYLE STRESS AND CHANGES SLEEP DISORDERS SUPPORT ENTIRE HP AXIS? PREGENOLONE STEAL/CHOLESTEROL/SEX HORMONES

DEPRESSION AND LLLT



Randomized Controlled Trial

AM Packet

Adrenaliv® Capsules

Cortisolv® Capsules

MinRex® Capsules

Bio C 1:1[™] Capsules

B ACTIV

ADRENAL

SUPPORT

MedPax

edit name

Qty: 2

Qtv: 1

Qty: 1

Qty: 1

AM

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 the intervention 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)

CLINICAL CONSIDERATIONS FOR REDUCING HISTAMINES 700 *1-6 HistDAO per day (use pre meal) Total Allergen Load 1-2 AllerDHQ daily combo Symptoms Infectious Stressors AllerDHO Nutritional Stressors XYMOGEN'EP **Emotional Stressors** 60 Vege Hormonal Stressors Chemical/Toxin Stressors HistDAO Food Allergens Inhalant Allergies

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 Duplication, citate without permit LOW HISTAMINE DIETT whole or in part is prohibited.

HISTAMINES



FOODS THAT PROMOTE

DAO PRODUCTION:

ORGAN MEATS

ESPECIALLY KIDNEY

GREEN PEA SPROUTS

FATS: OLIVE OIL

OMEGA 3'S

Histamine-rich foods are:

There are also a number of foods that trigger histamine release in the body, such as:

- alcohol and other fermented beverages
- fermented foods and dairy products, such as yogurt and sauerkraut
- dried fruits
- eggplant
- spinach
- processed or smoked meats
- <u>shellfish</u>
- aged cheese

Foods that block DAO production include:

- alcohol
- black tea
- mate tea
- green tea
- energy drinks

specifically walnuts, cashews and peanuts food dyes and other additives 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-B, 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-y 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 CONSIDER SINUSITIS SETTING: 20-60-160-400 **Allergic Rhinitis Mouse Model** WITH HANDHELDS. 2 MINUTES PFC

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- <u>alcohol</u>
- <u>bananas</u>
- tomatoes
- wheat germ
- beans
- <u>papaya</u>
- chocolate
- citrus fruits
- nuts.

ANXIETY TREATMENT IDEAS



Anxiousness is associated with decreased alpha waves, increased beta waves, and can be affected by low delta and

theta waves. Anxiousness and feelings of panic can be caused by more than fear and insecurities. They can be chemically driven states from an imbalanced and poorly regulated brain. It's a delicate balance where every wave plays their part.

ERCHONIA HAND HELD LASER "ANXIETY PROTOCOL" 1-10-1-10 PREFRONTAL CORTEX MINIMUM 5 MINUTES (UP TO 6 VIOLET/10 RED) PLUS VAGAL NERVE STIMULATION PROTOCOL 2 MINUTES SCM/2 MINUTES OVER THE GUT CONSIDER BIAS TO RIGHT PREFRONTAL CORTEX

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An analysis of data from five large-scale studies found somewhat lower white matter fractional anisotropy in certain regions of the brain in persons diagnosed with attention deficit hyperactivity disorder (ADHD). Fractional anisotropy is generally considered an indicator of connectivity in the brain. Similar microstructural anomalies were not present for anxiety, mood, or externalizing problems. The study was published in Biological Psychiatry.

analysis showed that both the level of ADHD traits and ADHD diagnosis were associated with "altered microstructure of the inferior longitudinal fasciculi (ILF) and the left uncinate fasciculus". The overall size of these differences was small. "White matter tract

microstructural anomalies were not as prominently associated with problems related to mood, anxiety or other externalizing problems," the study authors conclude.

* THE ILF IS MAINLY FOR SEMANTIC PROCESSING CONNECTING THE OCCIPITAL AND TEMPORAL LOBES. EX. VISUAL PROCESSING AND LANGUAGE COMPREHENSION. CONSIDER 1-10-1-10 TO PREFRONTAL CORTEX (AND CEREBELLUM) WHILE PRACTICING HAND WRITING. THE LEFT UNCINATE IS A FRONTAL - ANTERIOR TEMPORAL CONNECTION. A MAJOR FUNCTION IS RECOGNITION OF FAMILIAR/FAMOUS FACES. FLASH IMAGES OF KNOWN PEOPLE AND HAVE THE PATIENT CALL OUT THE NAME OF THE IMAGE WHILE LASERING PFC

A mega-analytic study of white matter microstructural differences across five cohorts of youth with attention deficit hyperactivity disorder

Duplication, citation or distributed by the state of the

Published:September 25p 2022DOI Inttos://doi.org/1081016/hbiopsych.2022.09.021

ADHD ADVANCED FN THERAPIES



- -OVERLAP SACCADES (VS GAP). FOCUS BUILDER APP
- -GO NO GO EXERCISES
- -ANTI-SACCADES
- -ISOMETRICS
- -LEFT SIDE INTERACTIVE METRONOME
- -DEEP BREATHING 4:4:8
- -MIRROR CURSIVE HANDWRITING
- -FACIAL RECOGNITION

-1111 LASER SETTING or 1-10-1-10 PFC (5+ MINUTES), CEREBELLUM (2MIN) + VAGAL STIM PROTOCOL (SCM/GUT 2 EACH)

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

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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)

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)



-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)

ERCHONIA

ERCHONIA

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"



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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**



FINALLY A SOLUTION FOR TOENAIL FUNGUS (ONYCHOMYCOSIS)

711

- · 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 ERCHONIA PATHOGENS: COMMON SETTINGS

- 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*

ornus ID: 74396187

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 UP⁷ VISITS

IMMUNE FUNCTION AND PATHOGENS

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 ocks 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 1/4/HSN/2/2/2010 herpes zaster have only virostatic effect





rent





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.

Schind TREATMENT PLAN: ACUTE EBV 10 TREATMENTS TOTAL (DAILY) Author in 12 MINUTES TOTAL ERCHONIA SETTING 28-73-274-728 Depart **6 MINUTES TRANSCRANIAL** Abstract 6 MINUTES RESPIRATOR SYSTEM AND GI Recurr CONTINUE TREATMENT APPLICATIONS ALL FOLLOW-UP VISITS introdu therap MONITOR BRAIN AUTOIMMUNE WITH VIBRANT NEURAL ZOOMER PLUS influen OTHER CONSIDERATIONS FOR ANTI-VIRAL SUPPORT: patient VIRAGRAPHIS, LAURICIDIN, BIOCIDIN, ARGENTYN SILVER randon 2 wk,

en 1 in pain he in 50

ons for 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, neginvasive 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

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(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

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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,*



NUTRAFOL

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)

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* 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

PARKINSON'S DISEASE AND LLLT



- > 24 hour water fast 1x/week (minimum every 2 weeks), FIX GUT/LEAKY GUT
- ADDRESS DOPAMINE NEUROCHEMISTRY/CO-FACTORS/MICRONUTRIENTS
- LLLT GI AND SCM vagal setting (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 Mastoid vestibular stimulation (4-8, 50-150) 5 minutes. CO-ACTIVATE
- LLLT MASTER BRAIN SETTING 1-10-40-60 (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).
- ▶ Initial treatment 3x/week 4 weeks, monitor progress and phase down.
- BIAS TREATMENTS TO FX 405 OR EVRL BUT STACK WITH GVL WHEN AVAILABLE


ERCHONIA

WHEN IN DOUBT, DEFAULT TO THE MASTER BRAIN PROTOCOL FOR ANY NEUROLOGICAL DISORDER INCLUDING DEMENTIAS LIKE ALZHEIMER'S, VASCULAR DEMENTIA ETC - "MASTER BRAIN SETTING" 1-10-40-60 HAND HELDS 1-40PE (1-40 EX 405 AND) 40-60 EOR ARICALS

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 3-3-3-2-2 XLR8, 2-2-2-2 GVL OR EVRL

MASTER BRAIN SETTING

BASIC BRAIN PROTOCOLS REVIEW FOR HANDS ON:

TRANSCRANIAL BASIC 101 SETTING SUGGESTIONS-

OLD FX 635 1-40-10 *10 MINUTES RED* SAGITTAL MOHAWK 1 FRONT 10 BRAIN STEM or TURN TO CROSS TEMPORAL AT 5 MINUTES (NEW FX 1-40 PFC, 40-60 APICAL, 10-10 BASE OF SKULL) 6 MINUTES VIOLET/RED FX 405 (3&3) (1-40 FOR APICAL DIODE)

XLR8 1-10-40-60: 3 MINUTES PFC, 3 MINUTES APICAL, 3 MINUTES STEM/BASE OF SKULL, 2 MINUTES SCM/VAGAL, 2 MINUTES STOMACH

EVRL OR GVL 1-10-40-60: 2 MINUTES PFC, 2 MINUTES APICAL, 2 MINUTES STEM/BASE OF SKULL, 2 MINUTES SCM VAGAL, 2 MINUTES STOMACH

MISCELLANEOUS SUPPORTING DOCUMENTS AND SLIDES

723



3 PRIMARY VARIABLES DEMONSTRATING NON-THERMAL LOW LEVEL LASER THERAPY SUPERIORITY VS OTHER LIGHT DEVICES



1.DOSE VS ENERGY 2.WAVELENGTH 3.FREQUENCIES

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Biomedicine. 2017 Dec;7(4):22. doi: 10.1051/bmdcn/2017070422. Epub 2017 Nov 13.

Only lasers can be used for low level laser therapy. Moskvin SV1

Abstract

The question of lasers' exclusivity, as well as <u>the degree of influence of special</u> <u>properties of low-intensity laser illumination (LILI), such as coherence, polarity and</u> <u>monochromaticity, on the effectiveness of low level laser therapy (LLLT)</u> continues to cause arguments. The study analyzes publications from 1973 to 2016, in which laser and conventional light sources are compared, and the following conclusions are drawn. First, there are a lot of publications with incorrect comparison or unfounded statements. Secondly, other sources of light are often meant by LILI without any justification. Thirdly, all studies, in which the comparison is carried out correctly and close parameters of the impact and the model are used, have a firm conclusion that laser light is much more effective. Fourthly, it is uniquely identified that the <u>most important parameter that determines</u> the efficiency of lasers is monochromaticity, i.e., a much narrower spectral width

than for all other light sources. Only laser light sources can be used for LLLT! 725

OPTIMAL LASER PHYSIOLOGY



<u>J Biomed Opt.</u> 2018 Dec;23(12):1-17. doi: 10.1117/1.JBO.23.12.120901.

Review of light parameters and photobiomodulation efficacy: dive into complexity. <u>Zein R1, Selting W1, Hamblin MR</u>2,3,4.

Author information Abstract

Photobiomodulation (PBM) therapy, previously known as low-level laser therapy, was discovered more than 50 years ago, yet there is still no agreement on the parameters and protocols for its clinical application. Some groups have recommended the use of a power density less than 100 mW/cm2 and an energy density of 4 to 10 J/cm2 at the level of the target tissue. Others recommend as much as 50 J/cm2 at the tissue surface. The wide range of parameters that can be applied (wavelength, energy, fluence, power, irradiance, pulse mode, treatment duration, and repetition) in some cases has led to contradictory results. In our review, we attempt to evaluate the range of effective and ineffective parameters in PBM. Studies in vitro with cultured cells or in vivo with different tissues were divided into those with higher numbers of mitochondria (muscle, brain, heart, nerve) or lower numbers of mitochondria (skin, tendon,

cartilage). Graphs were plotted of energy density against power density. Although the results showed a high degree of variability, cells/tissues with high numbers of mitochondria tended to respond to lower doses of light than those with lower number of mitochondria. Ineffective studies in cells with high mitochondrial activity appeared to be more often due to over-dosing than to under-dosing.

OPTIMAL LASER **PHYSIOLOGY**



Dose Response. 2011;9(4):602-18. doi: 10.2203/dose-response.11-009.Hamblin. Epub 2011 Sep 2.

Biphasic dose response in low level light therapy - an update.

Huang YY1, Sharma SK, Carroll J, Hamblin MR.

Author information

Abstract

Low-level laser (light) therapy (LLLT) has been known since 1967 but still remains controversial due to incomplete understanding of the basic mechanisms and the selection of inappropriate dosimetric parameters that led to negative studies. The biphasic dose-response or Arndt-Schulz curve in LLLT has been shown both in vitro studies and in animal experiments. This review will provide an update to our previous (Huang et al. 2009) coverage of this topic. In vitro mediators of LLLT such as adenosine triphosphate (ATP) and mitochondrial membrane potential show biphasic patterns, while others such as mitochondrial reactive oxygen species show a triphasic dose-response with two distinct peaks. The Janus nature of reactive oxygen species (ROS) that may act as a beneficial signaling molecule at low concentrations and a harmful cytotoxic agent at high concentrations, may partly explain the observed responses in vivo. Transcranial LLLT for traumatic brain injury (TBI) in mice shows a distinct biphasic pattern with peaks in beneficial neurological effects observed when the number of treatments is varied, and when the energy density of an individual treatment is varied. Further understanding of the extent to which biphasic dose responses apply in LLLT will be necessary to optimize clinical treatments.

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OPTIMAL LASER PHYSIOLOGY: LED VS LASER



lasers Med Sci

2016 Dec;31(9):1957-1970. doi: 10.1007/s10103-016-1977-9. Epub 2016 Jun 7.

Low-level phototherapy to improve exercise capacity and muscle performance: a systematic review and meta-analysis

Abstract

LED vs LLLT

The aim of this study was to evaluate the effectiveness of pre-exercise low-level phototherapy (Light-Emitting Diode therapy [LEDtherapy] or Light Amplification by Stimulate Emission of Radiation therapy [LASERtherapy]) in increasing exercise capacity and muscle performance of people undergoing exercise when compared to placebo treatment. Randomized controlled trials and crossover studies were sought on CENTRAL, MEDLINE, EMBASE, SciELO, PEDro and LILACS from its inception up to February 2015. References lists of included studies were sought for additional relevant research. Two authors independently extracted data on study design, treatment parameters, exercise capacity (number of repetitions, time to exhaustion, blood lactate concentration and lactate dehydrogenase activity) and muscle performance (torque, power and strength) using an structured table. Agreement should be reached by consensus or by a third reviewer. Sixteen studies involving 297 participants were included.

Improvement of number of repetitions (mean difference [MD] [95 % confidence interval] = 3.51 repetitions [0.65-6.37]; P = 0.02), delay in time to exhaustion (MD = 4.01 s [2.10-5.91]; P < 0.0001), reduction in lactate levels (MD = 0.34 mmol/L [0.19-0.48]; P < 0.00001) and increased peak torque (MD = 21.51 Nm [10.01-33.01]; P < 0.00001) were observed when LASERtherapy was applied. *LEDtherapy meta-analyses were performed* with two studies and retrieved no between-group statistically significant difference in power, differenc

LED LIGHTS AND SKULL PENETRATION



Can Infrared Light Reach the Brain?

Can 0.5-W LEDs penetrate human scalp and skull to reach the brain? The answer is "No." My colleague, Larry Morries, DC, and I showed that these LEDs did not even penetrate 2 mm of human skin.

The human scalp and skull provide a significant barrier. Infrared light energy needs to be in the range of 0.9 to 15 J/cm2 at the target tissue to activate mitochondria and other cellular events.2-3,8-9 Even if a 0.5-W LED only had to penetrate the skull to reach the surface of the brain, it could only deliver 0.0064 J/cm2, or 1/140th of the minimum energy necessary to induce PBM.₁₀ No energy would be expected to reach the depths of the brain needed to treat stroke, Parkinson disease, Alzheimer disease, or

many brain injuries. Although more than 40% of the incident light from a light source may penetrate mouse skull, only 4.2% penetrates human skull.8,10

2. Henderson TA, Morries, LD. <u>Near-infrared photonic energy penetration: can infrared phototherapy effectively reach the human brain?</u> *Neuropsychiatr Dis Treat*. 2015;11:2191-2208.⁷²⁹

LED LIGHTS AND SKULL PENETRATION



There is a hairier problem facing LED devices: human hair blocks infrared light. More than 98% of infrared light can be blocked by 2 mm of hair (ie, 9.764 W of a 10-W beam of 810 nm infrared light is absorbed by human hair).11 *If 98% of the energy from a 0.5-W LED is absorbed by hair, 80% to 90% is absorbed by 2 mm of skin, and 96% of incident energy is attenuated by skull, then claims of neurophysiological benefits of LED-based devices become highly questionable.*

8. Ando T, Xuan W, Xu T, et al. <u>Comparison of therapeutic effects between pulsed and continuous wave 810-nm wavelength</u> laser irradiation for traumatic brain injury in mice. *PLoS One*. 2011;6(10):e26212.

9. Yip KK, Lo SC, Leung MC, So SK, Tang CY, Poon DM. <u>The effect of low-energy laser irradiation on apoptotic factors</u> following experimentally induced transient cerebral ischemia. *Neuroscience*. 2011;190:301-306.

10. Lapchak PA, Boitano PD, Butte PV, et al. <u>Transcranial near-infrared laser transmission (NILT) profiles (800 nm): systematic</u> comparison in four common research species. *PLoS One*. 2015;3;10(6):e0127580.

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-LED LIGHTS ARE SCATTERED PHOTONS (LIKE SHINING A RED LASER POINTER ON SOMEONE)

- -THEY ARE WHAT ERCHONIA USES AS THE PLACEBO "SHAM" DURING FOR THEIR DOUBLE BLIND PLACEBO CONTROLLED FDA CLEARANCES
- -DURING THE ERCHONIA AUTISM STUDY LED'S HAD ZERO EFFECT ON PATIENT OUTCOMES ON HEAD EXPOSURE
- -FOR FAT LOSS, ERCHONIA'S TRUE RED LASER WAS 6X MORE EFFECTIVE THAN LED'S
- -FOR PAIN STUDY 1 YEAR FOLLOW UPS, ERCHONIA'S LASER TREATMENT GROUP REMAINED IMPROVED WHERE AS LED FOLLOW UPS RETURN BACK TO OR CLOSE TO BASELINE WHERE THEY STARTED

-OF ALMOST 400 COMPARATIVE STUDIES, ONLY 3 SHOWED LED HAD A BENEFICIAL EFFECT, A TOOTH SENSITIVITY STUDY, A STUDY ON BEES AND A POST CARDIAC SURGICAL WOUND PAIN STUDY

- READ TUNER AND HODE: LASER PHOTOTHERAPY REGARDING LASER PHYSIOLOGY, TRUE LASER VS. LED AND BIPHASIC DOSE

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LOW LEVEL LASER RESEARCH HISTORY



Abstract

In Russia (formerly USSR) study of biomodulation action (BMA) mechanisms of low-intensity laser irradiation (LILI) began in 1964, immediately after the development of lasers. During the period from 1965 to 1972 several dozens of scientific conferences were held, hundreds of studies were published. Generally, secondary mechanisms and results of LILI effect on patients with various diseases were studied. This data was immediately implemented into practical medicine in the fields of oncology, surgery, dermatology and dentistry, and since 1974 low level laser therapy (LLLT) is included in the standard of state medical care. For 50 years no less than 1000 books were published (monographs, collections, methodical and clinical materials), thousands of researches were carried out. Primary mechanism and patterns of interaction of LILI with acceptors within cells can be represented in the following order: absorption of photon's energy - emergence of a local temperature gradient - release of Ca₂₊ from intracellular stores - stimulating Ca₂₊-dependent processes. Understanding of this process allowed the explanation of all known secondary effects, optimized methods and extremely increased effectiveness of LLLT. Owing to the knowledge of BMA mechanisms of LILI, numerous associated and combined LLLT techniques were developed and are widely used nowadays: locally, on the projection of internal organs, laser acupuncture, reflexology, intracavitary, transdermal and intravenous laser blood

illumination, magnetic-laser therapy, laser phoresis, laser-vacuum massage, biomodulation, etc. About 400 000 laser therapeutic devices are used in Russian practical healthcare.

HIGHLIGHTS: -RED 635 NM -PULSED -TRUE LASER ONLY -<u>DO NOT USE CLUSTER BEAMS OF LED AND LASER.</u> SCATTERED LED PHOTONS BOUNCE OFF THE TRUE LASER BEAMS CAUSING THEM TO BE SCATTERED AS WELL!

J Lasers Med Sci

2017 Spring;8(2):56-65. doi: 10.15171/jlms.2017.11. Epub 2017 Mar 28.

Low-Level Laser Therapy in Russia: History, Science and Practice

Sergey Vladimirovich Moskvin 1

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HELPING THOSE ABROAD





Sometimes a situation comes along as a business and as human beings, and i t's nice to be able to give something back.

We have donated 2 of our Nonthermal lasers to Dr Serhii Danylkov pictured here with his wife, Tatiana, at a Dr Trevor Berry non-thermal laser masterclass in Hilversum, Netherlands yesterday. Dr Danylkov is a surgeon by trade and due to what is happening in his Ukranian homeland, he is splitting his time between his family in Poland and his home city of Kyiv. He travels back and forwards 14 hours each way by bus to Kyiv twice per month in order to provide his skilled services to treat wounded Ukrainian soldiers and wounded members of the general public who have been injured in this awful war.

He saw our nonthermal lasers being used in the <u>HOLIS-MED</u> clinic in Torun, Poland, that Tatiana is working at and saw their impact on pain management and accelerated healing so reached out to us to see if we could help. We are all hoping for a resolution to this unnecessary and barbaric site ation, and it's down to the bravery of the Ukranian people and the likes of Dr Danvlkov which gives us all hope !!

DEPTH OF PENETRATION White light (all colors) Colors separated for visualization Wavelength 400 300 500 600 700 800 (nm)Depth (m)100 200 300

© 2005 Brooks/Cole - Thomson

THE DEPTH OF PENETRATION FALLACY IS ONE OF THE MOST COMMON MISTAKES IN LASER PHYSIOLOGY AND **COMPANY RHETORIC. THERE IS AN OUTDATED CADAVER STUDY CLAIMING THAT** LONGER WAVELENGTHS HAD **GREATER DEPTH OF PENETRATION THAN** SHORTER WAVELENGTHS. UNLESS YOU ARE TREATING **DEAD PEOPLE WITHOUT WATER (65-95% OF OUR TISSUES AND CELLS) AND OTHER ORGANIC MATERIAL** (LET ALONE LIFE ENERGY -**QUANTUM TRANSPORT -**SIERPINSKI TRIANGLE) THIS **IS NOT ACCURATE IN LIVING** TISSUE AND HUMANS OR **OTHER ANIMAL STUDIES!**

ERCHONIA

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DEPTH OF PENETRATION MYTH



<u>J Biophotonics.</u> 2018 Jul 25:e201800173. doi: 10.1002/jbio.201800173. [Epub ahead of print]

Wang P1,2, Li T1,2.

Abstract

One of the challenges in transcranial low-level laser therapy (LLLT) is to optimally choose illumination parameters, such as wavelength. However, there is sparse study on the wavelengths comparison especially on human transcranial LLLT. Here, we employed Monte Carlo modeling and visible human phantom to compute the penetrated photon fluence distribution within cerebral cortex. **By comparing the**

fluence distribution, penetration depth and the intensity of laser-tissueinteraction within brain among all candidate wavelengths, we found that 660, 810 nm performed much better than 980, 1064 nm with much stronger, deeper and wider photon penetration into cerebral tissue; 660 nm was shown

to be the best and slightly better than 810 nm. Our computational finding was in a surprising accordance with previous LLLT-neurobehavioral studies on mice. This study not only offered quantitative comparison among wavelengths in the effect of LLLT light penetration effectiveness but also anticipated a delightful possibility of online, precise and visible optimization of LLLT illumination parameters.

Transcranial red and near infrared light transmission in a cadaveric model. Jagdeo JR, Adams LE, Brody NI, Siegel DM.

PLoS One. 2012;7(10)

This study demonstrates penetration of intact cadaver soft tissue, skull bone and brain tissue using both 633 nm and 830 nm wavelengths with the shorter wavelength having more "scatter"

BIPHASIC DOSE RESPONSE AND INFLAMMATION



Lasers in Medical Science (2023) 38:66 https://doi.org/10.1007/s10103-022-03664-3

REVIEW ARTICLE

Biphasic dose response in the anti-inflammation experiment of PBM

Fang Nie¹ · Shaolong Hao^{1,3} · Yu Ji^{1,3} · Yan Zhang¹ · Hao Sun¹ · Melissa Will² · Wei Han^{1,3} · YuChuan Ding²

Received: 10 December 2021 / Accepted: 22 October 2022 © The Author(s), under exclusive licence to Springer-Verlag London Ltd., part of Springer Nature 2023

Abstract

Non-invasive laser irradiation can induce photobiomodulation (PBM) effects in cells and tissues, which can help reduce inflammation and pain in several clinical scenarios. The purpose of this study is to review the current literature to verify whether PBM can produce dose effects in anti-inflammatory experiments by summarizing the clinical and experimental effects of different laser parameters of several diseases. The so-called Andt-Schulz curve is often used to describe two-phase dose reactions, assuming small doses of therapeutic stimulation, medium doses of inhibition, and large doses of killing. In the past decade, more and more attention has been paid to the clinical application of PBM, especially in the field of anti-inflammation, because it represents a non-invasive strategy with few contraindications. Although here are different types of lasers available, their use is adjusted by different parameters. In general, the parameters involved are wavelength, energy density, power output, and radiation time. However, due to the biphasic effect, the scientific and medical communities remain puzzled by the ways in which the application of PBM must be modified depending on its clinical application. This article will discuss these parameter adjustments and will then also briefly introduce two controversial theories of the molecular and cellular mechanisms of PBM. A better understanding of the extent of dualistic dose response in low-intensity laser therapy is necessary to optimize clinical treatment. It also allows us to explore the most dependable mechanism for PBM use and, ultimately, standardize treatment for patients with various diseases.

 $\textbf{Keywords} \ \ Photobiomodulation \cdot Inflammation \cdot Low-energy \ laser \ therapy \cdot Biphasic \ dose \ response \cdot Arndt-Schulz \ curve$

BIPHASIC DOSE RESPONSE AND INFLAMMATION



Lasers in Medical Science (2023) 38:66 https://doi.org/10.1007/s10103-022-03664-3 REVIEW ARTICLE

Biphasic dose response in the anti-infammation experiment of PBM

Fang Nie1 · Shaolong Hao1,3

REVIEW ARTICLE HIGHLIGHTS FOR OPTIMAL PARAMETERS:

-OPTIMAL POWER SUGGESTED RANGE 1 - 500 mW (0.001 - .5 WATTS)

-OPTIMAL IRRADIANCE 10 mW/CM2

OTHER EXAMPLES OF PAPERS CITED INT HE REVIEW:

-IRRADIATION TIME OF 128 SEC AND 4 J/CM2 INHIBITED INFLAMMATION WHEREAS 640 SEC AND 20 J/CM2 HAD THE OPPOSITE EFFECT

-HEAT SHOCK PROTEIN HSP70, PRO-INFLAMMATORY IL-6, TNF-ALPHA AND APOPTOSIS FACTOR CASPASE-3 ON SPINAL CORD INJURY PBM SHOWED UP TO 300 mW WAS SAFE, 500 mW CAUSED "A LITTLE DAMAGE TO THE RADIATION SITE" AND 1000 mW (1W) LED TO SIGNIFICANT EXPRESSION OF INJURY INFLAMMATION MARKERS LISTED

-50mW LLLT WAS MORE EFFICIENT THAN 100 mW IN MODULATING IL-1B AND IL-6, MACROPHAGES AND NEUTROPHILS (2 REFERENCES)

-IDEAL OPTICAL WINDOW OF WAVELENGTH BETWEEN 600 AND 1100 NM



LIGHT THERAPIES AND CANCER



AS WE INCREASE USE OF LLLT TO THE HEAD, FACE AND UPPER BODY, HOW SAFE IS LASER WITH THE RISK OF CANCER CELLS PRESENT?



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Lasers Med Sci. 2017 Jan;32(1):229-242. doi: 10.1007/s10103-016-2056-y. Epub 2016 Aug 19. The use of low-level light therapy in supportive care for patients with breast cancer: review of the literature.

<u>Robijns J</u>1,2, <u>Censabella S</u>3, <u>Bulens P</u>4,3, <u>Maes A</u>4,3, <u>Mebis J</u>5,4,3. Author information

Abstract

Breast cancer is the most common cancer in women worldwide, with an incidence of 1.7 million in 2012. Breast cancer and its treatments can bring along serious side effects such as fatigue, skin toxicity, lymphedema, pain, nausea, etc. These can substantially affect the patients' quality of life. Therefore, supportive care for breast cancer patients is an essential mainstay in the treatment. Low-level light therapy (LLLT) also named photobiomodulation therapy (PBMT) has proven its efficiency in general medicine for already more than 40 years. It is a noninvasive treatment option used to stimulate wound healing and reduce inflammation, edema, and pain. LLLT is used in different medical settings ranging from dermatology, physiotherapy, and neurology to dentistry. Since the last twenty years, LLLT is becoming a new treatment modality in supportive care for breast cancer. For this review, all existing literature concerning the use of LLLT for breast cancer was used to provide evidence in the following domains: oral mucositis (OM), radiodermatitis (RD), lymphedema, chemotherapy-induced peripheral neuropathy (CIPN), and osteonecrosis of the jaw (ONJ). The findings of this review suggest that LLLT is a promising option for the management of breast cancer treatment-related side effects. However, it still remains important to define appropriate treatment and irradiation parameters for each condition in order to ensure the effectiveness of LLLT.

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LIGHT THERAPIES AND CANCER



Table 3

Different Complications Resulted From Head and Neck Cancer Treated by Photobiomodulation

Head & Neck Cancer Complications	Photobiomodulation Therapy	Wavelengths	Power Supply	Efficiency
Oral mucositis	IR & NIR & LLLT	630-830 nm	10-150 mW	Positive
Dermatitis	IR LED Red & IR LED	630-680 nm 630-680 nm	20-150 mW 20-80 mW	Positive Positive
Dysphagia	IR-NIR	980nm	-	Positive
Osteoradio necrosis	Red & IR	660-800 nm	100 mW	Positive
Trismus	Diode Laser Ga-Al-As	980 nm 830 nm	30 mW 30 mW	Positive Negative
Head & neck lymphedema	LLLT LLLT	650-1000 nm 808-905 nm	20-80 mW 20-80 mW	Positive Positive

<u>J Lasers Med Sci.</u> 2020 Autumn; 11(4): 369–380.

Published online 2020 Oct 3. doi: 10.34172/jlms.2020.60

PMCID: PMC7736953 PMID: <u>33425286</u>



Lasers Med Sci. 2016 Dec;31(9):1775-1782. Epub 2016 Aug 12.

The effects of low-level laser irradiation on breast tumor in mice and the expression of Let-7a, miR-155, miR-21, miR125, and miR376b.

Khori V1, Alizadeh AM2, Gheisary Z3, Farsinejad S3, Najafi F4, Khalighfard S3, Ghafari F3, Hadji M3, Khodayari H3.

Abstract

Low-level laser therapy (LLLT) is a form of photon therapy which can be a non-invasive therapeutic procedure in cancer therapy using low-intensity light in the range of 450-800 nm. One of the main functional features of laser therapy is the photobiostimulation effects of low-level lasers on various biological systems including altering DNA synthesis and modifying gene expression, and stopping cellular proliferation. This study investigated the effects of LLLT on mice mammary tumor and the expression of Let-7a, miR155, miR21, miR125, and miR376b in the plasma and tumor samples. Sixteen mice were equally divided into four groups including control, and blue (VIOLET), green, and red lasers at wavelengths of 405, 532, and 632 nm, respectively. Weber Medical Applied Laser irradiation was carried out with a low power of 1-3 mW and a series of 10 treatments at three times a week after tumor establishment. Tumor volume was weekly measured by a digital vernier caliper, and gRT-PCR assays were performed to accomplish the study. Depending on the number of groups and the p value of the Kolmogorov-Smirnov test of normality, at test, a one-way ANOVA, or a non-parametric test was used for data analyses, and p < 0.05 was considered to be statistically significant. The average tumor volume was significantly less in the treated blue (VIOLET 405) group than the control group on at days 21, 28, and 35 after cancerous cell injection. Our data also showed an increase of Let-7a and miR125a expression and a decrease of miR155. miR21, and miR376b expression after LLLT with the blue laser both the plasma and tumor samples compared to other groups. It seems that the non-invasive nature of laser biostimulation can make LLLT an attractive alternative therapeutic tool.

LIGHT THERAPIES AND CANCER



Support Care Cancer. 2016 Jun;24(6):2497-501. doi: 10.1007/s00520-015-3051-8. Epub 2015 Dec 16. Effects of pre-radiation exposure to LLLT of normal and malignant cells.

Barasch A1,2, Raber-Durlacher J3, Epstein JB4,5, Carroll J6.

Author information Abstract PURPOSE:

Low-level laser therapy (LLLT) efficacy for the prevention of cancer treatment-induced oral mucositis (OM) has been amply described. However, potential protection of malignant cells remains a legitimate concern for clinicians. We tested LLLT-induced protection from ionizing radiation killing in both malignant and normal cells.

METHODS:

We treated six groups each of normal human *lymphoblasts (TK6) and human leukemia cells (HL60)* with He-Ne LLLT (632.8 nm, 35 mW, CW, 1 cm(2), 35 mW/cm(2) for 3-343 s, 0.1-12 J/cm(2)) prior to exposure to ionizing radiation (IR). Cells were then incubated and counted daily to determine their survival. Optimization of IR dose and incubation time was established prior to testing the effect of LLLT. RESULTS:

Growth curves for both cell lines showed significant declines after exposure to 50-200 cGy IR when

compared to controls. **Pre-radiation exposure to LLLT (4.0 J/cm(2)) followed by 1-h** incubation blocked this decline in TK6 but not in HL60 cells. The latter cells were sensitized to the killing effects of IR in a dose-dependent manner. CONCLUSION:

This study shows that pre-IR LLLT treatment results in a differential response of normal vs. malignant cells, suggesting that LLLT does not confer protection and may even sensitize cancer cells to IR killing.



Lasers Med Sci. 2018 Aug 24. doi: 10.1007/s10103-018-2563-0. [Epub ahead of print] Photo-modulation of zinc phthalocyanine-treated breast cancer cell line ZR-75-1 inhibited the normal tumor activity in vitro.

Zamani ARN1, Mashayekhi MR2, Jadid MFS1, Faridvand Y3, Tajalli H4, Rahbarghazi R5,6,7. Author information

Abstract

Regarding post-complication of convenient therapies against breast cancer, the emergence of effective approaches is essential. Photodynamic therapy is touted as a novel invasive therapeutic approach by the application of a photosensitizer promoted by laser irradiation. This study aimed to investigate the combined regime of low-level laser irradiation with zinc phthalocyanine in human breast cancer ZR-75-1 cell line. Cells were treated with 0.01 and 5 μ g/ml of ZnPc for 24 h and exposed to radiation (**70 mW**) for 60 s. Cell viability was evaluated by MTT and flow cytometry. Cell migration capacity was monitored by scratch test, Transwell migration insert, and gelatin zymography. The function of MDR in treated cells was examined by Rhodamine 123 exclusion test. The level of GALNT11 was measured by ELISA. The expression of Bax and Bcl-2 genes was evaluated by real-time PCR. Laser irradiation and zinc phthalocyanine induced cell cytotoxicity in a dose-dependent manner. Flow cytometry analysis showed the induction of apoptotic and necrotic changes in treated cells. We found a reduction in migration rate and MMP-9 activity in cells undergoing the experimental procedure (p < 0.05). Immunofluorescence imaging revealed the intracellular accumulation of Rhodamine 123 coincided with a reduction in the level of GALNT11 in treated cells,

showing the reduction of MDR activity and tumor cell resistance. Similar to flow cytometry

assay, the reduction of Bcl-2 (approximately twofold) and upregulation of Bax genes were found in treated cells. Photodynamic therapy could be as an effective and alternative method for the treatment of breast cancer in a human.

LIGHT THERAPIES AND CANCER



Cell Prolif. 2017 Nov 20. doi: 10.1111/cpr.12417. [Epub ahead of print]

Low level laser therapy induces increased viability and proliferation in isolated cancer cells.

Kara C1, Selamet H1, Gökmenoğlu C1, Kara N2.

Abstract

OBJECTIVES:

Low level laser therapy (LLLT), which stimulates natural biological processes in the application region, is frequently used in dental treatments. The aim of our study was to evaluate the effects of LLLT which could activate precancerous cells or increase existing cancerous tissue in case of clinically undetectable situations.

MATERIALS AND METHODS:

Saos-2 osteoblast-like osteosarcoma cells and A549 human lung carcinoma cells were used. Twenty-four hours after preparation of cell culture plates, laser irradiation was performed 1, 2 and 3 times according to the test groups using

Nd:YAG laser with the power output 0.5, 1, 2 and 3 W. Cell

proliferation analysis was performed by MTT assay at the 24th hour following the last laser applications. **RESULTS:**

Generally, it was observed that the proliferation rates increased as the number of applications increased, when compared to the controls, especially in those cases in which the irradiation was performed 2 or 3 times more. **CONCLUSION:**

The findings of this study have led to the conclusion that LLLT increases cancer cell proliferation, depending on the power output level of the laser and the number of applications. In addition to the proliferation and mitotic

activity of the cancer tissue cells, we concluded that LLLT, which is frequently used in dental practice, could activate precancerous cells or increase existing cancerous tissue.

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748

Lasers Med Sci. 2018 Apr 5. doi: 10.1007/s10103-018-2461-5. [Epub ahead of print] High doses of laser phototherapy can increase proliferation in melanoma stromal connective tissue.

Frigo L1, Cordeiro JM1, Favero GM2, Maria DA3, Leal-Junior ECP4, Joensen J5, Bjordal JM5,6, Roxo DC1, Marcos RL7,8, Lopes-Martins RAB9

It is well established that laser phototherapy (LP) is contraindicated directly over cancer cells, due to its bio modulatory effects in cell and blood vessel proliferation. The aim of the present study was to analyze the influence of typical low-level laser therapy (LLLT) and high intensity laser therapy (HILT) and an in-between dose of 9 J on collagen fibers and blood vessels content in melanoma tumors (B16F10) implanted in mice. Melanoma tumor cells

were injected in male Balb C mice which were distributed in four groups: control (no irradiated) or irradiated by 3_{μ}

9, or 21 J (150; 450, or 1050 J/cm2). LP was performed in daily sessions for 3 days with a InGaAIP-660 nm (mean output: 50 mW, spot size: 2 mm2). Tumor volume was analyzed using (1) picrosirius staining to quantify collagen fibers content and (2) Verhoeff's method to quantify blood vessels content. Tumor growth outcome measured in the 3-J group was not significantly different from controls. Nine and 21-J groups, presented significant and dose-dependent increases in tumor volume. Quantitative analysis of the intensity of collagen fibers and their organization in stroma and peri-tumoral microenvironment showed significant differences between irradiated

and control group. Blood vessels count of 21-J group outnumbered the other groups. High doses (29 J)

of LP showed a dose-dependent tumor growth, different collagen fibers characteristics, and eventually blood vessel growth, while a typical LLLT dose (3 J) appeared harmless on melanoma cell activity. EVEN A 1 WATT LASER AT 0.1 CM BEAM (SOME GO ABOVE 15) AT 1 MINUTE WILL GENERATE APPROXIMATELY 600 J/CM2 TO THE SURFACE TISSUE!!

Page 1 of 1

510(k) Number (if known): __K123474_____ Device Name: AVANT LASER MODEL LZ30 and LZ30-X

Indications for Use:

The Avant Laser Model LZ30 and LZ30-X are intended to emit energy in the visible and near infrared spectrum to provide topical heating for the purpose of elevating tissue temperature for a temporary relief of minor muscle and joint pain and stiffness, minor arthritis pain, or muscle spasm; the temporary increase in local blood circulation; and the temporary relaxation of muscles.

ALL LASERS ABOVE 500 mW ARE CONSIDERED TOPICAL HEATING LAMPS BY THE FDA WITH THE DESIGNATION OF "ILY" AND ARE "HIGH INTENSITY"

ONLY LASERS UNDER 500 mW CAN BE CLASSIFIED AS LOW LEVEL LASER

Prescription Use x (Part 21 CFR 801 Subpart D) Over-The-Counter Use _____ (Part 21 CFR 807 Subpart C)

(PLEASE DO NOT WRITE BELOW THIS LINE -CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Neil R Ogden 2013.12:02:14:15:37 -05'00

(Division Sign-Off) for BSA

Division of Surgical Devices

510(k) Number Duplication, citation or distribution of this material in whole or in part **K123***4*/4 without permission of Trevor Berry, DC, DACNB is prohibited.



international Journal of Pharmacology 7 (2): 149-160, 2011 ISSN 1811-7775 / DOI 10.3923/ijp.2011.149.160 © 2011 Asian Network for Scientific Information

An Overview of Laser Principle, Laser-Tissue Interaction **Mechanisms and Laser Safety Precautions for Medical** Laser Users

³Mohammed M. Jawad, '.³Sarah T Abdul Qader, ⁴.⁵AA Zaidan, ⁴.⁵B.B. Zaidan, 6AW, Naji and 1Ibraheem T Abdul Oader

laser to raise skin temp to 40 C, starts to destroy proteins, DNA, RNA, and causes necrosis. (Every Class 4 laser is FDA cleared to raise skin temperature to 40-45 degrees C).

TO GET ILY FDA CLEARANCE EXEMPTION FOR THEIR **510K CLEARANCE CLASS 4 LASERS HAVE TO SHOW** THEIR DEVICE RAISES SKIN TEMPERATURE TO THIS LEV[€]EL!

LIGHT THERAPIES AND CANCER



ME Darvin et al. on of Free Radicals in Human Skin

Formation of Free Radicals in Human Skin during Irradiation with Infrared Light

many, Infrare ² for 30 min

Heating to about 45°C, free radical formation was almost doubled, and antioxidant power was reduced to about 50%.

HEATING ABOVE 43 DEGREES CELSIUS CAN ACTUALLY FACILITATE PAIN SENSITIZATION! TRP cation channel subfamily V member 1 (TRPV1) receptors open at temperatures

greater than 43 °C 12. Tominaga M. The Role of TRP Channels in Thermosensation. In: Liedtke W.B., Heller S., editors. TRP Ion Channel Function in Sensory Transduction and Cellular Signaling Cascades. CRC Press; Boca Raton, FL, USA: 2007. [PubMed] [Googl

em against the destruc-free radicals, consisting mins, carotenoids, and and Sies, 2003; Sander Darvin et al., 2006). 70% of carotenoids in e β-carotene and lyco-al., 2000), which can er for the where artice leads to the

the Charité. The study adhered to the Declaration of Helsinki Principles. Even the initial values show a broad

The average for the magnitude of des-truction (changes relative to the initial values) in all volunteers was determivalues) in all volunteers was determined to be 30 ±12% for β -content and 37±10% for hycopene. These results are in agreement with previous investigations (Darvin et al., 2007). An increase in sin temperature was observed during irradiation, from \$1.6±1.0^{-C}. For the direct measurement of IR. For the direct measurement of the induced for a walk-to be a sector be direct measurement of the sector bedieved for a walk-to be a sector bedieved for a sector be

mission of Trevor Berry, DC, DACNB

(PCA) (Sigma, Steinh The radical-formation vestigated on six pig e The PCA marker was a

	510(K) PREMARKIERation, citatioSECTIONID4tiSTATEMENTAGEI in whole or in part SUBMISSIONWIthout permission Motor Provider or DiseDACNB is Disting to 1	751
	K120604	
	INDICATIONS FOR USE	
*NOTE: AS	5 OF MAY 2019, DEVICES WITH SUBSTANTIAL EQUIVALENCE TO TOPICAL HE	ATING FOR
	RY RELIEF OF MINOR PAIN NO LONGER NEED 510K CLEARANCE. EVERY LIG F AN LINSLIBSTANTIATED CLAIM THAT THEIR DEVICE WORKS ON PAIN ONE	MORE
REASON TO	O FOLLOW THE CONDITION SPECIFIC FDA CLEARANCE AND THE INDEXED L Indications for Use:	ITERATURE!!!
	AUNIT SECTION 4: SEAT	
	K-laser Cube 1,2,3, and 4 device is indicated for emitting energy in the Infrared Spectrum	
0	to provide topical heating for the purpose of elevating tissue temperature for temporary	
	relief of minor muscle and joint pain, muscle spasm, pain and stiffness associated with	5
	arthritis and promoting relaxation of the muscle tissue and to temporarily increase local	
	blood circulation.	
	VS ERCHONIA 510(K) DE NOVO (NEW DEVICE) FOR ACTUAL LOW LEVEL LASER CLEARANCE	
	Prescription Use X Over-The-Counter Use (Part 21 CFR 801 Subpart D) AND/OR (21 CFR 801 Subpart C)	
	(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE OF NEEDED)	
	the second s	

To: Seiple, Robert <Robert.Seiple@ul.com>

Cc: K181168@docs.fda.gov; Pilli, Minnie <Minnie.Pilli@ul.com> Subject: RE: K181168: K-Laser Platinum Series

You can go ahead and market the K-Laser Platinum Series devices submitted under K181168 without a letter. Please note that this does not automatically mean that you are

free to market future devices without 510(k) clearance. *I recommend that*

you read and understand 21 CFR 890.9 on the limitations of exemption before marketing another infrared lamp for medical purposes.

Additionally, please note that the K-Laser Platinum Series devices are NOT EXEMPT from Registration and Listing Requirements (21 CFR Part 807), Labeling Requirements (21 CFR Part 801 and 21 CFR 809.10), Good Manufacturing Practice Requirements set in the Quality System Regulation (21 CFR Part 820); and Medical Device Reporting Requirements (21 CFR Part 803).

> Amber Ballard, PhD Biomedical Engineer | Lead Reviewer

Physical Medicine and Rehabilitation Devices Branch (PMDB) Division of Neurological and Physical Medicine Devices (DNPMD) Office of Device Evaluation (ODE) Center for Devices and Radiological Health (CDRH)

U.S. Food and Drug Administration (FDA)

Tel: (240) 402-9983

Email: amber.ballard@fda.hhs.gov

Excellent customer service is moder tand to be a provide without permission of Trevor Berry, DC, DACNB is prohibited.



Eltech K-Laser wins the legal battle against Summus and Dr. Richard Albright Dearest Customers,

we are writing this comunication to address the confusion that has currently arisen from the declaration of SUMMUS MEDICAL LASER LLC, a former distributor of Eltech K-Laser S.r.l.

Nor SUMMUS MEDICAL LASER LLC nor Dr. Richard Albright have ever been the inventors, manufacturers or legitimate owners of K-Laser products and brands.

SUMMUS MEDICAL LASER LLC at the beginning was known as Laser Therapy LLC, founded by Dr. Richard Albright, and then later it changed its name in K-Laser USA LLC, as an exclusive distributor, for USA and Canada, of Eltech K-Laser S.r.l. for the K-Laser products made in Italy.

Eltech K-Laser S.r.I. is the inventor and owner of the K-Laser brands, and the manufacturer of the K-Laser devices, all made exclusively in Italy.

The distribution agreement with SUMMUS, at the time K-Laser USA LLC, was terminated by Eltech K-Laser S.r.l. in February 2018 when we found out that K-Laser USA LLC, in violation of the agreement, had affixed the K-Laser mark to a new Made in China product not manufactured by Eltech K-Laser S.r.l., more precisely on the laser device called "Platinum", stating that it was a new K-Laser device model of higher quality.

After the termination of the distribution agreement, a great confusion arose the same k-Laser USA LLC and Dr. Richard Albright refused to stop using the K-Laser marks in their business and continuing selling the Platinum as a K-Laser product.

LIGHT THERAPIES, OPTIMAL DOSE AND CANCER



Front Oncol. 2020; 10: 1255.

Published online 2020 Jul 24. doi: 10.3389/fonc.2020.01255

Review on the Cellular Mechanisms of Low-Level Laser Therapy Use in Oncology

-The influence of energy density in LLLT-induced mitochondrial changes are very similar to the findings with cellular proliferation and favors a lower level of energy density. When the intracellular calcium profiles were compared between 3.6 and 12 J/cm2; 3.6 J/cm2 induced transient increase of intracellular calcium without any cell damage whereas 12 J/cm2 induced a linear increase of intracellular calcium and damage in cardiomyocytes (27).

- Zungu et al. (28) studied two different energy densities (632.8 nm, 5 and 16 J/cm₂) and demonstrated that 5 J/cm₂ increased mitochondrial membrane potential, intracellular calcium, ATP and cyclic adenosine monophosphate (cAMP), while 16 J/cm₂ produced an opposite mitochondrial changes in the damaged fibroblast.

-LLLT also promotes angiogenesis. For the direct effect of LLLT on human umbilical vein endothelial cells (HUVEC), Terena et al. (<u>31</u>) showed red laser (660 nm, 1–20 J/cm₂) could increase the viability and protein concentration of HUVEC from the second post-irradiation day. While infrared laser (780 nm, 1–20 J/cm₂) generally reduced cell viability.

LASER DOSE AND SAFETY



Treatment Times Comparison Example (depth = 1cm, area = 6 cm2*) Based on Laser Power

Surface Density ()

<u>100mW System</u> (REMEMBER ERCHONIA'S DIODES ARE 7.5 AND 17.5 mW) 10 J/cm2 = 600 sec (10 minutes) 24 J/cm2 = 1440 sec (24 minutes) 48 J/cm2 = 2880 sec (48 minutes)

> 1000mW (1 Watt) System 10 J/cm2 = 60 seconds 24 J/cm2 = 144 seconds 48 J/cm2 = 288 seconds

 $\frac{10,000 \text{mW} (10 \text{ Watt}) \text{ System (REMEMBER MANY CLASS 4 LASERS ARE 30}}{\text{WATTS OR ABOVE!}}$ 10 J/cm2 = 6 seconds 24 J/cm2 = 14.4 seconds $48 \text{ J/cm2} = _{75}28 \text{ seconds}$

LASER DOSE AND SAFETY



SIMPLE EQUATION: WATTAGE X TIME = TOTAL JOULES EX. ERCHONIA HANDHELD RED DIODES ARE 7.5 mW SO A 10 MINUTE TREATMENT WOULD BE CALCULATED AS 0.0075 WATTS X 600 SECONDS = 4.5 JOULES

FX 635 HAS 3 17.5 mW DIODES SO A 10 MINUTE TREATMENT WOULD BE 0.0175 X 600 SECONDS = 10.5 JOULES X3 = 31.5 JOULES TOTAL BUT THESE ARE STATIC JOULES VS ERCHONIA USES LINE BEAM AND ROTATING DIODES

OTHER VARIABLES HAVE TO BE FACTORED IN WHICH MAKES CALCULATING DOSE TO TISSUE VERY COMPLEX. PULSED VS CONTINUAL WAVE, DISTANCE FROM TISSUE, ABSORPTION TO TARGET TISSUE (IE ~6% TRANSCRANIAL TO CORTICAL MATTER), IS THE BEAM MOVING OR STATIC, SKIN COLOR, BEAM ANGLE, CLOTHES AND MORE.

THEN GENERAL RULE IS TO NOT EXCEED 20 JOULES TO THE SURFACE FOR OPTIMAL SAFETY AND EFFICACY WHICH IS EFFECTIVELY ACCOMPLISHED WITH ERCHONIA'S TECHNOLOGY. MOST OTHER LASER COMPANIES EXCEED THIS THRESHOLD (SOME DRAMATICALLY EXCEED THIS CUT OFF) WITH "NORMAL" Duplication, citation or religibility in the praterial in whole or in part



Front Oncol. 2020; 10: 1255.

Published online 2020 Jul 24. doi: 10.3389/fonc.2020.01255

Review on the Cellular Mechanisms of Low-Level Laser Therapy Use in Oncology

-Frigo et al. (35) compared the effects of 2 energy densities using GaAlAs 660 nm laser (50 mW, 150 or 1,050 J/cm2, three consecutive daily irradiation) on human keloid fibroblasts and murine fibroblast 3T3. *MTT assay showed an increased cell proliferation and reduced cell death from hypodiploid cell with LLLT of 150 J/cm2 LLLT. However, at 1,050 J/cm2 it decreased cell proliferation and increased the percentage of hypodiploid cells, which could be due to apoptosis or a reduced number of active dividing cells.*

-In summary, LLLT at lower energy range inhibits apoptosis but paradoxically promotes apoptosis at a higher energy range.

-Wavelength ex. Bamps et al. (56) studied the post-LLLT (830 nm, 1 and 2 J/cm₂) proliferation of head and neck squamous cell carcinoma (HNSCC) cell lines and reported an increased cell proliferation with expression of phosphor-protein kinase B (Akt), phospho-ERK and Ki67 gene markers implying an increased cancer aggressiveness.

SUMMARY STATEMENT: THE RULE OF 10's. DO NOT GO ABOVE 10 J/CM2 OR 10 eV

WHY LOW LEVEL LASER THERAPY AND NOT OTHER LIGHT DEVICES



-IONIZING RADIATION STARTS AT ABOUT 10 EV (ELECTRON VOLTS) -VIOLET LASERS WILL REACH 3.06 EV -YOU HAVE PLENTY OF BUFFER ROOM FOR ALL OF ERCHONIA'S WAVELENGTHS -YOU CAN TREAT FOR LONGER PERIODS WITH ANY COLOR OF ERCHONIA'S DIODES, BUT IT IS NOT NECESSARY TO EXCITE VALENCE ELECTRONS, CREATE THERAPEUTIC ROS ETC.

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

VIOLET WAVELENGTHS AND DOSE



Biology (Basel). 2022 Feb; 11(2): 301. Published online 2022 Feb 12. doi: <u>10.3390/biology11020301</u>

Blue Laser Irradiation Decreases the ATP Level in Mouse Skin and Increases the Production of Superoxide Anion and Hypochlorous Acid in Mouse Fibroblasts

Eiko Nakayama,1,

Photobiomodulation studies have reported that blue light irradiation induces the production of reactive oxygen species. We investigated the effect of **blue laser (405 nm)** irradiation on the ATP levels in mouse skin and determined the types of reactive oxygen species and reactive nitrogen species using cultured mouse fibroblasts. Blue laser irradiation caused a decrease in the ATP level in the mouse skin and triggered the generation of superoxide anion and hypochlorous acid, whereas nitric oxide and peroxynitrite were not detected. Moreover, blue laser irradiation resulted in reduced cell viability. It is believed that the decrease in the skin ATP level due to blue light irradiation results from the increased levels of oxidative stress due to the generation of reactive oxygen species. This method of systematically measuring the levels of reactive oxygen species and reactive nitrogen species may be useful for understanding the effects of irradiation conditions.

405 nm blue laser irradiation at 100 mW/cm2 for 180 s decreased cell viability

Red Laser Power	1000mW
Violet Laser Power	300mW
Red Laser Wavelength	637nm
Violet Laser Wavelength	405nm
Pulsing	\checkmark
Continuous	\checkmark
Laser Class	3B
Cordless	\checkmark
Weight	5oz
Rechargeable	\checkmark
Treatment Presets	>225 ¹

ALZHEIMER'S DETECTION



Scientists reveal how you can spot Alzheimer's Disease 9 years before diagnosis

Early intervention is vital.

A new study has indicated that it is possible to spot signs of dementia — including conditions like Alzheimer's Disease — up to nine years before actual diagnosis.

The study, which was <u>published</u> in the journal titled "Alzheimer's & Dementia: The Journal of the Alzheimer's Association", said that there were some early signs to watch out for that could end up becoming full blown symptoms of such conditions. **VOLUME 14, 2022**

The study took into account the data derived from the UK Biobank, and identified two early signs that could

indicate whether or not a patient could end up developing dementia - problem

solving and number recall.

The data showed that those who went on to develop Alzheimer's had scored worse compared to healthy individuals during **exercises involving problem solving, reaction times, remembering lists of numbers, pair matching, and the ability to remember things after time had passed.**

CLINICAL: DON'T SKIP BASICS ON YOUR BEDSIDE EXAM INCLUDING PERSON, PLACE AND THING. SERIES 7 REPEAT (BILATERAL). 7 NUMBER COUNT BACK (AUSCULTATION CHALLENGE?). 3 OBJECT DELAY RECALL. CAN THEY FILL OUT THEIR OWN PAPERWORK/COMPETENCY. MMSE. without permission of Trever Berry, DC, DACNB is prohibited ALZHEIMER'S DETECTION



EARLY WARNING SYMPTOMS...

NUCLEUS ACCUMBENS => APATHY

Article
 Rublished: 00 February 2022

Amyloid-β oligomers in the nucleus accumbens decrease motivation via insertion of calciumpermeable AMPA receptors

Changyong Guo, Di Wen, Yihong Zhang, Richie Mustaklem, Basil Mustaklem, Miou Zhou, Tao Ma & Yao-Ying Ma

SUMMARY OF EARLY WARNING SYMPTOMS: MEMORY RECALL/NUMBER RECALL APATHY LOSS OF SMELL PROBLEM SOLVING

ALZHEIMER'S DETECTION



Published: 04 December 2018

Genetic data and cognitively defined late-onset Alzheimer's disease subgroups

• Shubhabrata Mukherjee, Jesse Mez, [...]Paul K. Crane Molecular Psychiatry volume

25, pages 2942-2951(2020)

The largest subgroup, comprising around 39 percent of subjects, displayed similar scores across all four cognitive domains, and the second largest subgroup, at 27 percent, displayed similar average scores except on memory, which revealed significantly lower numbers. This means that particular subgroup displayed greater memory impairment than other Alzheimer's subjects. Other more smaller subgroups displayed greater impairment in specific cognitive domains, and 6 percent of subjects revealed two domains with substantially lower scores than others.

The team hopes the research can act as a bouncing off point for a new way of thinking about Alzheimer's disease. By understanding the disease as six different conditions, each with its own unique cognitive and biological fingerprint, there may be a way forward not only for more personalized treatments tailored to individual patients, but more effective and better targeted clinical trials.

THERE IS NOT ONE SMOKING GUN WHEN IT COMES TO TREATMENT AND PREVENTION OF NEURODEGENERATIVE DISEASES LIKE ALZHEIMER'S. YOU MUST BE ABLE TO IDENTIFY AND ADDRESS EVERYTHING FROM BRAIN INFLAMMATION CAUSES AND THE IMMUNE SYSTEM, AUTOIMMUNITY, PATHOGENS, DIET AND LIFESTYLE, CHEMISTRY MARKERS, BRAIN INJURIES, BARRIER SYSTEMS, MICRONUTRIENTS, ENVIRONMENTAL TOXINS AND MORE...

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Landmark study presents evidence Alzheimer's disease begins in the liver

An impressive new study is presenting robust evidence showing the toxic proteins thought to be the cause of Alzheimer's disease may be produced in the liver and travel through the blood before landing in the brain causing neuron damage.

The vast majority of people developing the disease instead experience what is known as sporadic Alzheimer's, where the disease develops in older age, with no familial or genetic history.

But if this liver-amyloid hypothesis is further validated in future studies a number of other outcomes could arise. Alzheimer's disease risk may be estimated at a young age by evaluating an individual's propensity for synthesizing amyloid in the liver. Plus, dietary interventions could hypothetically be deployed to improve liver health and decrease a person's risk of developing Alzheimer's.

CONTROL AMYLOID LIPOPROTEINS MADE IN THE LIVER CONSIDER AN ANNUAL LIVER DETOX CLEANSE

763

Synthesis of human amyloid restricted to liver results in an Alzheimer disease–like neurodegenerative phenotype

Virginie Lam ,
Ryusuke Takechi , Published: September 14, 2021PLoS1

ALZHEIMER'S DETECTION



New Blood Test Accurately Predicts Alzheimer's Years Ahead of First Symptoms

Researchers at the University of Washington (UW) created the novel blood test. It's designed to pick up on a molecular precursor in the blood that can cause proteins to irregularly fold and clump in the brain, ultimately forming amyloid beta (Aβ) plaques.

But recent studies have shown that Aβ plaques are only present in a third of Alzheimer's patients, and sometimes, they are present in the brains of people who experience no cognitive deficits.

In other words, extracellular Aβ plaques in the brain aren't necessarily toxic in and of themselves, but they might stem from notoriously difficult-to-detect molecular toxins.

UW researchers to make an impressively accurate *soluble oligomer binding assay*, nicknamed SOBA.

By measuring toxic Aβ oligomers in the blood plasma, **SOBA picked out all 53 participants with Alzheimer's who were later confirmed to have the disease post-mortem.**

SOBA: Development and testing of a soluble oligomer binding assay for detection of amyloidogenic toxic oligomers

Dylan Shea, Elizabeth Colasurdo https://orcid.org/0000-0002-3259-0098, Alec Smith https://orcid.org/0000-0002-3001-8484, +7, and Valerie Daggett @uw.eduAuthors Info & Affiliations Edited by Claudio Soto The University of Texas Health Science Center at Houston TX: received Aueust 12, 2022, accented October 17, 2022 by Editorial Board Member Angela M. Grogenborn

December 9, 2022 119 (50) e2213157119

naterial in whole or in part , DACNB is prohibited.	ributionagfnthis n revor Berry, DC	tianerationsensolist oute permission sof T	Mild co Ghinplica impairme with	
NOF		of dementia		
	×	 Image: A second s	~	Occasionally forgetting a date, word, appointment, or task
MILD	~	~	~	Slight difficulty paying attention or multitasking
(EST		~	~	Forgetting important info that you usually would've recalled easily, such as recent events or the name of a good friend
OF 65		~	~	Having more trouble coming up with words than others of the same age
A 1 Y		~	~	Struggling with more complex planning or tasks, like balancing a checkbook
AL:		~		Difficulty with basic activities, like using the bathroom, eating, or getting dressed
75% (~		Problems with language or trouble carrying on a conversation
		~		Odd or inappropriate behaviors
OTHE		×		Personality changes
D		~		Problems with balance and coordination
(KEM) V/		~		Repeating a question or story multiple times
LE		~		Excessive tripping, falls, or tremors
	765	1		Wandering or getting lost

NORMAL AGING VS MILD COGINITIVE IMPAIRMENT (ESTIMATED 20%) **OF MCI OVER AGE OF 65 WILL BECOME ALZHEIMER'S OVER** A 1 YEAR PERIOD) VS **ALZHEIMER'S** (APPROXIMATELY 75% OF DEMENTIA CASES) AND **OTHER FORMS OF** DEMENTIA **(REMAINING ~ 25%) VASCULAR**, LEWY BODY)

ALZHEIMER'S DETECTION



FDA Permits Marketing for New Test to Improve Diagnosis of Alzheimer's Disease MAY 04, 2022

FDA NEWS RELEASE

The U.S. Food and Drug Administration today permitted marketing for the first in vitro diagnostic test for early detection of amyloid plaques associated with Alzheimer's

disease. The Lumipulse G β -Amyloid Ratio (1-42/1-40) test is intended to be used in adult patients, aged 55 years and older, presenting with cognitive impairment who are being evaluated for Alzheimer's disease

and other causes of cognitive decline.

"The availability of an in vitro diagnostic test that can potentially eliminate the need for time-consuming and expensive PET scans is great news for individuals and families concerned with the possibility of an Alzheimer's disease diagnosis," said Jeff Shuren, M.D., J.D., director of the FDA's Center for Devices and Radiological Health. "With the Lumipulse test, there is a new option that can typically be completed the same day and can give doctors the same information regarding brain amyloid status, without the radiation risk, to help determine if a patient's cognitive impairment is due to Alzheimer's disease."

ALZHEIMER'S DETECTION





Neuron specific enolase (NSE) - Severity of nerve cell damage and prognosis. Also a cancer marker (glioblastoma, small cell lung cancer)

- Glial fibrillary acidic protein (GFAP) BBB disruption; TBI, Stroke, Cord injury, "Leaky brain"
- Amyloid beta
 - Abeta 1-42 Closely associated with Alzheimer's (AD)
 - Abeta 1-40 Also an AD amyloid marker but 42 elevation is considered more toxic
- Abeta42/Abeta40 ratio Lower ratio associated with AD
- Total Tau general marker of neurodegeneration
- s100b similar to GFAP, brain injury, "Leaky brain"
- Neurofilament light Axonal injury in Neurological diseases such as AD, CTE, Down's syndrome etc
- alpha synuclein Lewy body protein. Elevated in conditions like Parkinson's, MSA and Lewy body dementia
- Brain-derived neurotrophic factor (BDNF) Needed for neuroplasticity. Low levels associated with neurological conditions such as Depression₇₆MCI and other neurodegenerative conditions, Schizophrenia and more

ALZHEIMER'S DETECTION



Testing:

- 1. New Patient Intake, Brain Region Localization Form, Metabolic Assessment Form, Past Record Review, Food Journal
- 2. MMSE, Mini-Cog, GPCOG (General Practitioner assessment of cognition)
- 3. VNG, HRV, CDP, Grip Strength, Spirometer, Bilateral BP and Pulse-Oximeter with BPI
- 4. Full Bedside Neurological Work-up (Motor, Sensory, ANS, Orthopedic, Gait etc)
- 5. NSI Baseline Prosaccades, Interactive Metronome
- 6. ROF
- 7. Labs (Prefer to have in advance):
 - a. CBC with diff, CMP, Full Thyroid Panel, Sex Hormones, HS-CRP, HBA1c, Homocysteine
 - b. ViT D/Micronutrient Panel, Ox-LDL/sdLDL/PLa2/Boston Heart Panel, Wheat Zoomer (Intestinal Permeability), Food Sensitivity Panel, Neural Zoomer Plus, Gut Zoomer 3.0
- 8. Other: MRI, MRA, CT, C-spine Xrays, SPECT

Educational Packet for Caregiver

Bredeson Study Summary

Laser Studies (see below)

Books: The End of Alzheimer's, Dr. Dale E. Bredeson; Alzheimer's: What if there was a Cure? Dr. Mary Newport; The Brain's Way of Healing, Dr. Norman Doidge, Excitotoxins: The Taste that Kills, Dr. Russel Blaylock, Brain Maker, Dr. David Perlmutter

Panel For the detection of patterns of imbalance in critical brain proteins

VibrantWellness

Neural Health

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ALZHEIMER'S DETECTION



Treatment overview:

12 visit treatment minimum with patient centric focus

Advanced brain setting 1-10-40-60 (1-40, 40-60, 10-10) TCT plus Vagal nerve stimulation protocol

Co-activate Laser with Binaural therapies, Brain Tap, Olfaction, Focus Builder APP, O2 source

Vestibular therapies. Ex. Yaw axis recall with auditory and olfaction stimulation, Balance modalities

Oculomotor therapies

NSI, Neurosage, Tone Pacer with Gait Dual task, Focus Builder APP

Brain Based LDA (Chiropractic, PNF, ART, Fast Stretch, NMR, K-Tape, Localization, FNOR, Resistance Training)

Mapping (Retinotopic, Somatotopic, Tonotopic)

HOME: Lumosity/Brain HQ apps, music/art/instrument learning, interactive video games/ card games, aromatherapy, dance/yoga/tai chi, meditation

3x/week 4 weeks TREATMENT TRIAL

ALL OF YOU CAN TAKE ON THIS ROLE OR CO-MANAGE YOUR PATIENTS WITH SOMEONE THAT DOES

CLINICAL CONSIDERATIONS...



OTHER RESEARCH BACKING GREEN WAVELENGTH LASER THERAPY:

- SKIN CONDITIONS! EXAMPLES, BASAL CELL CARCINOMA REDUCTION, CHRONIC LICHEN SCLEROSUS, SOLAR KERATOSES, PORT WINE STAINS, ACTINIC KERATOSIS (AK), PROMOTE HUMAN NORMAL EPIDERMAL KERATINOCYTE AND DERMAL FIBROBLASTS (PRIMARY CELLS OF THE EPIDERMIS)
- -BONE REGENERATION
- BENIGN PROSTATE ENLARGEMENT REDUCTION (ABLATIVE)
- SUPERFICIAL LESIONS LIKE BED SORES AND DIABETIC. ULCERS
- -BODY CIRCUMFERENCE AND CELLULITE REDUCTION

Int J Mol Sci. 2021 Mar; 22(5): 2437. Published online 2021 Feb 28. doi: <u>10.3390/ijms22052437</u>

PMCID: PMC7957604 PMID: <u>33670977</u>

Effect of Different Wavelengths of Laser Irradiation on the Skin Cells Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CLINICAL CONSIDERATIONS...



Abstract

Breast cancer is responsible for one of the top leading causes of cancer deaths among women. Radiotherapy (RT) uses high energy radiation to kill cancer cells, but this method has been reportedly linked to risks of toxicity. Post-therapeutic relapse from RT believed to be caused by its toxicity is one of the challenges encountered during tumour therapy. Therefore, further attention should be devoted to developing novel anti-tumour therapeutic approaches. The role of low-level laser therapy (LLLT) in breast cancer management is to alleviate the side effects arising from RT, instead of acting against the tumour cells directly. **This study investigated the effects of low-level laser (532 nm), as well as single and fractionated irradiation, on breast cancer MCF 7 cell line.** Additionally, this study assessed the most effective laser parameter for fractionated irradiation. The MCF 7 cells were irradiated with green laser power at 1.5, 45.0, and 100.0 mW with a spot size diameter of 0.7 mm for 1, 5, 10, and 15 min. The irradiation was carried out in single, double, and triple fractionation separated by 5- and 10-min intervals in between the fractional regimes. The laser output of 100 mW

showed a promising potential in killing cells with single fractionation. However, as the irradiation was fractionated into two, power of 1.5 mW appeared to be more effective in cell death, which contributed to the lowest percentage cells viable of 31.4% recorded in the study. It was proven that fractionated regime was more successful in tumor cell death.

Lasers Med Sci 2022 Mar;37(2):1265-1271. doi: 10.1007/s10103-021-03384-0. Epub 2021 Jul 31. Fractionated low-level laser irradiation on breast cancer (MCF 7 cells) treatment

Nursakinah Suardi 1, Pegah Moradi Khaniabadi 23, Aijesta Taggo 4771

<section-header>ODEINAL BRAIN FREQUENCIES DESCRIPTION OF A CONSTRUCTION OF A CON





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.

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 7 Wu 3

MASTER BRAIN PROTOCOL FREQUENCIES



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 thout permission of Trevor Berry, DC, DACNB is prof



MASTER BRAIN PROTOCOL FREQUENCIES







HEG (HOMOENCEPHALOGRAPHY) MONITORS ENERGY CONSUMPTION PRIMARILY IN THE PRE-FRONTAL CORTEX TO TRAIN EXECUTIVE FUNCTION (IE FOCUS AND CONCENTRATION). "CLIMB" AND "DIVE"



USE DELTA WAVE (1-1-1-1 OR 1-2-3-4) OR DELTA-VAGAL (1-10-1-10) STRESS REDUCTION ANTI-ANXIETY SLEEP/CALM ETC

VAGAL NERVE STIMULATION: 10 HZ FREQUENCY



Long Term Effects of Low Frequency (10 Hz) Vagus Nerve Stimulation on EEG and Heart Rate Variability in Crohn's Disease: A Case Report

November 28, 2000; 55 (10) EXPEDITED PUBLICATION Effects of vagus nerve stimulation on respiration during sleep A pilot study B.A. Malow, J. Edwards, M. Marzec, O. Sagher and G. Fromes

Autonomic Regulation Therapy via Left or Right Cervical Vagus Nerve Stimulation in Patients With Chronic Heart Failure: Results of the ANTHEM-HF Trial Author links open overlay panel

Rajendra K.PremchandyDarkamalSharmaMD2SanjayMittalMD3RufinoMonteiroMD4SatyajitDixitMD2ImadLibbusPhD5Lorenzo A.DiCarloMD6Jeffrey L.ArdellPhD7Thomas S.RectorPharmD, PhD8BadriAmurthurMS5Bruce H.KenKnightPhD5Inder S.AnandMD, DPhil (Oxon), FRCP8

Vagus nerve stimulation inhibits cytokine production and attenuates disease severity in rheumatoid arthritis

Frieda A. Koopmana,

Chronic vagus nerve stimulation reduces body fat, blood cholesterol and triglyceride levels in rats fed a high-fat diet

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Photomed Laser Surg

2017 Aug;35(8):432-441. doi: 10.1089/pho.2016.4227. Epub 2017 Feb 10.

Significant Improvement in Cognition in Mild to Moderately Severe Dementia Cases Treated with Transcranial Plus Intranasal Photobiomodulation: Case Series Report

Anita E Saltmarche 1, Margaret A Naeser 23, Kai Fai Ho 4, Michael R Hamblin 56, Lew Lim 7

Neuropsychiatr Dis Treat

2015 Aug 20;11:2159-75. doi: 10.2147/NDT.S65809. eCollection 2015.

Treatments for traumatic brain injury with emphasis on transcranial near-infrared laser phototherapy

Larry D Morries 1, Paolo Cassano 2, Theodore A Henderson 3

Therapeutic effects of 10-HzPulsed wave lasers in rat depression model: A comparison between near-infrared and red wavelengths.

Salehpour F, Rasta SH, Mohaddes G, Sadigh-Eteghad S, Salarirad S Lasers Surg Med. 2016 Sep; 48(7):695-705.

MASTER BRAIN PROTOCOL FREQUENCIES: 40 HZ



Exp Brain Res. 2017 Apr 13. doi: 10.1007/s00221-017-4957-9. [Epub ahead of print] **The effect of gamma-enhancing binaural beats on the control of feature bindings.** Colzato LS1,2,3, Steenbergen L4, Sellaro R4.

Healthy adults listened to gamma-frequency (40 Hz) binaural beats or to a constant tone of 340 Hz (control condition) for ten minutes before and during a feature-repetition task. While the size of visuomotor binding costs (indicating the binding of visual and action features) was unaffected by the binaural beats, the size of visual feature binding costs (which refer to the binding between the two visual features) was considerably smaller during gamma-frequency binaural beats exposure than during the control condition. Our results suggest that binaural beats enhance selectivity in updating episodic memory traces and further strengthen the hypothesis that neural activity in the gamma band is critically associated with the control of feature binding.

Technol Health Care. 2015;23 Suppl 2:S465-71. doi: 10.3233/THC-150983.

Studying frequency processing of the brain to enhance long-term memory and develop a human brain protocol.

Friedrich W1, Du S2, Balt K3.

Fifty-eight participants aged between 6 and 60 years participated in long-term memory experiments. It is envisaged that the brain could be stimulated through binaural beats (sound frequency) at 40 Hz (gamma) to enhance long-term memory capacity. EEG recordings have been transformed to sound and then to an information standard, namely ASCII.

Statistical analysis showed a proportional relationship between long-term memory and gamma activity. Results from EEG recordings indicate a pattern. The pattern was obtained through the de-codification of an EEG recording to sound and then to ASCII. **CONCLUSIONS:**

Stimulation of gamma should pem han ce the PA package of the state of

MASTER BRAIN PROTOCOL FREQUENCIES: 40 HZ



Published: 20 May 2020

Physical exercise during exposure to 40-Hz light flicker improves cognitive functions in the 3xTg mouse model of Alzheimer's disease

Sang-Seo Park, Hye-Sang Park, Chang-Ju Kim, Hyun-Sik Kang, Dong-Hyun Kim, Seung-Soo Baek & Tae-Woon Kim

Abstract

Background

Exercise promotes brain health and improves cognitive functioning in the elderly, while 40-Hz light flickering through the visual cortex reduces amyloid beta ($A\beta$) by stabilizing gamma oscillation. We examined whether exercise was associated with hippocampus-mediated improvement in cognitive functioning in the 3xTg-Alzheimer's disease (3xTg-AD) murine model following exposure to 40-Hz light flickering and exercise.

Conclusion

Our results show that exercising in a 40-Hz light flickering environment may improve cognitive functioning by reducing A β and tau levels, thereby enhancing mitochondrial function and neuroplasticity.

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MASTER BRAIN FREQUENCIES



First human trials test light & sound therapy for Alzheimer's disease

A new study published in the journal *PLoS ONE* has reported on the first human tests of an experimental therapy using sound and light

to treat Alzheimer's disease (AD). The initial findings are promising, with the unique treatment leading to some neurological and cognitive improvements

Over the last seven years, Li-Huei Tsai and colleagues at MIT's Picower Institute for Learning and Memory have been investigating an unusual hypothesis. The researchers found toxic proteins associated with Alzheimer's disease could be

eliminated from mouse brains following exposure to flickering lights. Further research found

the magic frequency was 40 Hz. When animals were exposed to both sound and light at that frequency, improvements in brain health were detected.

Gamma frequency sensory stimulation in mild probable Alzheimer's dementia patients: Results of feasibility and pilot studies

Diane Chan ,
Ho-Jun Suk ,

• Published: December cititio 2022 butio 78% f this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

MASTER BRAIN PROTOCOL FREQUENCIES: 40 HZ



PMCID: PMC6758739

<u>J Neurosci.</u> 2002 Dec 1; 22(23): 10501–10506. doi: <u>10.1523/JNEUROSCI.22-23-10501.2002</u>

Activation of Human Cerebral and Cerebellar Cortex by Auditory Stimulation at 40 Hz

Maria A. Pastor, 1 Julio Artieda, 1 Javier Arbizu, 2 Josep M. Marti-Climent, 2 Ivan Peñuelas, 2 and Jose C. Masdeu 1 Abstract

We used functional brain imaging with positron emission tomography (PET)-H₂₁₅O to study a remarkable neurophysiological finding in the normal brain. Auditory stimulation at various frequencies in the gamma range elicits a steady-state scalp electroencephalographic (EEG) response that peaks in amplitude at 40 Hz, with smaller amplitudes at lower and higher stimulation frequencies. We confirmed this finding in 28 healthy subjects, each studied with monaural trains of stimuli at **12 different stimulation rates (12, 20, 30, 32, 35, 37.5, 40, 42.5, 45, 47.5, 50, and 60 Hz)**. There is disagreement as to whether the peak in the amplitude of the EEG response at 40 Hz corresponds simply to a superimposition of middle latency auditory evoked potentials, neuronal synchronization, or increased cortical synaptic activity at this stimulation frequency. To clarify this issue, we measured regional cerebral blood flow (rCBF) with PET-H₂₁₅O in nine normal subjects at rest and during auditory stimulation at four different frequencies

(12, 32, 40, and 47 Hz) and analyzed the results with statistical parametric mapping. The behavior of the ${f rCBF}$

(CEREBRAL BLOOD FLOW) response was similar to the steady-state EEG response, reaching a peak at 40

Hz. This finding suggests that the steady-state amplitude peak is related to increased cortical synaptic activity.

Additionally, we found that, compared with other stimulation frequencies, 40 Hz selectively activated the auditory region of the pontocerebellum, a brain structure with important roles in cortical inhibition and timing.

MASTER BRAIN PROTOCOL FREQUENCIES: 40 HZ



Nature

2016 Dec 7;540(7632):230-235. doi: 10.1038/nature20587.

Gamma frequency entrainment attenuates amyloid load and modifies microglia

Hannah F laccarino 1 2, Annabelle C Singer 3 2 4,

Abstract

Changes in gamma oscillations (20-50 Hz) have been observed in several neurological disorders. However, the relationship between gamma oscillations and cellular pathologies is unclear. Here we show reduced, behaviourally driven gamma oscillations before the onset of plaque formation or

cognitive decline in a mouse model of Alzheimer's disease. Optogenetically driving fast-spiking

parvalbumin-positive (FS-PV)-interneurons at gamma (40 Hz), but not other frequencies, reduces levels of amyloid-β

(A β)1-40 and A β 1-42 isoforms. Gene expression profiling revealed induction of genes associated with morphological transformation of microglia, and histological analysis confirmed increased microglia co-localization with A β . Subsequently, we designed a non-invasive 40 Hz light-flickering regime that reduced A β 1-40 and A β 1-42 levels in the visual cortex of pre-depositing mice and mitigated plaque load in aged, depositing mice. Our findings uncover a previously unappreciated function of gamma rhythms in crecruiting both neuropal and glial responses to attenuate Alzheimer's-disease-associated pathologythout permission of Trevor Berry, DC, DACNB is prohibited.

MASTER BRAIN PROTOCOL FREQUENCIES



OCTOBER 12, 2016

Scientists research effects of infrasonic vibrations in humans

It is known that the human body can generate mechanical vibrations at very low frequencies, so-called infrasonic waves. Such low-frequency vibrations are produced by physiological processes—heartbeats, respiratory movements, blood flow

in vessels, and other processes. Different organs of the human body produce different resonance frequencies. The heart resonance frequency is ~ 1 hz. The brain has a resonance frequency of ~ 10 hz, blood circulation about 0.05 to 0.3 hz.

The scientists discovered that the observed vibrations are connected with the cardiovascular system, which has its own proper movements occurring simultaneously with the work of the heart. Three types of infrasonic vibrations were registered. Waves of the first type are connected with the heartbeat; the second with the human respiratory rhythm; the third, called Traube-Hering waves, with states of emotional tension. Thus, it could be possible to judge the human emotional state via the amplitude frequency response of the the waves.

MASTER BRAIN PROTOCOL FREQUENCIES



Eur J Neurosci

. 2006 Apr;23(7):1956-60. doi: 10.1111/j.1460-9568.2006.04717.x.

Reduction in subthalamic 8-35 Hz oscillatory activity correlates with clinical improvement in Parkinson's disease

Andrea A Kühn 1, Andreas Kupsch, Gerd-Helge Schneider, Peter Brown

Abstract

Strong synchronization of neuronal activity occurs in the 8-35 Hz band in the subthalamic nucleus (STN) of patients with Parkinson's disease (PD) and is evident as oscillatory local field potential (LFP) activity. To test whether such synchronization may contribute to bradykinesia and rigidity, we sought correlations between the suppression of synchronization at 8-35 Hz in STN and the reduction in Parkinsonism with levodopa. LFPs were recorded on and off medication from STN deep-brain stimulation electrodes in nine PD patients. LFP power was calculated over the frequencies of the most prominent spectral peak within the 8-35 Hz frequency band on each of 17 sides (off medication), and over the frequencies of any peak in the 60-90 Hz band, if present (seven sides, on medication). Levodopa-induced reduction of LFP power over these two frequency ranges was then correlated with improvement in motor impairment as assessed by the Unified Parkinson's Disease

Rating Scale (UPDRS). The reduction in peak activity in the 8-35 Hz band with levodopa positively correlated with the improvement in the contralateral hemibody motor UPDRS score with levodopa (r = 0.811, P < 0.001) as well as with hemibody subscores of akinesia-rigidity (r = 0.835, P < 0.001), but not

tremor. A trend for negative correlations was found between peak 60-90 Hz LFP power and UPDRS hemibody score, suggesting that positive correlations were relatively frequency-specific. Our results support a link between levodopa-induced improvements in bradykinesia and rigidity and reductions in population synchrony at frequencies < 35 Hz in the region of the STN in patients with PD.

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MASTER BRAIN PROTOCOL FREQUENCIES



2019 Dec;160(12):2751-2765. doi: 10.1097/j.pain.000000000001666.

Brain dysfunction in chronic pain patients assessed by restingstate electroencephalography

Son Ta Dinh 1 2, Moritz M Nickel 1 2, Laura Tiemann 1 Abstract

Chronic pain is a common and severely disabling disease whose treatment is often unsatisfactory. Insights into the brain mechanisms of chronic pain promise to advance the understanding of the underlying pathophysiology and might help to develop disease markers and novel treatments. Here, we systematically exploited the potential of electroencephalography to determine abnormalities of brain function during the resting state in chronic pain. To this end, we performed state-of-the-art analyses of oscillatory brain activity, brain connectivity, and brain networks in 101 patients of either sex suffering from chronic pain. The results show that global and local measures of brain activity did not differ

between chronic pain patients and a healthy control group. However, we observed significantly

increased connectivity at theta (4-8 Hz) and (High) gamma (>60 Hz) frequencies in frontal brain areas as well as global network reorganization at gamma frequencies in chronic

pain patients. Furthermore, a machine learning algorithm could differentiate between patients and healthy controls with an above-chance accuracy of 57%, mostly based on frontal connectivity. These results suggest that increased theta and gamma synchrony in frontal brain areas are involved in the pathophysiology of chronic pain. Although substantial challenges concerning the reproducibility of the

THIS BECOMES VERY IMPORTANT AS WE "SWIM UP STREAM" USING TRANSCRANIAL APPLICATIONS TO"TREAT CHRONIC PAIN SUFFERERS

MASTER BRAIN PROTOCOL FREQUENCIES



Pain Med. 2017 Oct; 18(10): 1921–1931. Published online 2017 Jan 13. doi: <u>10.1093/pm/pnw294</u>

PMCID: PMC6407607 PMID: <u>28087845</u>

Waking EEG Cortical Markers of Chronic Pain and Sleepiness

Danny Camfferman, PhD,* G. Lorimer Moseley, PhD,* Kevin Gertz, MPA,† Mark W. Pettet, PhD,‡ and Mark P. Jensen, PhD†

Thalamic hyperpolarization among individuals with chronic pain is posited to explain the detection of low-frequency resonances in the theta range (4-8 Hz) [1-5]localized in the thalamus [1] and high theta (6–9 Hz) activity [2–5,21] localized in the posterior insular, inferior posterior parietal cortex [2], somatosensory cortex [5,21], and frontal areas [4]. Further, low theta (4–6 Hz) activity has been reported to be localized in the parieto-temporal cortex [2]. Alpha band activity is thought to represent inhibitory drive from activation of GABAergic synapses from the thalamic projections to the cortex [22]. Alpha (8–12 Hz) activity is also reported to be lower in localized in the insule [2] and frontal electrodes via nain [0] SUMMARY: AVOID THETA/HIGH THETA (4-9HZ) TO THE FRONTAL, PARIETAL AND INSULAR LOBES. tl CONVERSELY, INCREASED MID TO HIGH ALPHA a (10-15HZ) IS NEEDED THALAMOCORTICAL, FRONTAL AND INSULAR REGIONS FOR CHRONIC PAIN

Without permission of Trever Berry, DC, DACNB is prohibited MASTER BRAIN FREQUENCIES



40 Hz sensory stimulation significantly benefits Alzheimer's patients. PLOS ONE Jan. 3, 2023:

A pair of early stage clinical studies testing the safety and efficacy of 40Hz sensory stimulation to treat Alzheimer's disease has found that the potential therapy was well tolerated, produced no serious adverse effects and was associated with some significant neurological and behavioral benefits among a small cohort of participants.

"In these clinical studies we were pleased to see that volunteers did not experience any safety issues and used our experimental light and sound devices in their homes consistently," said Li-Huei Tsai, Picower Professor in the The Picower Institute for Learning and Memory at MIT and senior author of the paper describing the studies in PLOS ONE.

Study results

In the Phase 1 study volunteers filled out a questionnaire on side effects, reporting a few minor but no major adverse effects. The most common was feeling "sleepy or drowsy." Meanwhile, measurements taken with EEG scalp electrodes clustered at frontal and occipital sites showed significant increases in 40Hz rhythm power at each cortical site among cognitively normal younger and older participants as well as volunteers with mild Alzheimer's. The readings also demonstrated significant increase in coherence at the 40Hz frequency between the two sites. Between the two volunteers with epilepsy, measurements showed significant increases in 40Hz power in deeper brain regions such as the gyrus rectus, amygdala, hippocampus and insula with no adverse events including seizures. In the Phase 2A study, neither treated nor control volunteers reported serious adverse events. Both groups used their devices 90 percent of the time. The eight volunteers treated with 40Hz stimulation experienced several beneficial effects that reached statistical significance compared to the seven

volunteers in the control condition. Control participants exhibited two signs of brain atrophy as expected with disease progression: reduced volume of the hippocampus and increased volume of open spaces, or ventricles. Treated patients did not experience significant changes in these measures. Treated patients also exhibited better connectivity across brain regions involved in the brain's default mode and medial visual networks, which are related to cognition and visual processing respectively. Treated patients also exhibited more cons³¹/₃ stent sleep patterns than controls.

MASTER BRAIN PROTOCOL FREQUENCIES



NEWS I SCIENCE & TECH I DEC 29, 2019 AT 10:06 PM

Scientists Cleared Alzheimer's Plaque From Mice Using Nothing but Light and Sound

A previous study showed that flashing **light 40 times a second** into the eyes of mice treated their Alzheimer's disease. If this was not amazing enough, researchers also added sound of a similar frequency and this dramatically improved the results. In a way, we can be talking of "healing frequencies"... some "alternative" therapies in medicine may not sound so crazy now, huh?

This noninvasive treatment also greatly reduced the number of amyloid plaques found in the brains of these mice. Plaques were cleared in large swaths of the brain, including areas critical for cognitive functions such as learning and memory.

MASTER BRAIN SETTING FOR OPTIMAL TRANSCRANIAL OUTCOMES 1-10-40-60 HAND HELDS DEFAULT FOR ANY BRAIN APPLICATION OR FOR UPSTREAM/DOWNSTREAM APPROACHES



Scientists Just Uncovered A Whole New Layer of Brain Anatomy

Researchers from the University of Copenhagen and University

of Rochester have identified a layer of tissue that helps protect our gray and white matter, one that hasn't been distinguished before.

Only a few cells thick, this membrane seems to play a role in mediating the exchange of small, dissolved substances between compartments in the brain. It also appears to be the home base of brain-specific immune cells, not to mention assisting in the brain's waste-removal (glymphatic) system.

their discovery the Subarachnoid LYmphatic-like Membrane (SLYM).

While much of their research on this structure is so far from mice, using two-photon microscopy and dissections, they have confirmed the SLYM's presence in an adult human brain too.

Møllgård and team found several types of immune cells – including myeloid cells and macrophages – camping out in the SLYM, keeping surveillance over the brain.

Tears in the SLYM may explain some of the long term symptoms of traumatic brain injury, Møllgård and team speculate. Disruption of this barrier would allow immune cells from the skull direct access into the brain, cells that are not calibrated for brain conditions. This could explain ongoing inflammation.

A mesothelium divides the subarachnoid space into functional compartments

KJELD MØLLGÅRD **SCIENCE. 5 Jan 2023** Vol 379, Issue 6627. pp. 84-88

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CAUSES OF NEURODEGENERATION



ERCHONIA

A New Approach to Halting the Effects of Aging: Boosting Immune Cells Improves Brain Waste Clearance

They discovered that immune cells surrounding the brain play a role in the efficiency of waste removal and that these immune cells are impaired in old mice, as well as in humans and mice with Alzheimer's

disease. Additionally, they found that treating old mice with an **immune-stimulating compound can**

rejuvenate these immune cells and improve waste clearance from the brain.

"Cerebrospinal fluid flow is impaired in numerous neurodegenerative diseases, such as Alzheimer's, stroke, Parkinson's, and multiple sclerosis," Drieu said. "If we can restore fluid flow through the brain just by boosting these macrophages, maybe we can slow the progression of these diseases. It's a dream, but who knows? It might work."

Further investigation revealed that parenchymal border *macrophages* are altered in people with Alzheimer's disease and mice with an Alzheimer's-like condition: The immune cells are less able to consume and dispose of waste, and cannot efficiently regulate fluid flow.

"It doesn't look likely that we will be able to revive dead or dying neurons, **but the immune cells that sit on the**

borders of the brain are a feasible target for treating age-related brain diseases." SLYM AND GLYMPH SYSTEMS

Reference: "Parenchymal border macrophages regulate the flow dynamics of the cerebrospinal fluid" by Antoine Drieu, Siling Du, Steffen E. Storck, Justin Rustenhoven, Zachary Papadopoulos, Taitea Dykstra, Fenghe Zhong, Kyungdeok Kim, Susan Blackburn, Tornike Mamuladze, Oscar Harari, Celeste M. Karch, Randall J. Bateman, Richard Perrin, Martin Farlow, Jasmeer Chhatwal, Dominantly Inherited Alzheimer Network, Song Hu, Gwendalyn J. Randolph, Igor Smirnov and Jonathan Kipnis, **9** November

2022, *Nature*. Duplication, citation or distributio796f this material in whole or in part DOI: 10.1038/s41586-022-05397-3 without permission of Trevor Berry, DC, DACNB is prohibited.

EFFECT OF LLLTON



Braz J Phys Ther. 2014 Jul-Aug; 18(4): 308–314. doi: <u>10.1590/bjpt-rbf.2014.0046</u>

Effect of low-level laser therapy on the modulation of the mitochondrial activity of macrophages

Nadhia H. C. Souza, 1 Raquel A. M. Ferrari, 1, 2 Daniela F. T. Silva, 3 Fabio D. Nunes, 4 Sandra K. Bussadori, 2 and Kristianne P. S. Fernandes 2

CONCLUSIONS: These results show that 660 nm and 780 nm LLLT can modulate the cellular activation status of macrophages in inflammation,

highlighting the importance of this resource and of the correct determination of its parameters in the repair process of skeletal muscle.

Open Access
 Published: 04 April 2017

Low-level laser facilitates alternatively activated macrophage/microglia polarization and promotes functional recovery after crush spinal cord injury in rats

 Ji Wei Song, Kun Li, Zhuo Wen Liang, Chen Dai, Xue Feng Shen, Yu Ze Gong, Shuang Wang, Xue Yu Hu & Zhe Wang <u>Scientific Reports</u> volume

7, Article number: 620 (2017)

Results from this study show that low-level laser therapy has the potential for reducing inflammation, regulating macrophage/microglia polarization, and promoting neuronal survival.

THE 5 MINUTE BASIC NETWORK INTERFERENCE BEDSIDE EXAMINATION

-THIS IS MEANT TO BE A SCREENING TOOL FOR ALL LEVELS OF HEALTHCARE PROVIDER TRAINING -IT IS NOT MEANT TO BE THE FULL COMPREHENSIVE WORK UP

-ANY ABNORMAL SECTION OF THE EXAM WARRANTS FURTHER INVESTIGATION

-PRACTICE OBSERVATION SKILLS. LIKE ANY TEST, THE MORE THE EXAMINER OBSERVES "NORMAL" FINDINGS THE EASIER IT IS TO RECOGNIZE WHEN SOMETHING IS ABNORMAL

-WHEN IN DOUBT, DOCUMENT YOUR OBSERVATIONS OR CALL IN SOMEONE THAT CAN VALIDATE YOUR OBSERVATIONS

-WITH THAT SAID, SOME POSITIVE FINDINGS WILL FATIGUE OR CHANGE AFTER INITIAL ASSESSMENT -ALSO BE AWARE OF FATIGUE. IT MAY BE INDICATED TO OBSERVE SOME TESTS FOR LONGER PERIODS OR REPETITIVE TIMES TO EXPOSE THE LESION

-KEEP DIAGNOSES WITHIN YOUR SCOPE OF PRACTICE. FOR EXAMPLE, YOUR TRAINING MAY NOT ALLOW YOU TO MAKE THE DIAGNOSIS OF A PARKINSON'S PATIENT (ICD-10 CODE G20) BUT YOU CAN MAKE A DIAGNOSIS SUCH AS ICD-10 CODE R26.81 "UNSTEADINESS ON FEET"

-CONSIDER VIDEO RECORDING THE EXAM (WITH PATIENTS PERMISSION). THIS ALLOWS FOR OBSERVATION CONFIRMATION, PATIENT PRE- AND POST-OUTCOMES AND MEDICAL LEGAL PURPOSES


Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC. DACNB is prohibited. THE 5 MINUTE BASIC NETWORK INTERFERENCE BEDSIDE EXAMINATION

THE BEDSIDE NEUROLOGICAL EXAM:

- 1.GAIT PATTERNS (OBSERVE THE PATIENT ENTERING THE EXAM ROOM). BASE/STABILITY, STRIDE, ARM SWING, TURNS, ASYMMETRY
- 2. MENTAL STATUS/AUTONOMIC SYSTEM (HISTORY TAKING, PERSON-PLACE-THING, AFFECT). WHAT IS YOUR NAME? WHERE ARE YOU? WHO IS THE PRESIDENT? MOOD CHANGES? SERIES 7 RECALL (BILATERAL?). YOU OR STAFF CAN BE TAKING BILATERAL BP, PULSE OX (HR, BPI AND 02 SATURATION), AUSCULTATION. CONSIDER MMSE OR MCOG EXAMS
- 3. CRANIAL NERVES (1-12 INTACT, FATIGUE RATIOS, OPTOMETRIC BASICS). GENERAL SMELL TEST, LIGHT REFLEX INTO OCULOMOTOR TRACKING (H IN SPACE-PURSUITS/CONVERGENCE/ SACCADES), SMILE/FROWN/CLENCH TEETH-INTO VOR, WHISPER WITH CONTRA EAR SOUND, "AH" X MULTIPLE, TONGUE STICK OUT AND SHOULDER SHRUG. PERFORM FINGER TO NOSE WITH ARMS EXTENDED OUT FRONT OF THE PATIENT THEN RAPID ALTERNATING MOVEMENTS (CEREBELLAR DYSMETRIA AND DYSDIADOCHOKINESIA) GO STRAIGHT INTO MOTOR BELOW...

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THE BEDSIDE NEUROLOGICAL EXAM CONTINUED:

4.MOTOR FUNCTION (STRENGTH, APRAXIA, SACCADES) HOLD ARM AT 90 RESIST ALL DIRECTIONS (SHOULDER ABDUCTION C5, BICEPS C6, TRICEPS/WRIST/HAND EXTENSION C7, FINGER FLEXION/SQUEEZE C8 (IF PALM UP, OBSERVE CAP REFILL), FINGER ABDUCTION/FLARE T1). GO INTO FINGER TAPPING APRAXIA. THEN LOWER EXTREMITY SEATED (HIP FLEXION L2, KNEE EXTENSION L3, ANKLE DORSIFLEXION L4, GREAT TOE EXTENSION L5, FOOT PLANTAR FLEXION/KNEE FLEXION S1). PERFORM ALL TESTS BILATERALLY

4b.WHILE TESTING LEGS, SUPPORT ANKLE INTO LEG EXTENSION AND PERFORM HEEL TO SHIN AND TEST FOR BABINSKI'S SIGN (SHOULD NOT BE PRESENT >2YO)

5. DTR'S. AFTER COMPLETING BABINSKI'S FOOT STROKE, CONTINUE INTO DEEP TENDON REFLEXES. ACHILLES (S1-2), PATELLAR (L3-4), MOVE TO UE: TRICEPS (C7), BRACHIORADIALIS (C6), BICEPS (C5)





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THE BEDSIDE NEUROLOGICAL EXAM CONTINUED:

6.SENSORY FUNCTION (LARGE AND SMALL DIAMETER TESTING) COTTON BALL TEST INTO WOOD STICK BREAK OR PAINT BRUSH INTO PINWHEEL (FACE, BILATERAL UE AND LE). VIBRATION INTACT VS STOP. LIMB APPRECIATION (TOUCH EITHER ARM OR BOTH AND HAVE PATIENT STATE WHERE YOU ARE TOUCHING). ALL TESTS SHOULD BE PERFORMED EYES CLOSED. SENSORY IS VERY SUBJECTIVE AND A LOT OF OVERLAP IE. UE DERMATOMES SO BE CAUTIOUS. CONSIDER WEBER/RINE WHEN USING TUNING FORK AND CN5 WITH PINWHEEL 3 BRANCHES AND LAMINAR DISTRIBUTION ROSTRAL TO CAUDAL

7. SIT TO STAND (OBSERVE EASE AND SPEED) INTO STANDING TESTS EYES CLOSED ROMBERG'S (OBSERVE SWAY PATTERNS-PROPRIOCEPTION TEST), PUSH PULL (TRUNK STABILITY/VERMIS), HEEL-TOE STANCE, FAKUDA STEP TEST (MARCH IN PLACE EYES CLOSED. PATIENTS TEND TO TURN TO SIDE OF BRAINSTEM WEAKNESS). IF GAIT WAS NOT OBSERVED DURING ENTRANCE, PERFORM GAIT ANALYSIS NOW. CONSIDER DUAL TASK GAIT

ERCHONIA LLLT CAN BE USED DIAGNOSTICALLY AT EVERY STEP OF YOUR EXAM! IF A PATIENT SHOWS A POSITIVE SIGN OR DEFICIT, LASER THE ASSOCIATED REGION WITH THE MASTER 1-10-40-60 SETTING FOR 1-2 MINUTES AND RECHECK

795

MORE LASER CLINICAL CONSIDERATIONS...



-SENSORY TEST (PINWHEEL, VIBRATION ETC) LLLT PNS, CORD, CNS RE-TEST

- MOTOR TEST (MUSCLE TEST/MYOTOMES, DYNAMOMETER ETC) LLLT PNS, CORD, CNS AND RE-TEST
- -AUTONOMICS (BP, PULSE 0X, HRV) LLLT VAGAL STIM/GUT, VMPFC AND MONITOR OR RE-TEST

- ANY NEUROLOGICAL EXAM (CEREBELLUM, CORTICOSPINAL, BALANCE CHECK) LLLT TO THAT REGION AND RE-TEST

-OTHER TECHNIQUES LIKE AK UPREGULATE AND RE-TEST

CLINICAL MANAGEMENT: THAT LASER THERAPY MAY ONLY NEED ONE BALANCING AND TONING VISIT. IT MAY NEED 4, 6, 12 VISITS OR IT MAY NEVER "HOLD". IF IT IS NOT RESPONDING AND HOLDING IMPROVEMENT START TO LOOK AT ADVANCED MARKERS (LABS, IMAGING ETC) IF YOU HAVEN'T ALREADY!

USE YOUR LASER AS A DIAGNOSTIC TOOL USING "UPSTREAM" OR "DOWNSTREAM" APPLICATIONS WITH PRE AND POST OUTCOME ASSESSMENTS! without permission of Trever Berry, DC, DACNE IS prohibited. **CEREBELLAR TESTING (FINGER TO NOSE, DYSDIADOCHOKINESIA, HEEL/SHIN ETC)**

FRONTAL (FINGER TAPPING, SACCADES)

RHOMBERG'S

WHERE TO LASER???

NERVE ROOT UP AND DOWN-REGULATION USING RED AND VIOLET LASERS

ANTAGONISTIC ROM

THE LASER DIAGNOSTIC PROTOCOL... "SWIM UP STREAM". PERIPHERAL TISSUE - N. ROOT - IPSI VAGAL STIM - IPSI CEREBELLUM - CONTRALATERAL FRONTAL/PARIETAL

CEREBELLUM - CONTRALATERAL FRONTAL/PARIETAL

VAGAL NERVE STIMULATION

TRANSCRANIAL DIRECT CURRENT VAGAL STIMULATION ->

VNS Placement Consider with all Brain-Gut Digestive/Seizure/Migraine CONCHA

NTS/LC Placement Consider for Arousal/Focus/Attention Consider avoiding for nausea TRAGUS







ERCHONIA

TRAUMATIC BERGER BOACNE is prohibited URY

...up to 50% of patients sustaining mTBI received an inaccurate emergency department diagnosis. Poor identification likely impacts clinical management of these patients.

 Marin JR, Weaver MD, Yealy DM, et al.. Trends in visits for traumatic brain injury to emergency departments in the United States. *JAMA* 2014;311:1917–9. 10.1001/jama.2014.3979 [PubMed] [CrossRef] [Google Scholar]
 Gaw CE, Zonfrillo MR. Emergency department visits for head trauma in the United States. *BMC Emerg Med* 2016;16:5 10.1186/ s12873-016-0071-8 [PMC free article] [PubMed] [CrossRef] [Google Scholar]



799

TRAUMATIC BRAIN INJURY

- Around 1.7 million Americans sustain a TBI every year.
- Automobile accidents are the #2 cause behind falls.
- About 1,365,000 Americans are treated for a TBI and released from an emergency department annually.
- An estimated 5.4 million people in the US live with disabilities associated with a TBI.
- Without support, 75% of persons in the US with a TBI lose their jobs within 90 days of returning to work.
- The lifetime cost for each severe TBI survivor is estimated to be more than \$4 million.
- <u>About 80% of all TBI cases are categorized</u> as a mild TBI.





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Conclusions and relevance: In this cohort study of more than 350,000 veterans, even mild TBI without LOC was associated with more than a 2-fold increase in the risk of

dementia diagnosis. Studies of strategies to determine mechanisms, prevention, and treatment of TBI-related dementia in veterans are urgently needed.

JAMA Neurology

2018 Sep 1;75(9):1055-1061. doi: 10.1001/jamaneurol.2018.0815.

Association of Mild Traumatic Brain Injury With and Without Loss of Consciousness With Dementia in US Military Veterans

Deborah E Barnes 123, Amy L Byers 123, Raquel C Gardner 14, Karen H Seal 125, W John Boscardin 15, Kristine Yaffe 1234 • PMID: 29801145 PMCID: PMC6143113 DOI: 10.1001/jamaneurol.2018.0815



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TRAUMATIC BRAIN INJURY

Average Auto Accident Settlement

The average settlement agreement is \$15,443 for accidents with physical injuries. For property damage only, the average settlement is \$3,231. The final amount depends

on the losses of the victim, the insurance policy of the responsible party, and the laws that apply to the case.

In 2020, *Jury Verdict Research* found the average TBI personal injury verdict in California was \$1.81 million... the median damage award was \$114,000.

1Cost of Auto Crashes and Statistics. Rocky Mountain Insurance Information Association. Retrieved 26 May 2022.



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TRAUMATE C BRAIN of the material in whole o INJURY

ERCHONIA



INTERDISCIPLINARY AND MULTI-MODAL APPROACHES = BETTER OUTCOMES

TRAUMATIC BRAIN INJURY

COMMON SHORTCOMINGS OF MTBI AND POST-CONCUSSION SYNDROME:

-MISSED DIAGNOSIS OR NOT UNDERSTANDING MTBI

-LACK OF OBJECTIVE BIOMARKERS FOR DIAGNOSIS AND MEDICAL-LEGAL SUBSTANTIATION

-THE "I HOPE THOSE NEUROLOGICAL SYMPTOMS GO AWAY ON THEIR OWN" APPROACH

-EFFICACIOUS TREATMENT STRATEGIES FOR BRAIN INJURIES

-A SYSTEM TO ADDRESS ALL OF THE ABOVE



SUMMARY OF OBJECTIVE BIOMARKER DIAGNOSTIC TESTING:

- VIDEONYSTAGMOGRAPHY (VNG)
- COMPUTERIZED DYNAMIC POSTUROGRAPHY (CDP-CTSIB)
- CNS-VS: COMPUTERIZED VITAL SIGNS DURING COGNITIVE SKILLS
- NEUROPSYCH AND SOMATIC SCREENS/ QUESTIONNAIRES/SCALES/INDICES
- DIFFUSION TENSOR IMAGING, CT, XRAYS
- LABS?



TRAUMATIC BRAIN INJURY

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TREATMENT SOLUTIONS: WHAT ONE TREATMENT MODALITY HAS THE POTENTIAL TO HELP THE FOLLOWING TBI SIGNS AND SYMPTOMS?



SOMATIC DOMAIN SYMPTOMS

- EYE MOVEMENTS
- BALANCE/DIZZINESS
- HEADACHE
- PAIN
- VISION
- AUDITORY PROCESSING
- TACTILE PROCESSING
- SMELL
- TASTE
- SLEEP
- FATIGUE



PSYCHIATRIC DOMAIN SYMPTOMS

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- DEPRESSION
- ANXIETY
- POST-TRAUMATIC STRESS
- BEHAVIORAL DYSREGULATION

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COGNITIVE DOMAIN SYMPTOMS

- MEMORY
- ATTENTION
- EXECUTIVE FUNCTIONING
- SPEED OF PROCESSING
- LANGUAGE





LASER THERAPY AND ACUTE TBI



J Neurotrauma

2023 Feb;40(3-4):210-227. doi: 10.1089/neu.2022.0140. Epub 2022 Aug 30.

Photobiomodulation in Acute Traumatic Brain Injury: A Systematic Review and Meta-Analysis

Andrew Robert Stevens 1

Abstract

Photobiomodulation (PBM) is a therapeutic modality that has gained increasing interest in neuroscience applications, including acute traumatic brain injury (TBI). Its proposed mechanisms for therapeutic effect when delivered to the injured brain include antiapoptotic and antiinflammatory effects. This systematic review summarizes the available evidence for the value of PBM in improving outcomes in acute TBI and presents a meta-analysis of the pre-clinical evidence for neurological severity score (NSS) and lesion size in animal models of TBI. A systematic review of the literature was performed, with searches and data extraction performed independently in duplicate by two authors. Eighteen published articles were identified for inclusion: seventeen pre-clinical studies of *in vivo* animal models and one clinical study in humar patients.

Mechanistic substudies within included *in vivo* studies are presented and were found to support hypotheses of antiapoptotic, anti-inflammatory, and proproliferative effects, and a modulation of cellular

metabolism. This systematic review provides substantial meta-analysis evidence of the benefits of PBM on functional and histological outcomes of TBI in *in vivo* mammalian models. Study design and PBM parameters should be closely considered for future human clinical studies.



September 14, 2020

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, MD, MSc1; Can Ozan Tan, PhD12; Suk-tak Chan, PhD13; et al

JAMA Netw Open. 2020;3(9):e2017337. doi:10.1001/jamanetworkopen.2020.17337

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.

1.3 J/CM2 TO THE BRAIN TISSUE

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OF THE NEARLY 12,000 PUBLISHED, PEER-REVIEWED PAPERS ON LOW LEVEL LASER THERAPY, HERE IS A SMALL SAMPLE OF MTBI AND POST-CONCUSSION RELATED APPLICATIONS LASERS CAN BE USED FOR:

-BRAIN INFLAMMATION (ACUTE AND CHRONIC GLIAL RESOLUTION) -LEAKY GUT AND BLOOD BRAIN BARRIER -ALL MUSCULOSKELETAL INJURIES -PAIN -HEADACHES -ANXIETY AND PTSD -DEPRESSION -COGNITION AND EXECUTIVE FUNCTION -HP-AXIS SUPPORT -ENERGY PRODUCTION, MITOCHONDRIAL OUTPUT AND BIOGENESIS -GLUTAMATE EXCITOXICITY, APOPTOSIS AND SPREADING EFFECT (PRE-TREAT!) -SLEEP, RELAXATION, VAGAL NERVE STIMULATION -BDNF AND NEUROGENESIS -AND MUCH MORE!

WHAT'S THE CATCH...



...FINDING A TRUE LASER THAT DOES NOT EXCEED 15 J/ CM2 TO THE BRAIN

ERCHONIA LOW LEVEL LASERS - THE NEUROLOGICAL JEDI LIGHT SABER FOR <u>ALL</u> HEALTHCARE SPECIALTIES

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Photomed Laser Surg. 2016 Dec 1; 34(12): 587–598. Published online 2016 Dec 1. doi: <u>10.1089/pho.2015.4051</u>

Transcranial Low-Level Laser (Light) Therapy for Brain Injury

Connor Thunshelle1,,2 and Michael R. Hamblin, PhD 22,,3,4 Conclusions

tLLLT has strong evidence for many beneficial effects on TBI and stroke in both animal models and human patients. Both acute stroke and acute TBI have a growing number of studies being published showing that tLLLT is an effective means of treating both. The many benefits of tLLLT are thought to be based on several different biological mechanisms. Nearinfrared PBM functions by improving mitochondrial energy production by stimulating the enzyme CCO and increasing ATP synthesis. Laser therapy can result in neuroprotection and help prevent the spread of brain cell death after injury as shown by the long-term **development of smaller lesion areas** in mice treated with LLLT, when delivered at 4 h post-TBI. Protection against toxins, increased cellular proliferation, and reduction in apoptosis and anti-inflammatory and antiedema effects may also contribute to the mechanisms that underlie the beneficial effects of PBM. The most exciting prospect is the possibility that tLLLT may stimulate both neurogenesis (the ability of the brain to repair itself) and synaptogenesis (encourage cells to form new synaptic connections). This could lead to the application of **tLLLT** as a treatment modality for neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Well-funded, controlled studies are necessary.



Mol Neurobiol. 2016 Mar;53(2):1181-94. doi: 10.1007/s12035-014-9070-5. Epub 2015 Jan 20.

Differential Roles of M1 and M2 Microglia in Neurodegenerative Diseases.

IN SUMMARY, WE NEED M1 FOR ACUTE PHASE BUT WE NEED RESOLUTION, ANTI-INFLAMMATORY, NEUROGENESIS AND NEUROPROTECTIVE EFFECTS OF M2.

Exp Neurol. 2018 Jan;299(Pt A):86-96. doi: 10.1016/j.expneurol.2017.10.013. Epub 2017 Oct 19.

Photobiomodulation therapy promotes neurogenesis by improving poststroke local microenvironment and stimulating neuroprogenitor cells. Yang L1, Tucker D2, Dong Y2, Wu C3, Lu Y2, Li Y2, Zhang J2, Liu TC3, Zhang Q4.

Intriguingly, PBM could effectively switch an M1 microglial phenotype to an antiinflammatory M2 phenotype. Our novel findings

indicated that PBM isocapable and promoting neurogenesis rafter ischemic stroke. without permission of Trevor Berry, DC, DACNB is prohibited. Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



Stimulating the Central Nervous System to Prevent Intestinal Dysfunction After **Traumatic Brain Injury**

A. Leaky gut 6 hours after TBI with no vagal stimulation

B. Tight junction integrity 6 hours after TBI with vagal stimulation



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 permeability⁸¹⁷ IN DIFFERENT CONDITIONS.

ASSESSING THE AUTONOMIC **EFFECT OF 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



JAffect 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.

MA1. Cassano P2 Author information Abstract 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 (Major Depressive

Disorder). Future studies should clarify the ideal stimulation parameters as well as the overall efficacy, effectiveness and safety profile of this treatment.

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

Abstract

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 in been without permission of Trevor Berry, DC, DACNB is prohibited.



In TBI patients, abnormalities in higher level cognitive activities are associated with weak connections within and between the DMN, SN and CEN nodes, resulting in impaired dynamic interactions of these networks [179,180]. There is a

hypothesis that the geographical matching of light irradiation sites on the head, with the corresponding anatomical regions of intrinsic networks within the brain may allow re-establishment of these functions, and may have enhanced therapeutic benefit [174]. In this respect,

Naeser et al. [30] reported findings in TBI patients who received **transcranial therapy over**

the DMN, SN, and CEN nodes and displayed enhanced cognitive functions, likely through the

augmentation of metabolic capacity in these intrinsic networks. Moreover, Naeser et al proposed that the ability of PBM to reduce PTSD symptoms might stem from the modulation of DMN and SN activities [30]. On the

other hand, application of PBM in stroke patients with aphasia demonstrated neurotherapeutic efficacy through the stimulation of cortical nodes within the CEN network [181].

Published online 2018 Jan 11. doi:PMCID: PMC6041198NIHMSID: NIHMS934240PMID:

Brain Photobiomodulation Therapy: A Narrative Review



Abstract

This is the first controlled study demonstrating the beneficial effects of transcranial laser stimulation on cognitive and emotional functions in humans... The Positive and

Negative Affect Schedule (PANAS-X), which tracks self-reported positive and negative affective (emotional) states over time, was administered immediately before treatment and 2 weeks after treatment.

The PANAS showed that while participants generally reported more positive affective states than negative, **overall affect** *improved significantly in the treated group due to more sustained positive emotional states as compared to the placebo control group. These data imply that transcranial laser stimulation could be used as a non-invasive and efficacious approach to increase brain functions such as those related to cognitive and emotional dimensions.* Transcranial infrared laser

stimulation has also been proven to be safe and successful at improving neurological outcome in humans in controlled clinical trials of stroke. This innovative approach could lead to the development of non-invasive, performance-enhancing interventions in healthy humans and in those in need of neuropsychological rehabilitation.

Randomized Controlled Trial

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

D W Barrett 1, F Gonzalez-Lima

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TCT LASER APPLICATION FOR DEPRESSION

JAffect 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.

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



NEUROPSYCHOLOGY 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

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

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

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

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 Að 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 (Figure 4). The cell bodies of neurons lie within the dorsal nerve root ganglion, but the elongated cytoplasm (axons) of the neurons extends from the cell body to the bare nerve endings in the surface of the skin. The direct effect of LLLT are initially at the level of the epidermal neural network, but the effects move to nerves in subcutaneous tissues, sympathetic ganglia, and the neuromuscular junctions within muscles and nerve trunks.

PRE-TREATING WITH TRANSCRANIAL LOW LEVEL LASER THERAPY TO IMPROVE BRAIN INJURY OUTCOMES

<u>Dose Response.</u> 2014 Sep 22;12(4):619-49. doi: 10.2203/dose-response.14-032.Agrawal. **Pre-conditioning with low-level laser (light) therapy: light before the storm.** <u>Agrawal T</u>1, <u>Gupta GK2</u>, <u>Rai V</u>3, <u>Carroll JD</u>4, <u>Hamblin MR</u>5.

Recently it has become apparent that LLLT can also be effective if delivered to normal cells or tissue before the actual insult or trauma, in a pre-conditioning mode. Muscles are protected, nerves feel less pain, and LLLT can protect against a subsequent heart attack. These examples point the way to wider use of LLLT as a pre-conditioning modality to

The damage caused by surgery, heart attack, or stroke can be mitigated by pre-treating the local or distant tissue with Lowlevel laser (light) therapy (LLLT).

J Biomed Opt. 2014;19(10):108003. doi: 10.1117/1.JBO.19.10.108003. **Transcranial low-level laser therapy enhances learning, memory, and neuroprogenitor cells after traumatic brain injury in mice.** Xuan W1. Vatansever F2, Huang L3, Hamblin MR4.

Our study results suggest that tLLLT may improve TBI both by reducing cell death in the lesion and by stimulating neurogenesis. Duplication, citation or distribution of this material in whole or in part

PRECONDITIONING WITH TRANSCRANIAL OW LEVEL LASER AND MAGNESIUM TO IMPROVE BRAIN INJURY OUTCOMES

Preconditioning with transcranial low-level light therapy reduces neuroinflammation and protects blood-brain barrier after focal cerebral ischemia in mice. Lee et al. Restor Neurol Neurosci. 2016;34(2):201-14.

3LT applied 2x per day for 2 days prior to photothrombic cortical ischemia. Laser treated had significant decrease in infarct size and edema and improved neurological and motor status 24 hours post ischemic injury. It protected the BBB and decreased perifocal spreading of damage via leukocyte infiltration. Inflammatory markers MAPK and NF-kB were significantly reduced in the ischemic cortex!



SLEEP



Neuropsychopharmacology. **2020** Jan; 45(1): 205–216. Published online 2019 Jun 17. doi: <u>10.1038/s41386-019-0439-z</u>

Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications Monika Haack, 1.2 Norah Simpson, 3 Navil Sethna, 2.4 Satvinder Kaur, 1.2 and Janet Mullington 1,2

Pain can be both a cause and a consequence of sleep deficiency. This bidirectional relationship between sleep and pain has important

implications for clinical management of patients, but also for chronic pain prevention and public health more broadly. The

review that follows will provide an overview of the neurobiological evidence of *mechanisms thought to be involved in the modulation of pain by sleep deficiency, including the opioid, monoaminergic, orexinergic, immune, melatonin, and endocannabinoid systems; the hypothalamus-pituitary-adrenal axis; and adenosine and nitric oxide signaling.* In addition, it will provide a broad overview of pharmacological and non-pharmacological approaches for the management of chronic pain comorbid with sleep disturbances and for the management of postoperative pain, as well as discuss the effects of sleep-disturbing metheric of the management of the manage



Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications Monika Haack, 1,2 Norah Simpson, 3 Navil Sethna, 2,4 Satvinder Kaur, 1,2 and Janet Mullington 1,2

SUMMARY OF DRUGS OR OTHER CHEMICAL MEDIATORS THAT ARE COMMONLY USED IN PAIN MANAGEMENT THAT HAVE SHOWN IN RESEARCH TO PROMOTE SLEEP DISRUPTION THEREBY CONTRIBUTING TO THE CHRONIC PAIN **MECHANISMS:**

-CORTICOSTERIODS (HP AXIS IMBALANCE, INHIBIT MELATONIN AND GABA)

- -OPIOIDS (DECREASED REM AND ENDOGENOUS RECEPTOR SENSITIVITY)
- -NOREPINEPHRINE AGONISTS (PROMOTE LC/PAG TO INHIBIT PAIN, BUT THESE INHIBIT SLEEP BECAUSE THEY PROMOTE AROUSAL. FIGHT OR FLIGHT READINESS ETC. EX. INCREASED OUTPUT IN THESE AREAS WHEN GETTING KICKED IN THE SHIN BUT NOT GOING TO BE ABLE TO SLEEP)
- -DOPAMINE AGONISTS (ONCE AGAIN, THESE CAN HELP PAIN INHIBITION BY DAY BUT THEIR EFFECT ON D2 RECEPTORS IN THE NUCLEUS ACCUMBENS ALTERS THE SLEEP-WAKE CYCLE NEGATIVELY)
- -SEROTONIN (UNDETERMINED AT THIS TIME: SEROTONIN GOOD FOR CENTRAL AND PERIPHERAL PAIN INHIBITION BUT VARIOUS RECEPTOR TYPES OFFER **DIFFERING EFFECTS ON SLEEP). TRY MELATONIN INSTEAD**
- -ADENOSINE (IE. CAFFEINE. HAS GOOD ANALGESIC EFFECTS IN SLEEP **DEPRIVATION AND POST-OPERATIVE PAIN BUT ALSO PROMOTES WAKEFULNESS)**

827

SLEEP



Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications Monika Haack, 1,2 Norah Simpson, 3 Navil Sethna, 2,4 Satvinder Kaur, 1,2 and Janet Mullington 1,2

SUMMARY OF DRUGS OR OTHER CHEMICAL MEDIATORS THAT ARE COMMONLY USED IN PAIN MANAGEMENT THAT HAVE SHOWN IN RESEARCH TO PROMOTE SLEEP DISRUPTION THEREBY CONTRIBUTING TO THE CHRONIC PAIN MECHANISMS:

- -nNOS GOOD FOR PAIN BUT INOS EXCESS IN CNS BAD FOR SLEEP (ARGININE VS **VINPOCETINE**)
- -OREXIN IMBALANCE (OREXINS ARE AN INTEGRAL PART OF SLEEP-WAKE AND ANALGESIC EFFECTS. STIMULANTS BY DAY LIKE COFFEE/TEA EXTRACTS, VAGAL STIM AND ADAPTOGENS BY NIGHT LIKE ASHWAGANDHA, BANABA LEAF, **BERBERINE, LICORICE, L-THEANINE)**
- -HPA AXIS (ALTHOUGH CORTISOL WILL INHIBIT PRO-INFLAMMATORY CYOTKINES LIKE IL6 AND IL-1B, HPA DYSFUNCTION CAN LEAD TO IMMUNE SYSTEM IMBALANCE CAUSING A STATE OF LOW GRADE INFLAMMATION AND WAKEFULNESS)
- -ANYTHING CAUSING CHRONIC IMMUNORESPONSES AND PRO-INFLAMMATORY CYTOKINES (IE.IL6, IL-1B, TNF ALPHA, PGE2. EXAMPLES MAY INCLUDE SAD/HIGH SUGAR DIETS, FOOD ALLERGIES/SENSITIVITIES, CHRONIC PATHOGEN LOADS = INCREASE MRA) Duplication, citation or distributio#2% f this material in whole or in part



<u>J Healthc Eng.</u> 2017; 2017: 6030268. Published online 2017 May 11. doi: <u>10.1155/2017/603026</u>

PMCID: PMC5443998 PMID: <u>29065627</u>

Effect of Color Light Stimulation Using LED on Sleep Induction Time

Seonjin Lee 1 and Dongwook Kim 2, 3,*

Color	Time to fall asleep
white	21.2 minutes
darkness	21.1 minutes
random nonpreferred color	16.8 minutes
preferred color	12.3 minutes

SLEEP

Red light

It's thought that colors close to red on the light spectrum might stimulate melatonin production. However, at this time, more research is needed to support this theory. A Study from 2012 examined the effect of 30 minutes of nighttime red light

therapy in a group of 20 female basketball players. The researchers found that after 14 days, participants who received RED light therapy improved their sleep quality and melatonin levels significantly compared to participants not exposed to any light. One 2017 on mice found that red light at an intensity of 10 lux or higher had the potential to induce sleep.

<u>J Athl Train.</u> 2012 Nov-Dec; 47(6): 673–678. doi: <u>10.4085/1062-6050-47.6.08</u>

PMCID: PMC3499892 PMID: 23182016

Red Light and the Sleep Quality and Endurance Performance of Chinese Female Basketball Players ⁸²⁹ Jiexiu Zhao, PhD,* Ye Tian, PhD,* Jinlei Nie, PhD,† Jincheng Xu, MSS,‡ and Dongsen Liu, MS‡

RSV VACCINE AND GUILLAIN-BARRE RISKS



FDA declares that rare neurological disorder Guillain-Barré syndrome (GBS) is an important potential risk of Pfizer's RSV vaccine

FDA wrote: 'Given the temporal association and biological plausibility, FDA agrees with the assessments of the investigators that these events were possibly related to study vaccine.'

SOURCE:



https://www.fda.gov/media/165623/download

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What's the difference between a conspiracy theory and the truth?

About 6-12 months

DISCLAIMER: THESE ARE NOT CLAIMS TO TREAT OR CURE ANY MEDICAL CONDITION. LOW LEVEL LASER, NUTRITION SUPPORT OR ANY OTHER CAM THERAPIES ARE TO SUPPORT NORMAL ADAPTIVE PHYSIOLOGY OF THE HUMAN BODY.

IMMUNOLOGY AND PATHOGENS



In a startling disclosure at the EU COVI Hearings in Brussels on the 10th of October 2022, Janine Small admitted in response to a question posed by Dutch MEP Robert Roos that the Pfizer mRNA vaccine was never tested or shown before its release, to impact the transmission of the SARS-NCOV2 virus. In other words, whether the vaccine did, prevent transmission of Covid from one person to another. You can follow the entire hearing via the EU Media Center or watch the embedded video below.



Vaccinated people now make majority of **COVID** deaths in US: Report

"In September 2021, vaccinated people made up just 23 per cent of coronavirus fatalities. In January and February this year, it was up to 42 per cent," the report mentioned.

"We can no longer say this is a pandemic of the unvaccinated," said Kaiser Family Foundation vice president Cynthia Cox, who conducted the analysis on behalf of the Washington Post.

IMMUNOLOGY AND PATHOGENS

ERCHONIA

PFIZER AND RESEARCHERS HAVE CONFIRMED THAT COVID VACCINES HAVE NOT BEEN SHOWN TO PREVENT THE SPREAD OF THE DISEASE. **INSTEAD THEY TURNED THEIR FOCUS ON STATING VACCINES** DECREASE SYMPTOM SEVERITY AND THEY PROTECT THE **IMMUNOCOMPRIMISED ELDERLY, YET...**

More vaccinated people who catch Covid are reporting same four symptoms

A study has found that each new variant causes more people to suffer symptoms which disrupt their daily lives People who have had three doses of the Covid vaccine but still go on to catch the virus are reporting similar severe cold-like symptoms, a study has found. People say the symptoms are so severe they are affecting their day-to-day life and activities.

Each new variant of the virus sees more and more people reporting disruptive symptoms, a new study found.

Researchers at Imperial College London spoke to 17,000 people who tested positive for Covid between May 2020 and March 2022 as part of the Real-Time Assessment of Community Transmission (REACT) study. The study found that more than 5,000 vaccinated people who were infected with recent strains of Covid suffered symptoms that affected their day-to-day lives.

Open Access Published: 11 November 2022

Variant-specific symptoms of COVID-19 in a study of 1,542,510 adults in England

 Matthew Whitaker, Joshua Elliott, Barbara Bodinier, Wendy Barclay, Helen Ward, Graham Cooke, Christl A. Donnelly, Marc Chadeau-Hyam & Paul Elliott Nature Communications Duplication, citation or distribution of this material in whole or in part volume

13. Article number: 6856 (2022) Cite this article without permission of Trevor Berry, DC, DACNB is prohibited.



Study shows Pfizer vaccine effectiveness decreases with age

UT Southwestern researchers found that antibodies generated from the vaccine in people over 65 were less effective at performing antiviral functions than in younger adults.

"The fact that these antibody functions decrease with age is one reason why the elderly are still more susceptible to severe illness with COVID-19 and highlights the need to develop different approaches for older, vulnerable individuals," said lead author Dr. Lenette Lu, assistant professor of internal medicine and immunology in the UT Southwestern Division of Infectious Diseases and Geographic Medicine.

Nearly three years after the beginning of the pandemic, COVID-19 finally appears to be easing its grip on the U.S. health care system. Hospitalizations from the flu surpassed COVID-19 hospitalizations in North Texas on Monday, according to Dallas-Fort Worth Hospital Council data.

But the virus, first discovered in 2019, can still wreak havoc on the elderly and people with compromised immune systems. Nearly 460 patients were hospitalized with COVID-19 in Dallas-Fort Worth as of Tuesday.

COVID AND MASKS



Rigorous international study of N95 masks upends federal COVID narrative

Biden's former COVID adviser tries to discredit randomized controlled trial that found no statistically significant real-world performance difference between surgical masks, N95s. Fauci reportedly can't cite any evidence masks work in deposition.

Published in the Annals of Internal Medicine (AIM) this week and led by researchers at Canada's McMaster University, the study found no statistically significant difference in protection between the two kinds of masks in healthcare facilities in Canada. Israel, Pakistan and Egypt.

Funders included the Canadian government and World Health Organization, which unlike the U.S. opposes masking young children and also found "no evidence" face coverings made a difference against influenza in a 2019 study.

Fauci also couldn't cite what "studies or study changed his mind in that interim" between his private opposition to masking in February 2020 and public embrace of masking in April.

29 November 2022, ANNALS OF INTERNAL MEDICINE Medical Masks Versus N95 Respirators for Preventing COVID-19 Among Health Care Workers A Randomized Trial

COVID AND MASKS



Critics assail widely touted study on mask effectiveness against COVID

"Based on the 95% confidence intervals, we do not even know if surgical mask efficacy is more than 0%," Harvard Med professor says.

A much larger Bangladeshi study led by Yale researchers found that villages that got surgical masks reduced symptomatic infection by 11%, but the

confidence interval hit zero and statistical significance disappeared for every age group under 50. Cloth masks had an "imprecise zero" effect.

The infection rate in the N95 group was 9.27%, and the surgical, 10.46%. The former suffered a slightly higher rate of adverse events — discomfort, headache and skin irritation — from wearing the coverings (13.6%) than did the latter (10.8%).

"If N95s are more effective in long Twitter rants (EBM+) than in pragmatic randomized controlled trials (EBM), it might be time to move on and focus on other interventions," Johns Hopkins epidemiologist Stefan Baral tweeted, referring to evidence-based medicine supplemented by "mechanistic evidence" and true EBM.

"As one doctor told me, 'If you can wear an N95 for more than about 30 minutes you are wearing it wrong,'" tweeted George Mason University law professor Todd Zywicki, who successfully challenged GMU's COVID vaccine mandate in court.

The Impact of Community Masking on COVID-19: A Cluster-Randomized Trial in Bangladesh Jason Abaluck[†],[‡],

COVID AND MASKS



Reduction in COVID-19 Infection Using Surgical Facial Masks Outside the Healthcare System

Information provided by Henning Bundgaard, Rigshospitalet, Denmark (Responsible Party) Last Updated: July 29, 2020

The U.S. government does not review or approve the safety and science of all studies listed on this website.

Previous RCTs in the COVID era have found mediocre protection from face coverings at best. A Danish study of 6,000 that had trouble getting published – and eventually landed in AIM – found no statistically significant difference between surgical and no-mask wearers

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IMMUNOLOGY AND PATHOGENS



SHORTCOMINGS OF THE BIVALENT BOOSTER:

- 1. THE FEDS PURCHASED 171 MILLION DOSES. YET, ONLY 1 IN 9 AMERICANS HAVE RECEIVED IT
- 2. WE ARE NOW HEARING STUDIES THAT UNVACCINATED PEOPLE ARE MORE LIKELY TO HAVE CAR CRASHES!!??

COVID Vaccine Hesitancy and Risk of a Traffic Crash

Donald A. Redelmeier, MD, FRCPC, MSHSR, FACP

Jonathan Wang, MMASc Deva Thiruchelvam, MSc Published:December 02, 2022

3. THE FEDS PUMPED ANOTHER HALF BILLION INTO VACCINE/BOOSTER PROMOTION IN DECEMBER

4. THE BOOSTER WAS APPROVED FOR USE IN HUMANS AFTER A VERY SHORT TRIAL ON 8 MICE

5. APPROXIMATELY 1 IN 5000 MEN UNDER THE AGE OF 40 ARE DEVELOPING LIFE THREATENING MYOCARDITIS. THE DEATH RATES ARE 0.035% AND IS TYPICALLY ONLY FOUND WITH CO-MORBIDITIES LIKE OBESITY, DIABETES AND IMMUNOCOMPROMISED

6. THE BIVALENT EFFECTIVENESS IS 22% FOR THOSE OVER THE AGE OF 65, WHEREAS IT WAS STATED TO BE 90% EFFECTIVE AT IT'S INCEPTION

"If I believed that the vaccine was truly safe and effective I would get it; I am unconvinced that it is safe, and it is only modestly effective. I suspect that the risks per individual are low but non-zero, but so are COVID's. And I know beyond the shadow of a doubt that after the vaccine I can still get COVID and the risks associated with it. Why double up two small risks?" DAVID STROM, BIVALENT BOOSTER ARTICLE 12/14/22

IMMUNOLOGY AND PATHOGENS



'Pfizer and Moderna better get this clarified': Study finds worse antibodies after mRNA boosters

Increased proportion of post-booster IgG4 antibodies, known for helping humans adapt to allergies, "might result in longer viral persistence in case of infection," German researchers conclude. Italian study found IgG4 concentration correlated with COVID-related mortality.

A new peer-reviewed study is raising concerns that a third dose of Pfizer and Moderna vaccines may actually worsen immune response against COVID-19, creating waves in both mainstream and heterodox COVID circles.

The fourth subclass started ramping up several months after the full series and **reached a high of 19.27% "late after the third vaccination."**

"Importantly, this class switch was associated with a reduced capacity of the spike-specific antibodies to mediate antibodydependent cellular phagocytosis [ingesting and eliminating pathogens] and complement deposition," the study's introduction says.

Repeat covid-19 mRNA vaccination (boostering) was associated with a "class switch" to antibodies (Ig4) that have a potentially lower capacity to neutralize SARS-CoV-2, & clinical "breakthrough" infections

Class switch towards non-inflammatory, spike-specific IgG4 antibodies after repeated SARS-CoV-2 mRNA vaccination

PASCAL IRRGANG SCIENCE IMMUNOLOGY

22 Dec 2022 First Release

DOI: 10.1126/sciimmunol.ade2798



CDC admits natural immunity more protective than vax, yet hypes ICU, other perils for unvaxxed kids

Agency's assertion that vaccine prevents severe disease "is not based on the randomized trial evidence," Stanford Med professor says.

The CDC's Morbidity and Mortality Weekly Report last week found that COVID recovery became more protective against reinfection and hospitalization than "recent" vaccination alone once the Delta variant became dominant.

"The report finally acknowledges what many have suspected for a long time — that surviving COVID-19 provides excellent natural immunity not only [to] repeat infection but also to hospitalization and death for the delta variant of COVID-19," University of Southern California clinical medical professor Jeffrey Klausner and UCLA Health senior resident Noah Kojima wrote this week in The Hill.

"The pattern of improved protection after natural infection makes sense," they explain, **because natural infection exposes the body to "all parts of the virus" while vaccines target COVID's spike protein.** The CDC's tweet falsely leads parents to think "1/3rd of all kids who get COVID before the vax ... will be hospitalized and have severe disease," he wrote in an email. "The randomized trials in kids did not establish that vax prevents severe disease in children. The CDC's assertion that it does is not based on the randomized trial evidence."

Asked to respond to criticism of its portrayal of the severe pediatric COVID study, a CDC spokesperson referred Just the News to another spokesperson who has not responded. The lead author of the study, CDC researcher Rebecca Woodruff, referred Just the News to media relations but has not answered whether she was banned from talking to reporters.

Protective immunity after recovery from SARS-CoV-2 infection

Noah KojimaJeffrey D Klausner

Published:November 08, 2021DOI:<u>https://doi.org/10.1016/S1473-3099(21)00676-9</u> THE LANCET

COVID VACCINES



CORONAVIRUS (COVID-19)

Pfizer CEO Albert Bourla diagnosed with an unfortunate infection

Wednesday, August 17, 2022

If you've ever heard the advice "never get a haircut from a bald barber" you know that even something completely natural can tarnish the image of a brand in people's minds. So, much like being judged for being an overweight fitness instructor, it's a bit awkward for Albert Bourla that people now know he's been infected by Covid-19, considering he is the CEO of Pfizer.



CEO Bourla has received both vaccines as well as a first and even a second booster shot, all of the vaccines produced by his company Pfizer in association with BioNTech

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The CDC (Finallys) Aromitted the the Science on Natural Immunity. Why Did It Take so Long?Monday, August 22, 2022

n August 2021 *Science Magazine*, a peer-reviewed academic journal of the American Association for the Advancement of Science, highlighted groundbreaking research out of Israel that upended the public health establishment. The research, which relied on a database enrolling some 2.5 million Israelis and was led by Tal Patalon, head of the KSM Research and Innovation Center at

Maccabi Healthcare Services, and deputy Sivan Gazit, found that previous infection from Covid-19 conferred considerably stronger and longer-lasting protection against the Delta variant than vaccines. "never-infected people who were vaccinated in January and February were,

in June, July, and the first half of August, Six to 13 times more

likely to get infected than unvaccinated people" who had previously had Covid.

It's not an exaggeration to say that billions of dollars were at stake over whether Covid vaccines were mandatory or voluntary, which might explain why **Pfizer alone spent more than \$10** million lobbying in 2021 and even more in 2020.

Comparing SARS-CoV-2 natural immunity to vaccine-induced immunity: reinfections versus breakthrough infections Sivan Gazit, MD MA⁸⁴³,2*; Roei Shlezing

IMMUNOLOGY AND PATHOGENS



ERCHONIA

Local health stats show unvaxxed kids less likely to have COVID than vaccinated youth

Jul 19, 2022, 6:23 PM

Using figures based on every 100,000 people in King County, **if you have a 5**to 11-year-old and they are fully vaccinated, they are 46% more likely to catch or have COVID than unvaccinated 5to 11-year-olds.

What about local 12- to 15-year-olds? They're the youngest age group eligible for the booster. According to this same data, if your child is 12 to 15 and boosted, they are **16 percent more likely to get COVID compared to unvaccinated kids their same age.**

For 16- and 17-year-olds? They're 14 percent more likely to get COVID. This report appears to show that for every age range, children who are vaccinated and potentially boosted are more likely to get COVID than unvaccinated children.

COVID AND IQ DROP



Catching Covid can age your brain by 20 years and make your IQ drop, study finds People who have had a serious case of Covid may have seen their IQ levels drop by 10 points and had their brain age by 20 years, according to a new study

The study also suggests that these impacts are still noticeable more than six months after having the virus and that what recovery does happen is gradual.

Edward T. Bullmore, James B. Rowe, David K. Menon. **Multivariate profile and** acute-phase correlates of cognitive deficits in a COVID-19 hospitalised cohort. *eClinicalMedicine*, 2022; 47: 101417[®]OI: 10.1016/j.eclinm.2022.101417

CDC REPORT ON LONG COVID



Nearly One in Five American Adults Who Have Had COVID-19 Still Have "Long COVID"

<u>New data from the Household Pulse Survey</u> show that more than 40% of adults in the United States reported having COVID-19 in the past, and nearly one in five of those (19%) are currently still having symptoms of "long COVID."

The data were collected from June 1-June 13, 2022 by the U.S. Census Bureau and analyzed by CDC's National Center for Health Statistics (NCHS)

IMMUNE SUPPORT AND LELT COVID



Nervous system consequences of COVID-19

SCIENCE · 20 Jan 2022 · Vol 375, Issue 6578 ·

HIGHLIGHTS:

VERY LITTLE EVIDENCE OF ACTUAL COVID VIRUS PROTEINS FOUND IN THE CNS IE. SPIKE 2 OR NUCLEIC ACID PROTEINS suggesting that SARS-CoV-2 presence in the CNS does not incite classic viral encephalitis.

The most common neurologic conditions are anosmia (loss of smell), stroke, delirium, brain inflammation, encephalopathy, primary psychiatric syndromes, and peripheral nerve syndromes

Autopsy studies of patients with acute COVID-19 show infiltration of macrophages, CD8+ T lymphocytes in perivascular regions, and widespread microglial activation throughout the brain

TO PROTECT AGAINST THIS ACTIVE END STAGE INFLAMMATION DAMAGE (EX. INTERLEUKIN 1B, 6, 12, TNF ETC) AND PROMOTE PROPER IMMUNOREGULATION (EX. IL 4, 10, 13) TREAT THE CNS (TRANSCRANIAL AND VAGAL STIMULATION) WITH YOUR ERCHONIA DEVICE PREVENTATIVELY, DURING ACUTE AND SUB-ACUTE PHASES AND FOR LONG HAULERS

IMMUNE SUPPORT AND LLLT COVID



JOURNAL OF EXPERIMENTAL MEDICINE January 12 2021

Neuroinvasion of SARS-CoV-2 in human and mouse brain

A recent three-pronged study concludes that SARS-CoV-2 may infect nerve cells and impede blood flow in the central nervous system.

These findings suggest that the virus has the ability to alter cell metabolism to create an environment in which infected cells thrive and neighboring cells are unable to survive.

The organoid model also showed that the ACE2 receptor allowed the virus to enter brain cells, similarly to the way that this happens in the lungs. The result was surprising, as it had previously been unclear whether cells in the CNS produced ACE2 receptors.

Autopsies of COVID-19 patients

Finally, the researchers examined the brain regions of three patients who had died from severe COVID-19 complications. All had experienced respiratory failure and been admitted to the intensive care unit.

Within the infected brain regions, there were indications of tissue damage and cell death in the form of ischemic infarcts — areas of dead tissue caused by a lack of blood flow.

PROTECT THE BRAIN WITH RED OR VIOLET RED ERCHONIA LLLT ADVANCED BRAIN SETTING TRANSCRANIAL 1-10-40-60 FOLLOWED BY VAGAL STIM PROTOCOL 4 MIN SCM/GUT

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IMMUNE SUPPORT AND LELT COVID



Laser light with low-power intensity is applied to the surface of the skin to produce local and systemic effects. Based on the clinical

experience, peer-reviewed studies, and solid laboratory data in experimental animal models, LLLT attenuates cytokine storm at multiple levels and reduces the major inflammatory metabolites. LLLT is a safe, effective, low-cost modality without any side-effects that may be combined with conventional treatment of ARDS. We summarize the effects of LLLT on pulmonary inflammation and we provide a protocol for augmenting medical treatment in

COVID-19 patients. LLLT combined with conventional medical therapy has the potential to prevent the progression of COVID-19, minimize the length of time needed on a ventilator, enhance the healing process, and shorten recovery time.

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>

PMCID: PMC7428000 PMID: <u>32844112</u>

Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19

Soheila Mokmeli, MD Anesthesiologistta and Mariana Vetrici, MD, PhDa2

IMMUNE SUPPORT AND LLLT COVID



LLLT is an affordable modality compared with other treatments and medicines like IL-6 antagonists. LLLT is a safe, effective, low-cost modality without any reported side-effects compared with other approaches. A laser machine costs Can\$35,000.00–200,000.00, and each machine can fully treat 20,000 patients for COVID-19. In comparison, an IL-6 antagonist costs US\$1000.00 per injection, and each patient would need 3–6 injections for complete COVID-19 treatment. Treating 20,000 patients would cost US\$ 60,000,000.00–US\$ 120,000,000.00.

Based on this information, LLLT will accelerate recovery from COVID-19 and will get patients off ventilator support and out of the ICU more rapidly. This could significantly decompress our severely overburdened health care systems.

PMCID: PMC7428000

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>

PMID: <u>32844112</u> Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19



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IMMUNE SUPPORT AND LLLT COVID



Therapeutic technique and dosage of LLLT

Laser dose is the amount of energy delivered per second per cm2. The effect of laser therapy is related to the amount of laser energy per cm2. The Arndt-Schultz Law is considered the standard to describe the dose dependent effects of LLLT [42]. The minimum therapeutic dose for a bio-stimulatory effect for red and infrared laser is 0.01 J/cm2 while for ultraviolet, blue, green laser it is 0.001 J/

cm2. LLLT has a noticeable biphasic dose
response. The effective stimulation dose is 1 J/
cm2 on the target tissue. Doses greater than 10
J/cm2 produces inhibitory effects. The inhibitory effects are used in conditions requiring inhibition and suppression [2].

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>

PMCID: PMC7428000 PMID: <u>32844112</u>

Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19

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Soheila Mokmeli, MD Anesthesiologist
1 and Mariana Vetrici, MD, PhD
2

IMMUNE SUPPORT AND LLLT COVID



J Photochem Photobiol B

2021 Sep;222:112282. doi: 10.1016/j.jphotobiol.2021.112282. Epub 2021 Aug 8.

Pulsed blue light inactivates two strains of human coronavirus

Chukuka S Enwemeka 1, Violet V Bumah 2, John L Mokili 3

Therefore, we investigated the effect of 405 nm, 410 nm, 425 nm and 450 nm pulsed blue light (PBL) on human alpha coronavirus HCoV-229 E and human beta coronavirus HCoV-OC43

Like SARS-CoV-2, HCoV-229E and HCoV-OC43 are single stranded RNA viruses transmitted by air and direct contact; they have similar genomic sizes as SARS-CoV-2, and are used as surrogates for SARS-CoV-2. Irradiation was carried out either at 32.4 J cm-2 using 3 mW cm-2 irradiance or at 130 J cm-2 using 12 mW cm-2 irradiance. Results: (1) At each wavelength tested, PBL was antiviral against both

coronaviruses. (2) <u>405 nm light gave the best result</u>, yielding 52.3% (2.37 log10) inactivation against HCoV-OC43 (p < .0001), and a significant 1.46 log 10 (44%) inactivation of HCoV-229E (p < .01). HCoV-OC43, which like SARS-CoV-2 is a beta coronavirus, was more susceptible to PBL irradiation than

alpha coronavirus HCoV-229E. The latter finding suggests that PBL is potentially antiviral against multiple coronavirus strains, and that, while its potency may vary from one virus to another, it seems more antiviral against beta coronaviruses, such as HCoV-OC43. Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

IMMUNE SUPPORT AND LELT COVID



Abstract

A nonrandomized 50-person case study of COVID-19-positive patients was conducted employing (for the first time) a regimen of whole-organ deep-tissue transdermal dynamic photobiomodulation (PBM) as a primary (or exclusive) therapeutic modality

in the treatment of coronavirus. Therapy sessions comprised algorithmically alternating red (650 nm) and near-infrared (NIR; 850 nm) LEDs with an average irradiance of 11 mW/cm2 dynamically sequenced at multiple pulse frequencies. Delivered via 3D bendable polymeric pads maintaining orthogonal optical incidence to body contours over 1,000 cm2, a single 84-minute session concurrently delivered 20 kJ to the sinuses and 15 kJ to each lung at skin temperatures below 42°C. Therapeutic outcomes observed include significant reductions in the duration and severity of disease

symptoms. Acute conditions including fever, body aches (BA) and respiratory distress comprising paroxysmal coughing; lung congestion, dyspnea and hypoxia; sinus congestion; acute eye inflammation; and extreme malaise were eliminated in 41/50 patients within 4 days of commencing PBM treatments with 50/50 patients fully recovering within 3 weeks with no supplemental oxygen requirements. SpO2 concentrations improved as much as 9 points (average 2.5 points) across the entire study population. The PBM sessions required to completely resolve COVID– 19 conditions appears monotonically correlated to the time-to-treatment (TTTx) the delay between the onset of a patient's symptoms and commencing PBM therapy. In contrast, acute inflammatory symptoms were resolved within 4 days

<u>J Biophotonics.</u> 2021 Nov 22 : e202100194. doi: <u>10.1002/jbio.202100194</u> [Epub ahead of print]

PMCID: PMC8646787 PMID: <u>34658147</u>

Whole-organ transdermal photobiomodulation (PBM) of COVID-19: A 50-patient case study Richard K. Williams, 1 John Raimondo, 2 David Cahn, 3 Aldon Williams, 4 and Daniel Schell 5

IMMUNE SUPPORT AND LLLT COVID



Background: Because the major event in COVID-19 is the release of pre- and inflammatory cytokines, finding a reliable therapeutic strategy to inhibit this release, help patients manage organ damage and avoid ICU admission

or severe disease progression is of paramount importance. Photobiomodulation (PBM), based on numerous studies, may help in this regard, and the present study sought to evaluate the effects of said technology on cytokine reduction.

Results: During the study period, in PBM group, there was a significant decrease in serum levels of IL-6 (-82.5% +/- 4, P<0.001), IL-8 (-54.4% \pm 8, P<0.001), and TNF- α (-82.4% \pm 8, P<0.001), although we did not detect a significant change in IL-10 during the study. The IL-6/IL-10 Ratio also improved in PBM group. The Placebo group showed no decrease or even an increase in these parameters. There were no reported complications or sequelae due to PBM therapy throughout the study. LED Study

conclusion: The major cytokines in COVID-19 pathophysiology, including IL-6, IL-8, and TNF-α, responded positively to PBM therapy and opened a new window for inhibition

RED 635 NM DIODES

Front. Immunol., **08 July 2022** Sec. Cytokines and Soluble Mediators in Immunity

Photobiomodulation Improves Serum Cytokine Response in Mild to Moderate COVID-19: The First Randomized, Double-Blind, Placebo Controlled, Pilot Study

IMMUNOLOGY AND PATHOGENS



Int J Mol Sci. **2021 May;** 22(9): 4942. Published online 2021 May 6. doi: <u>10.3390/ijms22094942</u>

PMCID: PMC8124384 PMID: <u>34066560</u>

Probiotics, Photobiomodulation, and Disease Management: Controversies and Challenges

Laura Marinela Ailioaie1,2 and Gerhard Litscher3,*

GREAT REVIEW PAPER ON GUT MICROBIOME, LASER PHOTOBIOMODULATION (PBM), IMMUNOLOGY AND PATHOGENS. SOME OF THE KEY POINTS:

- LOW LEVEL LASER/PHOTOBIOMODULATION HAS BEEN SHOWN TO IMPROVE GUT MICROBIOME DIVERSITY
- PBM HAS BEEN SHOWN TO TO PROPERLY REGULATE IMMUNE RESPONSES INCLUDING IL-10, TGF-B
- PBM HAS AN ABSCOPAL (FAR REACHING SYSTEMIC) EFFECT ON PATHOGENS THROUGH MECHANISMS LIKE ACTIVATION OF CYTOTOXIC T CELLS
- THERE IS A HORMESIS IN PBM ON PATHOGENS, IMMUNITY AND GUT MICROBIOME ALTERATIONS SIMILAR TO NEUROLOGICAL CONDITIONS, CANCER, PAIN MANAGEMENT AND MORE

IMMUNE SUPPORT FOR VIRUS PROTOCOL





"VIRUS SETTING" 20-73-625-787

10 MINUTES TOTAL: 5 MINUTES TRANSCRANIAL 1 MINUTE NASAL/SINUSES 1 MINUTE THROAT, MAXILLARY AND MANDIBULAR GUM LINE 3 MINUTES TRACHEA DOWN TO THYMUS AND LUNG FIELDS AND GUT CONSIDER HANDS OR ANY OTHER POTENTIALLY

CONTAMINATED BODY SURFACES. GUT

ACUTE PHASE INFECTIOUS DISEASE MANAGEMENT EVRL OR FX 405 RECOMMENDED

ACUTE PHASE ACTIVE CARE: DAILY TO 3X/WEEK FOR MINIMUM 6 VISITS LASER RENTAL???

> ADD TO PREVENTATIVE/MAINTENANCE CARE ON ANY FOLLOW UP VISITS

BE AGGRESSIVE WITH BRAIN/BRAINSTEM/NASAL EXPOSURE WITH YOUR LASER FROM DAY 1 OF COVID TO CONFER PROTECTION AGAINS LONG COVID (LOSS OF SMELL, BRAIN FOG, IQ DECREASE, NEURODEGENERATION, DYSAUTONOMIA ETC) IMMUNOLOGY AND PATHOGENS



Potential New Treatment for "Brain Fog" in Long **COVID** Patients

FeaturedNeurologyNeuroscienceOpen Neuroscience Articles December 16, 2022

Arnsten has joined forces with Arman Fesharaki-Zadeh, MD, PhD, assistant professor of psychiatry and of neurology, who has been treating long COVID patients with a combination of guanfacine (an alpha2 adrenergic receptor agonist designed to relax blood vessels to improve blood flow. Has been applied

for ADHD and TBI) and N-acetylcysteine (NAC), an anti-

oxidant also used for the treatment of TBI.

CLINICAL CONSIDERATIONS FOR LONG COVID: LOW LEVEL LASER MASTER SETTING 1-10-40-60 **10 MINUTES TRANSCRANIAL PROTOCOL FOR RED, 6 FOR GREEN/VIOLET DIODES CONSIDER SINUS/NASAL EXPOSURE PLUS**

VAGAL STIMULATION PROTOCOL (2 MINUTES SCM/2 MINUTES GUT)

NUTRIENT SUPPORT:

NAC STACKED WITH S-ACETYL GLUTATHIONE, VINPOCETINE, CURCUMIN (BCM-95), OMEGA 3 WITH HIGH DHA (MAXSIM₃₄₇), VIRAGRAPHIS (ANDROGRAPHIS)

GLUTATHIONE

Glutathione Extends Lifespan in Animal studies by 24%

This compound seems to be valuable in controlling oxidative stress.

Conclusion

This study might put glutathione on the list of the most promising lifeextending molecules. GSH is known to be safe and is already marketed as a supplement,

*LOW LEVEL LASER THERAPY PROMOTES ALL FORMS OF ANTI-**OXIDANTS INCLUDING REDUCED GLUTATHIONE, CATALASE AND** SUPEROXIDE DISMUTASE!

Martirosyan D., Ashoori M. R., Mirmiranpour H. The effect of low level-laser irradiation on antioxidant enzymes and mineral levels in serum of atients with type 2 diabetes mellitus. Bioactive Compounds in Health and Disease. 2020; 3(5): 82-96. DOI: https://doi.org/10.31989/bchd.v3i5.705

CLINICAL CONSIDERATIONS TO IMPROVE REDUCED GLUTATHIONE (GSH) TO **OXIDIZED GLUTATHIONE (GSSG):**

1 AM MEDPAX S-ACETYL GLUTATHIONE (REDUCED GSH)



1 PM MEDPAX NAC (N-ACETYLE CYSTEINE) (GLUTATHIONE PRECURSOR AND RECYLING)



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[1] Kumar, P., Osahon, O. W., & Sekhar, R. V. (2022). GlyNAC (Glycine and N-Acetylcysteine) Supplementation in Mice Increases Length o Life by Correcting Glutathione Deficien Dublidation Stration Vorolistributed by of the sensing, and Genomic Damage. Nutrients, 14(5), 11 Without permission of Trevor Berry, DC, DACNB is prohibited.

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GLUTATHIONE



GLUTATHIONE'S ROLE IN INFLAMMATION:

- PROTECT DNA FROM FREE RADICAL DAMAGE
- DETOXIFICATION
- PROTECTS AGAINST OUR OWN WASTE PRODUCTS
- PROTECTS RBC'S FROM TOXINS AND FREE RADICALS
- PLAYS A ROLE IN VITAMIN C & E RECYCLING
- SUPPORTS TH1 DOMINANCE
- CONVERSELY, IF GLUTATHIONE IS DEPLETED IT WILL PROMOTE TH2 DOMINANCE

CLINICAL PEARL: GLUTATHIONE IS MADE UP OF 3 AMINO ACIDS: GLUTAMINE - GLYCINE - CYSTEINE

*** CYSTEINE IS THE MOST LIKELY OF THE 3 TO BE DEPLETED THEREBY THE LIMITING FACTOR IN MANUFACTURING. TGF-B NEEDS 9 CYSTEINE RESIDUES SO TH17 DOMINANCE "STEALS" CYSTEINE FROM THE BODY FOR TGF-B PRODUCTION LEAVING LESS FOR GLUTATHIONE***

(IF GLUTATHIONE IS HIGH IN ABSENCE OF SUPPLEMENTATION THINK INFLAMMATORY FREE RADICAL PROCESS WHERE THE BODY IS RAMPING UP PRODUCTION. IF THIS PROCESS STAYS CHRONIC EVENTUALLY GLUTATHIONE STORES WILL BE DEPLETED AND LAB READINGS WILL DROP)

GLUTATHIONE



CLINICAL PEARLS:

- WHEN USING TH1 STIMULATORS LIKE GLUTATHIONE THE PATIENT MAY EXPERIENCE BONE PAIN AND TH1 STIMULATORS NEED TO BE CUT BACK. YOU WILL OFTEN SEE THE PATIENT DO WELL INITIALLY ON GLUTATHIONE BUT THEN START TO EXPERIENCE THESE PAIN SYMPTOMS
- GLUTATHIONE IS CRITICAL FOR INNATE IMMUNE RESPONSES AND PROTECTION/ REPAIR OF BARRIER SYSTEMS (SINUS, LUNG, GUT, BRAIN). IV IS THE MOST BIOAVAILABLE IN THE SHORT TERM BUT STUDIES SHOW ORAL S-ACETYL GLUTATHIONE TO BE SUPERIOR AS TIME PROGRESSES AND IS MORE PRACTICAL FOR THE AVERAGE PATIENT. IT CAN BE NEBULIZED FOR SINUS/LUNG ABSORPTION.
- LIPOSOMAL TOPICAL GLUTATHIONE IS ALSO EFFECTIVE ESPECIALLY WHEN TARGETING SPECIFIC REGIONS OR ORGANS OF THE BODY, BUT IT MAY ALSO TRIGGER LOCALIZED IMMUNE RESPONSES.
- GLUTATHIONE CAN TRIGGER HISTAMINE RESPONSES SO BE CAREFUL IN MAST CELL ACTIVATION SYNDROME AND OTHER HISTAMINE MEDIATED CONDITIONS

- HIBISCUS TEA, WITH IT'S DEEP COLORED POLYPHENOLS, IS ONE OF THE BEST NATURAL SUPPORTS FOR GLUTATHIONE SYNTHASE ENZYME PRODUCTION OF GLUTATHIONE Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GLUTATHIONE ASSESSMENT



- 1. VIBRANT MICRONUTRIENT PANELS
- 2. HOMOCYSTEINE <5 (NOT A DIRECT MARKER BUT CAN INDICATE SHUNTING OF THE SULFUR CONTAINING HOMOCYSTEINE TO THE TRANSULFURATION PATHWAY TO OFFSET HIGH OXIDATIVE STRESS
- 3. HIGH TGF-BETA (VIBRANT CHRONIC INFLAMMATION PANEL) FUNCTIONAL RANGE 1500-3000 (BOTH NEED CYSTEINE FOR SYNTHESIS SO TGF-B "STEALS" FROM GLUTATHIONE)
- 4. HIGH GGT FUNCTIONAL RANGE 10-30 (GAMMA-GLUTANYL-TRANSAMINASE IS AN INDIRECT MARKER OF GLUTATHIONE SUFFICIENCY IN THE LIVER)
- 5. ORGANIC ACIDS TEST 80H-DG (REFLECTS DNA DAMAGE. HIGHER LEVELS INDICATE MORE FREE RADICAL DAMAGE TO DNA IN THE BODY)
- 6. ORGANIC ACIDS TEST PYROGLUTAMATE VIBRANT (HIGH OR LOW LEVELS OF THIS MARKER ARE A REFLECTION OF GLUTATHIONE RECYCLING. LOW MAY INDICATE PRECURSOR DEFICIENCY AND HIGH MAY INDICATE HIGHER THAN NORMAL GLUTATHIONE RECYCLING)

"405 NM LOW LEVEL LASER HAS THE BENEFITS OF UV LIGHT FOR STERILIZATION WITHOUT THE HARMFUL SIDE EFFECTS. YES, GET YOUR SUN AND VITAMIN D FOR IMMUNE BOOSTING AND STRESS REDUCTION BUT USE LOW LEVEL LASER THERAPY AS A PRIMARY DEFENSE."

-DR. TREVOR BERRY DC, DACNB

Wilhout permission of Trever Berry, DC, DACNB is prohibite LLLT AND ORAL MALADIES



Table 1

Soft and Hard Tissue applications of Low Level Laser Therapy (LLLT)

Soft Tissue Applications	Hard Tissue Applications
Herpetic Lesions	Temporomandibular Disorders
Aphthous/ Traumatic Ulcers	Orthodontic Treatment
Post Oncology Mucositis	Dentinal Hypersensitivity
Post Extraction Socket/ Post trauma Sites	Bone Remodelling
Trigeminal Nueralgia	Erosion
Periodontal Pocket Disinfection/ Periodontitis	Stimulatory effect on root development
Edema	Implants
Sinusitis	
Gag Reflex/ Nausea	
Postsurgical Pain	

Laser Ther. 2015 Oct 2; 24(3): 215–223. doi: 10.5978/islsm.15-RA-01

PMCID: PMC4639680 PMID: <u>26557737</u>

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Low Level Laser Therapy: A Panacea for oral maladies

Vartika Kathuria,1 Jatinder Kaur Dhillon,2 and Gauri Kalra3

PATHOGENS



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.
ORAL PATHOGENS AND ALZHEIMER'S





Bacteria in the Nose May Increase Risk of Alzheimer's Disease New research from Griffith University has shown that a bacterium commonly present in the nose can sneak into the brain and set off a cascade of events that may lead to Alzheimer's disease.

Summary: Chlamydia pneumoniae, a bacterium that is commonly present in the nose, can invade the brain via nerves in the nasal cavity. Once the bacterium is in the central nervous system, brain cells react within days, depositing Alzheimer's-associated amyloid-beta peptides.

"Chlamydia pneumoniae can infect the central nervous system via the olfactory and trigeminal nerves and contributes to Alzheimer's disease risk" by Anu Chacko et al. Scientific Reports

Vopr Kurortol Fizioter Lech Fiz Kult. 2006 Sep-Oct;(5):20-3.

[Low-intensity laser effects on local immunity of female reproductive system affected with chlamydia infection].

Gizinger OA, Dolgushin II.

Low-intensity laser stimulated not only phagocytosis but also intracellular generation of active oxygen forms. Thus, low-intensity

Immunological and microbiological aspects of low intensity laser effect on the factors of local immunity of the reproductive tract in women with chlamydia infection.

Assessment of immunological and microbiological efficacy of Chlamydia cervicitis management was made by a complex method with a low intensity laser. Positive clinical effect of the local use of the low intensity laser for Chlamydia cervicitis treatment was accompanied by positive changes in immunological indices of cervical secret, normal concentration of cytokines in cervical secret, restoration of the number and functions of neutrophils. Local use of the low intensity laser contributed to decreased number of concentration pathogenic microorganisms and their associations, and restored local flora

ORAL PATHOGENS AND ALZHEIMER'S



Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with smallmolecule 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, thick in the end of the end

PATHOGENS AND ALZHEIMER'S



alzheimer's R association

Diagnosis, Assessment & Disease Monitoring

12 April 2021

Periodontal dysbiosis associates with reduced CSF Aβ42 in cognitively normal elderly

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 A β 42 but not with CSF P-tau," the authors wrote in the paper.

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PATHOGENS AND ALZHEIMER'S



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.

<u>Stojkovic D</u>1, <u>Kostic M</u>1, <u>Smiljkovic M</u>1, <u>Aleksic M</u>2, <u>Vasiljevic P</u>2, <u>Nikolic M</u>1, <u>Sokovic M</u>1. **Author information Abstract**

The following review is oriented towards microbes linked to Alzheimer's disease (AD) and antimicrobial effect of compounds and extracts derived from aquatic organisms against specific

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).

PATHOGENS AND ALZHEIMER'S



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 in prmation

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. Aluminium disulphonated phthalocvanine, haematoporphyrin HCI and haematoporphyrin

These findings suggest that lethal photosensitization may be an effective means of eliminating periodontopathogenic bacteria from dental plaque.

PATHOGENS AND ALZHEIMER'S



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.

<u>Hope CK</u>1, <u>Strother M</u>2, <u>Creber HK</u>3, <u>Higham SM</u>4. Author information BACKGROUND:

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.



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).

PATHOGENS AND ALZHEIMER'S



<u>J Oral Microbiol.</u> 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.

<u>Olsen I</u>1, <u>Taubman MA</u>2,3, <u>Singhrao SK</u>4.

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-y 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-y-stimulated release of interferoninducible 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-y 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 perfective and the relative statistic to the associated without permission of Trevor Berry, DC, DACNB is prohibited.

MYCOTOXINS





KEY CONCEPTS: DETECT, REMEDIATE, DRAW AND BIND...

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MYCOTOXINS

SYMPTOMS TO WATCH FOR:

- UNEXPLAINED FATIGUE/MUSCLE WEAKNESS
- CHRONIC BURNING IN THROAT/NASAL PASSAGES
- COUGHING/WHEEZING/SHORTNESS OF BREATH
- METALLIC TASTE IN MOUTH
- UNUSUAL SKIN SENSATION OR RASHES
- UNEXPLAINED JOINT PAIN OR STIFFNESS
- VISUAL DISTURBANCES/LIGHT/SOUND SENSITIVE
- POOR CONCENTRATION/MEMORY RECALL
- CONFUSION
- EXCESSIVE URINATION OR THIRST
- HEART PALPITATIONS/ANS DISTURBANCES
- RANDOM FLARE UPS AFTER RESOLUTION
- OBVIOUS ENVIRONMENT/LOCATION CHANGE OF SYMPTOMS (BETTER ON VACATION ETC)*
- POOR TEMPERATURE REGULATION
- ANXIETY
- DIZZINESS

*CLINICAL PEARL: DRAW OUT MYCOTOXINS WITH FASTING AND GLUTATHIONE PRIOR TO TESTING



ERCHONIA

TEST: VIBRANT MYCOTOXINS URINE BASED MICROASSAY - TEST FOR THE 31 MOST COMMON MOLD FORMS - HOME TESTING KIT - DUE TO COMORBIDITY CONSIDER SCREENING FOR HASHIMOTO'S, CELIAC NEUROLOGICAL OR CONNECTIVE TISSUE

AUTOIMMUNITY AND LYME

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MYCOTOXINS



WHEN IN DOUBT START WITH BINDERS

- GI TRACT XYMOGEN OPTIFIBER LEAN BID BEFORE MEALS
- FULVIC/HUMIC ACID HM-ET (HEAVY METAL/ENVIRONMENTAL TOXIN) BINDER. CELLCORE 1 CAP BID (PREP WITH BIOTOXIN BINDER/MOLD SUPPORT KIT)
- SWEAT IT OUT! IR BATH, SAUNA (WET OR DRY), WORK OUTS, HOT BATH
- FASTING. 24 HOUR WATER FAST 1X/WEEK. FASTING WILL MOBILIZE TOXINS SO YOU MUST BE ON BINDERS AND GLUTATHIONE/NAC
- FOODS TO AVOID THAT EITHER FEED MOLDS OR TEND TO HAVE HIGH MOLD COUNTS: BANANAS, DRIED FRUIT, WHEAT, BARLEY, RYE, OATS, SPICES, COFFEE, CEREAL, OILSEEDS LIKE PEANUT, SOYBEAN AND SUNFLOWER. TREE NUTS LIKE PISTACHIO, ALMOND, WALNUT, BRAZIL NUT, WINE AND GRAPE JUICE, MAIZE

LET TH1 RUN WHILE DRAWING OUT (IE. AVOID TURMERIC FOR 48 HOURS) THEN RETURN

- MOLD REMEDIATION!!! <u>EMSL.COM</u> HOME OR OFFICE MOLD KIT TEST (~\$50/ROOM) BUT WHEN IN DOUBT BE SURE TO USE A PROFESSIONAL REMEDIATION COMPANY
- AN ERMI (ENVIRONMENTAL RELATIVE MOLD INDEX) LEVEL ABOVE 2.0 CAN BE BAD ENOUGH TO BE A MOLD PROBLEM AND IF A PATIENT HAS DEVELOPED SEVERE REACTIONS THEY MAY NEED THEIR ERMI SCORE TO BE AS LOW AS -1
- VISUAL CONTRAST SENSITIVITY (VCS) TEST ~85% CORRELATION TO MOLD PTS ALSO USED AS A MONITORING TEST TO PROGRESS (EX. 4 VCS TESTS \$40)

MYCOTOXINS



OTHER TREATMENT CONSIDERATIONS:

- SPORE BASED PROBIOTIC: ROTATE MEGASPORE MYCOBALANCE AND XYMOGEN PROBIOMAX IG 26 DF. (CONSIDER SNORTING IF NASAL BASED LIKE MARCONS)
- XYMOGEN RESVERATIN PLUS BID
- XYMOGEN CURCUPLEX-95 BID 2 WEEKS THEN ROTATE ONE OFF
- XYMOGEN HORMONE PROTECT X2 BID FOR DURATION OF FM THERAPIES
- XYMOGEN S-ACETYL GLUTATHIONE BID FOR DURATION OF FM THERAPIES
- XYMOGEN OPTICLEANSE GHI 1 SCOOP DAILY, 1 MONTH
- XYMOGEN ADRENALIV BID FOR DURATION OF FM THERAPIES
- MIMOSA PUDICA (SENSITIVE PLANT ORGANIC POWDER: START 1/2 TSP BID 2X/ WEEK THEN BUILD UP TO 1 TSP DAILY FOR 3 MONTHS
- MONOLAURIN (WORK UP TO 1+ SCOOP DAILY)/BIOCIDIN AS DIRECTED DAILY
- MYRRH ESSENTIAL OIL. INHALE USING A DIFFUSER. 1X DAILY. ELEVATES MSH
- WATCH LOW MSH (MELANOCYTE STIMULATING HORMONE). LEVELS BELOW 35 CAN INCREASE SUSCEPTIBILITY TO MARCONS AND MAY HAVE CORRELATION
- WATCH PROLONGED TURMERIC USING CAN SHUT DOWN PHASE 3 LIVER DETOX

OTHER CLINICAL CONSIDERATIONS: SUPPORT TH1 POLARIZATION WITH NAC CONSIDER ADRENAL SUPPORT IN CHRONIC INFECTIOUS DISEASE STATES DR. JILL CARNAHAN IS A GREAT REFERENCE FOR MOLD CASES Explication, citation or distribution ethics research in whole or is park MYCOTOXINS



ERCHONIA LASER PROTOCOL: RED OR EVRL RED/VIOLET SETTING 21-762-880-1146



WAND THE ENTIRE RESPIRATORY/GI SYSTEMS CONSIDER TCT 3 MIN EACH HEMISPHERE 3X/WEEK 4 WEEKS THEN EVERY VISIT FOR DURATION OF FUNCTIONAL MEDICINE SUPPORT

WITH STAPH/MARCONS CONSIDER NASAL COVERAGE 424-453-634-2600 UP TO 2 MINUTES EACH SIDE/ORAL

YOU MUST WATCH EMF EXPOSURE. EMF'S CAN INCREASE MYCOTOXINS 600% AS THEY THINK THEY ARE BEING "ATTACKED" WATCH 5G, HOME ROUTER TIMERS



- * EM-1 SOIL MICROBE THAT EATS MOLD
- * CAN MIX IN PAINT OR DIFFUSE IN ROOM
- * WATCH DYSAUTONOMIA WITH MOLD/LYME CASES. TILT TABLE THERAPIES???

OTHERS: GINGER TEA, DOPAMINE SUPPORT (XYMGOGEN COGNIQUIL) TO FACILITATE DRIVE/AMBITION, MUSHROOM COFFEE/MICRODOSE LION'S MANE/ PSILOCYBIN, ARGENTYN 23 SILVER SPRAY DAILY, HIGH DOSE CHARCOAL, CHOLASTYRAMINE (MEDICATION)³⁷CHLORELLA, CISTUS INCANUS TEA

R2 👩

EMF EXPOSURE AND CELL PHONES



Brain imaging study uncovers distinct neural mechanisms underlying excessive smartphone use

A study using functional magnetic resonant imaging (fMRI) compared brain activities of persons suffering from smartphone addiction (excessive smartphone use) and those who use their smartphones in a less intrusive way. It reported systematic differences in brain activity during rest between the two groups.

Additionally, two fMRI indicators of neural activity were found to be correlated with psychological assessments of excessive smartphone use. The study was published in *Brain and Behavior*.

Excessive smartphone use is very similar to the "internet gaming disorder (IGD)", which is a recognized disorder included in the Diagnostic and Statistics Manual of Mental Disorders, the handbook health practitioners in the US use as a guide for diagnosing mental disorders.

Studies have shown that **individuals with excessive smartphone use behaviors may exhibit structural and functional changes to their brains such as reduced gray matter volume or intrinsic neural activity in the region of the brain called anterior cingulate cortex**, altered functional connectivity and changes in activity in various parts of the cortex during processing of emotions.

Aberrant intrinsic neural network strength in individuals with "smartphone addiction": An MRI data fusion study

EMF EXPOSURE AND CELL PHONES



In an appeal to the European Union, more than 180 scientists and doctors from 36 countries warn about the danger of 5G, which will lead to a massive increase in involuntary exposure to electromagnetic radiation. The scientists urge the EU to follow Resolution 1815 of the Council of Europe, asking for an independent task force to reassess the health effects.

Slovenia halts 5G to Investigate Health and Safety

"Slovenia stops the introduction of 5G technology: We do not know if it is dangerous to humans". In Slovenia 5G had been halted, officials take more time to investigate health effects of the new technology. A letter from Minister Rudi Medved states they will reopen the debate on potential health risks.

Resolution opposing 5G by the municipality of Rome

Germans petition Parliament to stop 5G auction on health grounds

A petition asking the German Parliament to stop the award of 5G frequencies has reached 54,643 signatures, surpassing the quorum, reports 'Diagnose: Funk'. The petioners request the German Parliament to suspend the procedure to award 5G frequencies based on scientifically justified doubts about the safety of this technology.

Netherlands: Parliament asks for independent investigation on 5G health risks

Debate in England on health related effects of electromagnetic fields and 5G

A very powerful speech on the risks of 5G by MP Tonia Antoniazzi at Westminster Hall.

"We can no longer hide and pretend it is not happening and this cannot be swe 379 under the carpet"

EMF EXPOSURE AND CELL PHONES



- 5G IS THE NEXT (5TH) GENERATION CELLULAR NETWORK
- YOUR DEVICE WILL NEED TO BE A 5G DEVICE TO REAP THE BENEFITS
- SPEEDS WILL BE 10-100X FASTER THAN 4G BECAUSE 5G IS IN MILLIWAVES VS MICROWAVES AND CAN REACH CONNECTION SPEEDS OF 1GBPS+. HOWEVER, MILLIWAVES HAVE A HARD TIME TRAVELING DISTANCES, PENETRATING BUILDINGS, GLASS, EVEN HEAT AND TREES
- THERE ARE ACTUALLY 3 5G BANDS LOW, MID AND HIGH. LOW IS WHAT COMPANIES LIKE AT&T AND T-MOBILE ADVERTISE AS THEIR 5G NETWORK. ALTHOUGH TECHNICALLY BETTER THAT 4G, THEY ARE LOW FREQUENCY 5G TO BLANKET MORE AREA BUT NOT CLOSE TO TRUE HIGH 5G SPEED. SPRINT DID AN EARLY MID BAND 5G ROLL OUT AND IT IS ONE OF THE REASONS WHY T-MOBILE ACQUIRED SPRINT SO THEY COULD CLAIM ALL 3 FLAVORS OF 5G AS YOU WILL SEE IN THEIR ADVERTISING. VERIZON ADVERTISES THEIR TRUE 5G MILLIWAVE BAND AS "5G UWB" (ULTRAWIDEBAND) AND AT&T AS "5G PLUS" (THEIR 5GE ISN'T EVEN 5G AND REGULATORS ASKED THEM TO STOP ADVERTISING IT AS SUCH)
- THERE WILL BE SOME FUNCTIONAL BENEFITS TO 5G AND ITS "ECOSYSTEM". FOR EXAMPLE, IT WILL ALLOW FOR TECHNOLOGICAL ADVANCEMENTS LIKE DRIVERLESS CAR NAVIGATION, HEALTHCARE MONITORING SYSTEMS, ROBOTIC AND DRONE NAVIGATION AND FASTER DOWNLOAD TIMES (LOW LATENCY). BUT IS THERE A DARK UNDERBELLY TO THIS NEXT GENERATION...

EMF EXPOSURE AND bled. CELL PHONES



We Have No Reason to Believe 5G Is Safe

By Joel M. Moskowitz PhD on October 17, 2019

HIGHLIGHTS:

- 5G WAVES ARE IN MILLIMETERS (VS 2G & 4G MICROWAVES). THESE SHORTER WAVELENGTHS IN PURE FORM WILL REQUIRE CELL ANTENNAS EVERY 100 TO 200 METERS EXPOSING MANY MORE PEOPLE TO MILLIMETER WAVE RADIATION.
- DUE TO THE SHORT WAVELENGTHS, 5G WILL STILL TYPICALLY PIGGYBACK WITH 4G MICROWAVES. RESEARCHERS HAVE NOT STUDIED WHAT KIND OF SYNERGISTIC RISK THIS MAY POSE.
- MILLIMETER WAVES ARE MOSTLY ABSORBED WITHIN A FEW MILLIMETERS OF HUMAN SKIN AND SURFACE LAYERS OF THE CORNEA CREATING A NEW SET OF CLINICAL IMPLICATIONS SUCH SKIN CONDITIONS LIKE MELANOMA'S, OCULAR DAMAGE, EVEN GREATER MALE GONAD EXPOSURE (STERILITY, TUMORS?) AND PERIPHERAL NEUROPATHIES!

EMF EXPOSURE AND CELL PHONES



- THE FCC MERELY REAFFIRMED THE RADIO FREQUENCY RADIATION (RFR) EXPOSURE LIMITS THAT IT ADOPTED IN THE 1990'S BASED ON SHORT-TERM <u>HEATING</u> RISKS AND BEHAVIORAL CHANGES MICROWAVE RADIATION CAUSED RATS AND MOST OF THIS RESEARCH WAS DONE IN THE LATE 1980'S.
- YET THE PREPONDERANCE OF PEER-REVIEWED RESEARCH, MORE THAN 500 STUDIES, HAVE FOUND HARMFUL BIOLOGICAL EFFECTS OF EXPOSURE TO RFR AT INTENSITIES TOO LOW TO REACH THIS SIGNIFICANT HEAT THRESHOLD!
- MORE THAN 240 OF THESE PEER-REVIEWED SCIENTISTS SIGNED THE INTERNATIONAL EMF SCIENTISTS APPEAL WHICH CALLS FOR STRONGER EXPOSURE LIMITS DUE TO ACTUAL BIOLOGICAL AND HEALTH EFFECTS OF ELECTROMAGNETIC FIELDS (EMF'S)
- THESE SCIENTISTS COLLECTIVELY HAVE PUBLISHED OVER 2000 PAPERS ON EMF IN PROFESSIONAL JOURNALS.

EMF EXPOSURE AND CELL PHONES



- IN THE APPEAL THE SCIENTISTS MAKE THE FOLLOWING HEALTH HAZARD ASSERTIONS OF THESE EMF'S: CANCER RISK, CELLULAR STRESS/FREE RADICAL DAMAGE, GENETIC DAMAGES, REPRODUCTIVE SYSTEM CHANGES, LEARNING AND MEMORY DEFICITS, NEUROLOGICAL DISORDERS AND NEGATIVE EFFECTS ON GENERAL WELL-BEING IN HUMANS.

- THEY GO ON TO STATE, "THERE IS GROWING EVIDENCE OF HARMFUL EFFECTS TO BOTH PLANT AND ANIMAL LIFE."

> SCIENTIFIC AMERICA 883

EMF EXPOSURE AND CELL PHONES



Brussels Becomes First Major City to Halt 5G Due to Health Effects

A "Whistleblower" Blows The Lid Off 5G Microwave Towers http://www.theeventchronicle.com/geoengineering/awhistleblower-blows-the-lid-off-5g-microwave-towers/

5G radiofrequency (RF) radiation uses a 'cocktail' of three types of radiation, ranging from relatively low-energy radio waves, microwave radiation with far more energy, and millimeter waves with vastly more energy

https://needtoknow.news/2019/05/severe-health-risks-of-5g-have-beencovered-up/?fbclid=IwAR284iXDtySXISXUpjAjjPAIgwhv09y-90Pv-DnSySKmWesZFkDeWxHK14w

Is 5G Worth The Risks?

https://www.zerohedge.com/news/2019-05-04/5g-worth-risks

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5G: Great risk for EU, U.S. and International Health? Competing Evidence for Fight Distinct Types of Great Harm Caused by Electromagnetic Field (EMF) Exposures and the Mechanism that Causes Them Written and Compiled by Martin L. Pall, PhDProfessor Emeritus of Biochemistry and Basic Medical Sciences Washington State University Address: 638 NE 41st Ave., Portland OR 97232 USA martin_pall@wsu.edu 503-232-3883 May 17, 2018

Summary: We know that there is massive literature, providing a high level of scientific certainty, for each of eight pathophysiological effects caused by non-thermal microwave frequency EMF exposures. This is shown in from 12 to 35 reviews on each specific effect, with each review listed in Chapter 1, providing a substantial body of evidence on the existence of each effect. Such EMFs:

1. Attack our nervous systems including our brains leading to widespread neurological/neuropsychiatric effects and possibly many other effects. This nervous system attack is of great concern.

2. Attack our endocrine (that is hormonal) systems. In this context, the main things that make us functionally different from single celled creatures are our nervous system and our endocrine systems - even a simple planaria worm needs both of these. Thus the consequences of the disruption of these two regulatory systems is immense, such that it is a travesty to ignore these findings.

3. Produce oxidative stress and free radical damage, which have central roles in essentially all chronic diseases.

4. Attack the DNA of our cells, producing single strand and double strand breaks in cellular DNA and oxidized bases in our cellular DNA. These in turn produce cancer and also mutations in germ line cells which produce mutations in future generations.

5. Produce elevated levels of apoptosis (programmed cell death), events especially important in causing both neurodegenerative diseases and infertility.

6. Lower male and female fertility, lower sex hormones, lower libido and increased levels of spontaneous abortion and, as already stated, attack the DNA in sperm cells.

7. Produce excessive intracellular calcium [Ca2+] and excessive calcium signaling.

8. Attack the cells of our bodies to cause cancer. Such attacks are thought to act via 15 different mechanisms during cancer causation.

Italian court finds link between cell phone use and tumor By COLLEEN BARRY/Apr 19, 2017

886

MILAN – A longtime Telecom Italia employee has been awarded monthly social security payments after a court found that his brain tumor was caused by improper use of a company-issued cellphone.

Lawyer Stefano Bertone said Thursday that it was the first trial court verdict of which he was aware in the world "to recognize a link between cellphone use and the development of brain tumor."

<u>Electron Physician.</u> 2016 May 25;8(5):2452-7. doi: 10.19082/2542. eCollection 2016. **Effects of the Effect of Ultra High Frequency Mobile Phone Radiation on Human Health.** <u>Moradi M</u>1,

The results of this study and International Commission of Non Ionization Radiation Protection (ICNIRP) reports showed the people who spend more than **50 minutes** *a day using a cell phone could have early demonstration* of the period by t

EMF EXPOSURE AND CELL PHONES



Toxicol In Vitro

2020 Oct;68:104963. doi: 10.1016/j.tiv.2020.104963. Epub 2020 Aug 8.

Effects of pulse-modulated radiofrequency magnetic field (RF-EMF) exposure on apoptosis, autophagy, oxidative stress and electron chain transport function in human neuroblastoma and murine microglial cells

Jana Zielinski 1, Angélique D Ducray 2, Anja M Moeller 3, Manuel Murbach 4, Niels Kuster 5, Meike Mevissen 6

In this study, effects of ELF-modulated 935 MHz RF-EMF on apoptosis, autophagy, oxidative stress and electron exchange in N9 microglial and SH-SY5Y neuroblastoma cells were investigated.

indicating that short-time RF-EMF at SAR levels accepted by today's safety guidelines might cause autophagy and oxidative stress with the effect being dependent on cell type and exposure duration.

Food Funct

. 2014 Sep;5(9):2289-97. doi: 10.1039/c4fo00250d.

Extremely low frequency electromagnetic field exposure causes cognitive impairment associated with alteration of the glutamate level, MAPK pathway activation and decreased CREB phosphorylation in mice hippocampus: reversal by procyanidins extracted from the lotus seedpod

Yuqing Duan 1, Zhigao Wang, Haihui Zhang, Yuanqing He, Rong Fan, Yanxiang Cheng, Guibo Sun, Xiaobo Sun

The results showed that ELF-EMF exposure induced the increased contents of glutamate, GABA, excessively activated NMDA receptors, increasing the number of NMDA receptor 2B (NR2B) and intracellular Ca(2+) concentration [Ca(2+)] in hippocampus.

ELF-EMF exposure also increased JNK1/2 phosphorylation through the activated ASK1, which plays a pivotal role in hippocampal neuronal cell death.

EMF EXPOSURE AND CELL PHONES



Environ Res

. 2019 Sep;176:108525. doi: 10.1016/j.envres.2019.108525. Epub 2019 Jun 11.

Residential exposure to ultra high frequency electromagnetic fields emitted by Global System for Mobile (GSM) antennas and amyotrophic lateral sclerosis incidence: A geo-epidemiological population-based study

Jaime Luna 1, Jean-Philippe Leleu 2, Pierre-Marie Preux 3, Philippe Corcia 4, Philippe Couratier 5, Benoit Marin 3, Farid Boumediene 6, Fralim Consortium

A gradient effect between UHF-EMF exposure and ALS incidence was apparent with a statistically significant trend. A significant increased risk of ALS was observed between the non-exposure category and the highest exposure category, with a relative risk of 1.78 (95% CI: 1.28-2.48) in the non-cumulative model and 1.83 (95% CI: 1.32-2.54) in the cumulative model.

Our results suggest a possible association between residential UHF-EMF exposure and ALS. Brain Res

. 2010 Oct 14;1356:95-101. doi: 10.1016/j.brainres.2010.07.103. Epub 2010 Aug 4.

Purkinje cell number decreases in the adult female rat cerebellum following exposure to 900 MHz electromagnetic field

Osman Fikret Sonmez 1, Ersan Odaci, Orhan Bas, Süleyman Kaplan

Abstract

The biological effects of electromagnetic field (EMF) exposure from mobile phones have growing concern among scientists since there are some reports showing increased risk for human health, **especially in the use of mobile phones for a long duration. In the presented study, the effects on the number of Purkinje cells in the cerebellum** of 16-week (16 weeks) old female rats were investigated following exposure to 900 MHz EMF.

Results showed that the total number of Purkinje cells in the cerebellum of the EMFG was significantly lower than those of CG (p<0.004) and SG (p<0.002). In addition, there was no significant difference at the 0.05 level between the rats' body and brain weights in the EMFG and CG or SG. Therefore, it is suggested that long duration exposure to 900 MHz EMF leads to decreases of Purkinje cell numbers in the female rat cerebellum.



23 Studies show that EMR affects the voltage gated calcium channels and causes release of peroxinitrite

Pall, M. L. (2013). Electromagnetic fields act via activation of voltage-gated calcium channels to produce beneficial or adverse effects. Journal of cellular and molecular medicine, 17(8), 958-965.



Are Cell phones Dangerous? Why dake the chance?

REDUCE YOU AND YOUR FAMILY'S RADIATION

EXPOSURE UP TO



891



R2L ACTS ON ALL CELL NETWORKS (4G, 5G ETC)

INTRODUCING THE ERCHONIA R2L RADIATION TO LIGHT

It's the revolutionary, super-thin microprocessor that converts cell phone radiation into harmless light! The R2L attaches to ANY smart-phone or cell phone and reduces your exposure to cell phone radiation up to 70%-WITHOUT disrupting the quality of your calls!

The R2L was created by Erchonia Corporation—the world's largest manufacturer of therapeutic lasers.

Now you don't have to leave your cell phone at home, turn it off or hold it an inch from your head in order to significantly reduce your exposure to cell phone radiation. Just peel off the backing and place directly on your existing phone or case as shown —that's all there is to it!

SAR TESTED NO CALL INTERRUPTIONS WORKS WITH ANY PHONE

892

Why test for SAR?

EMC

Electromagnetic radiation (EMR), also known as non-ionising radiation, is generated by virtually all devices that transmit RF energy. It is known that exposure of the human body to high levels of EMR may lead to adverse health effects. To ensure the protection of personnel in the workplace and to protect members of the general public, the Australian Communications Authority (ACA) and other government bodies around the world such as the FCC and the European Commission, have mandated new regulatory frameworks which set compliance requirements for RF transmitting devices. Owners and operators of Radio and Telecommunications equipment, manufacturers and importers will need to establish compliance with the EMR standards which in many cases require SAR evaluation. In the USA, the FCC sets SAR limits and test methods for SAR evaluations. In Australia, compliance requirements are prescribed in the ACA EMR Standard 2003. Most other

EMC Technologies - SAR Testing



countries have similar requirements.

The first phase of the ACA EMR framework commenced on the 31 January 1999. The current scope of the ACA EMR standard 2003 includes mobile phones and mobile phone base stations such as AMPS, <u>GSM</u>, <u>CDMA</u>, <u>DECT</u>, CT2/CT3, PHS, spread spectrum devices, WLAN laptops, tablet PCs, PDAs etc. The ACA and FCC scope now includes devices



used at the head and devices used near any other part of the body. These devices must be evaluated against the ACA or FCC mandatory human exposure standard, which sets basic restrictions for the fore of the standard sta



893 ERCHONIA'S R2LS

- Reduce the possible harmful RF radiation up to 70%
- Minimal, if any; impact to device performance
- Works on GSM and/or CDMA networks
- Tested effective on major device manufacture
- FCC approved, SAR and T1 tested



R2L Product Placement

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Erchonia has put a list of cell phones together to help you and your family place the R2L Phone Protectors in the correct position for maximum RF reduction *(as close to the antenna as possible)*. If your phone is not listed below, please use the following video to locate the correct positioning on your phone: R2L Training ~ How to place the R2L on your cell phone.



Blackberry 8830 Position at Bottom/Center and Slightly Wrap-around Bottom





Blackberry Bold 9930 Bup Stitute at Bottom Center and tribution of the stitute at Bottom Center and tribution of the stitute at Bottom Center and the period bottom of the stitute at Bottom Center at Bottom Center at Bottom of the stitute at Bot



Google Nexus 4 Position on Back Upper/Right

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BE SURE TO CARRY R2L'S IN YOUR OFFICE!

896

Give a gift that protects your family this ho<mark>l</mark>iday season

Ask your doctor about the R2L, the product that fits onto any ell phone and reduces your jamilies exposure to radiation up to 7%, without interupting the quality of the call. Family package discounts are none available!







Unravelling the antimicrobial action of antidepressants on gut commensal microbes GUT ZOOMER

volume 10, Article number: 17878 (2020) OCTOBER 21, 2020

The scientists grew common gut bacteria on petri dishes and added varying doses of different antidepressants to

measure how the drugs affected these gut bacteria. They found that different

types of antidepressants inhibited the growth of common, and important, gut bacteria, and that <u>desipramine</u> (TCA Norpramin) and <u>aripiprazole</u> (Anti-psychotic/schizophrenia) had the greatest effects on the bacteria.

MANY MEDICATIONS INCLUDING RX FOR NEUROLOGICAL CONDITIONS LIKE DEPRESSION AND PARKINSON'S ACTUALLY DEPLETE CO-FACTORS THAT ARE NEEDED FOR THE PARTICULAR NEUROTRANSMITTER NEEDED IN THAT CONDITION! ONE SOURCE FOR CO-FACTOR DEPLETION BY MEDICATIONS IS <u>MYTAVIN.COM</u> <u>NATURALMEDICINES.COM</u>

DEPRESSION AND MEDICATIONS



3.0

Antidepressants Are **Not** Associated With Improved Quality of Life in the Long Run

Over time, using antidepressants is not associated with significantly better health-related quality of life, compared to people with depression who do not take the drugs.

"Antidepressants and health-related quality of life (HRQoL) for patients with depression: Analysis of the medical expenditure panel survey from the United States" by Omar Almohammed et al. *PLOS ONE* fAPRIL 20, 2022

Fetal Exposure to Antidepressants May Affect Infants'

Brain Development

Summary: Fetal exposure to antidepressants or antiepileptic medications may affect the development of newborn brain networks.

"<u>Cortical Cross-Frequency Coupling Is Affected by in utero Exposure to Antidepressant Medication</u>" by Anton Tokariev et al. *Frontiers in Neuroscience*

MARCH 03, 2022 plication, citation or distributions of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

without permission of Trever Berry, DC, DACNB is prohite MEMORY AND ADHD



Short-Term Memory Can Be Improved with Laser Therapy, Study Shows

- A new study shows that laser light therapy can improve short-term working memory (ex. Someones name you just met or a set of instructions like directions to a house) in young adults.
- The findings have future implications for treating health conditions that impact working memory, such as ADHD.

Researchers at the University of Birmingham in the United Kingdom and Beijing Normal University in China found that laser light therapy improved working memory by around 10% in healthy volunteers. The treatment, known as transcranial photobiomodulation (tPBM) delivers near-infrared to infrared light to the brain tissue through the scalp and skull. Researchers are currently investigating tPBM as a way to improve cognitive function as well as to treat other conditions that affect the brain including: major depressive disorder (MDD) traumatic brain injury (TBI) and Alzheimer's disease "People with conditions like ADHD or other attention-related conditions could benefit from this type of treatment, which is safe, simple and non-invasive, with no side-effects," study author Dongwei Li, a visiting PhD student in the University of Birmingham's Centre for Human Brain Health, said in a news release.

NEUROPHYSIOLOGY Share on

Transcranial photobiomodulation enhances visual working memory capacity in humans

CHENGUANG ZHAQ HTTPS://ORCID.ORG/0000-0002-1573-3878, DONGWELLI HTTPS://ORCID.ORG/0000-0002-2432-8882, YUANJUN KONG, HONGYU LIU, YIQING HU, HAUING NIU HTTPS://ORCID.ORG/ 0000-0002-3887-1966, OLE JENSEN HTTPS://ORCID.ORG/0000-0001-8193-8348, XIAOLI LI HTTPS://ORCID.ORG/0000-0003-1359-5130, HANLI LIU HTTPS://ORCID.ORG/0000-0002-9312-5691, AND YAN SONG HTTPS://ORCID.ORG/0000-0002-5923-7673 Authors Into & Affiliations. *NOTE THIS WAS A 1094 NM STUDY

Reflex	Purpose	Age Appears	Age Integrates	Signs of Retention
Moro Reflex	Fight or Flight reaction, sympathetic nervous system response	Birth	2-4 months	Hypersensitivity to one or more sensory systems, vestibular deficits (motion sickness, poor coordination and balance), oculomotor and visual-perceptual problems, poor pupillary reactions to light, hypersensitivity to auditory input, allergies and lowered immunity, adverse drug reactions, poor stamina, poor adaptability, reactive hypoglycemia
Rooting Reflex	Autonomic Response to locate food and breast	Birth	3-4 months	Anterior tongue tie, thumb sucking, oral hypersensitivity, poor eating, speech and articulation problems, swallowing and chewing deficits
Palmar Grasp Reflex	Autonomic Flexion of the fingers to grab when the palm is stimulated	Birth	3-6 months	Poor manual dexterity, deficits with pencil grip, poor visual coordination, poor posture during handwriting, poor writing skills, correlated speech and hand movements, dysfunction of the tactile and proprioceptive sensory systems
Asymmetric Tonic Neck Reflex (ATNR)	Assists with movement through the birth canal at delivery and is important for cross pattern movements	Birth	6 months	Decrease hand eye coordination, poor handwriting, uncoordinated gait, poor balance, poor visual motor skills and tracking, problems with math and reading, difficulty crossing midline
Spinal Galant Reflex	Important during the birthing process and helps to facilitate movement of the hips during descending the birthing canal	Birth	3-9 months	Postural issues like scoliosis, misaligned or rotated pelvis, and pain in lower back, bedwetting after potty training, hyperactivity, attention and concentration issues, decreased endurance, chronic digestive issues, decreased lower body coordination, pain and tension in legs
Tonic Labyrinthine Reflex (TLR)	Foundational for postural stability for large muscle groups	In Utero	3 ¹ / ₂ years	Decreased balance, poor spatial awareness, toe walking, hypermobility of joints, weak muscles, poor posture, motion sickness, poor ability to climb, atypical head position (forward or to side)
Landau Reflex	Necessary for postural development	4-5 months	1 year	Poor posture and muscle tone, summersaults are challenging, poor coordination for activities that require upper body and lower body to move together, delayed motor development
Symmetric Tonic Neck Reflex (STNR)	Foundational for crawling	6-9 months	9-11 monshoo	Poor posture in standing, poor seated posture, ape-like walk, low muscle tone, W sitting position common, sloppy/messy eater, poor hand/eye coordination

PRIMITIVE REFLEX SUMMARY

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PPLIE

Primitive Reflexes



Primitive reflexes are the reactive actions that a newborn body has to survive in the beginning stages of life. These reflexes are essential in allowing the body to develop properly, and in order for this to happen, these reflexes must be integrated before childhood. If these reflexes are retained and milestones of development are missed, the child may experience some developmental delays as well as significant difficulties with various functional skills later in life.







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NEURODEVELOPMENTAL COMMON PRIMITIVE REFLEX INTEGRATION EXERCISES



FACE STROKING FOR ROOTING REFLEX: STROKE THE CHILDS FACE UNTIL THE REFLEX STOPS. THIS CAN TAKE MORE THAN 5 TIMES. PERFORM 2X/DAY UNTIL REFLEX IS GONE.

BALL SQUEEZE FOR PALMER REFLEX: HAVE THE CHILD SQUEEZE A STRESS TOY OR TENNIS BALL MULTIPLE TIMES UNTIL REFLEX STOPS. PERFORM 2X/DAY UNTIL REFLEX INTEGRATES.

SNOW ANGELS FOR SPINAL GALANT REFLEX: SLOW "SNOW ANGELS" COMBINED WITH BREATHING FOCUS X5. INHALE AS ARMS GO OVER YOUR HEAD AND LEGS SPREAD. EXHALE WHILE ARMS GO BACK DOWN. PERFORM 2X/DAY UNTIL REFLEX INTEGRATES.

STARFISH FOR MORO REFLEX: CHILD STARTS IN FETAL POSITION (IN A CHAIR OR ON A MAT) WITH CLENCHED FISTS RIGHT OVER LEFT WRIST AND ANKLE THE HAVE THE PATIENT FLARE WIDE OPEN LIKE A STARFISH. HOLD THIS POSITION WHILE HOLDING INHALATION FOR ABOUT 5 SECONDS THEN RETURN TO FETAL FLEXION BUT OPPOSITE CROSSING (LEFT OVER RIGHT).

FENCER EXERCISE FOR ASYMMETRIC TONIC NECK REFLEX: UNTIL IT FATIGUES X3 EACH SIDE 2X/DAY UNTIL REFLEX INTEGRATES.





Left on Top

AUTISM RISK WITH VALPROIC ACID



Spinal Galant Exercise 'Snow Ange

Researchers determined that valproic acid prevents nervous system cells from properly developing and dividing

When used during pregnancy, the drug valproic (DEPAKOTE/ **DEPAKENE**) acid, which is used to treat bipolar disorder, migraines, and epilepsy, can lead to birth defects and Autism. Now, research recently published in the journal PLoS Biology by Bill Keyes of the Institute of Genetics and Molecular and Cellular Biology, France, and associates gives one explanation for why: Valproic acid (VPA) causes certain nervous system development cells to enter a condition known as senescence, which prevents them from properly growing and dividing.

Reference: "Aberrant induction of p19^{M-}-mediated cellular senescence contributes to neurodevelopmental defects" by Muriel Rhinn, Irene Zapata-Bodalo, Annabelle Klein, Jean-Luc Plassat, Tania Knauer-Meyer and William M. Keyes, 14 June 2022, PLoS Biology. DOI: 10.1371/journal.pbio.3001000 lication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

FOOD ALLERGIES AND NEUROLOGICAL INFLUENCES



APPROXIMATELY 10% OF AMERICANS SUFFER FROM FOOD ALLERGIES NOW https://www.foodallergy.org/resources/facts-and-statistics

some people who test positive on skin-prick allergy tests with moderate increases in IgE don't notice any allergy-related symptoms when they eat the allergen. This condition is sometimes referred to as asymptomatic sensitization. In many cases, people with this condition may not even be aware that they have a food hypersensitivity. Several recent studies in people have supported the association between food allergies and various neuropsychiatric disorders, including depression, anxiety, attention-deficit/hyperactivity disorder and autism. They strengthen the possibility that some reactions to food allergens could involve the nervous system and manifest as behavioral disorders.

Interestingly, BLG-sensitized mice showed anxiety-like behavior one day after receiving a large amount of the allergen. Another group of sensitized mice developed depression-like behavior after eating small amounts of allergen for two weeks. In addition, BLG-sensitized mice showed signs of brain inflammation and neuronal damage, suggesting that changes in the brain may be responsible for their behavioral symptoms.

We also investigated the long-term effect of allergen consumption by keeping BLG-sensitized mice on the allergen-containing diet for one month. We found that IgE levels declined in sensitized mice by the end of the month, indicating that continually eating small amounts of the allergen led to decreased immune responses, or "desensitization." In contrast, signs of brain inflammation remained, suggesting that the harmful effect of allergens persisted^{®5} in the brain.



DISCLAIMER: THESE ARE NOT CLAIMS TO TREAT OR CURE ANY MEDICAL CONDITION. LOW LEVEL LASER, NUTRITION SUPPORT OR ANY OTHER CAM THERAPIES ARE TO SUPPORT NORMAL ADAPTIVE PHYSIOLOGY OF THE HUMAN BODY. uplication, citation or distribution of this material in whole or in par without permission of income Bood Dodg NP is prohibited.

Int J Mol Sci. **2021 May;** 22(9): 4942. Published online 2021 May 6. doi: <u>10.3390/ijms22094942</u>

PMCID: PMC8124384 PMID: <u>34066560</u>

Probiotics, Photobiomodulation, and Disease Management: Controversies and Challenges

Laura Marinela Ailioaie1,2 and Gerhard Litscher3,*

GREAT REVIEW PAPER ON GUT MICROBIOME, LASER PHOTOBIOMODULATION (PBM), IMMUNOLOGY AND PATHOGENS. SOME OF THE KEY POINTS:

- LOW LEVEL LASER/PHOTOBIOMODULATION HAS BEEN SHOWN TO IMPROVE GUT MICROBIOME DIVERSITY
- PBM HAS BEEN SHOWN TO TO PROPERLY REGULATE IMMUNE RESPONSES INCLUDING IL-10, TGF-B
- PBM HAS AN ABSCOPAL (FAR REACHING SYSTEMIC) EFFECT ON PATHOGENS THROUGH MECHANISMS LIKE ACTIVATION OF CYTOTOXIC T CELLS
- THERE IS A HORMESIS IN PBM ON PATHOGENS, IMMUNITY AND GUT MICROBIOME ALTERATIONS SIMILAR TO NEUROLOGICAL CONDITIONS, CANCER, PAIN MANAGEMENT AND MORE



ERCHONIA

COVID VACCINES

ORONAVIRUS (COVID-19)

Pfizer CEO Albert Bourla diagnosed with an unfortunate infection

Wednesday, August 17, 2022

If you've ever heard the advice "never get a haircut from a bald barber" you know that even something completely natural can tarnish the image of a brand in people's minds. So, much like being judged for being an overweight fitness instructor, it's a bit awkward for Albert Bourla that people now know he's been infected by Covid-19, considering he is the CEO of Pfizer.



CEO Bourla has received both vaccines as well as a first and even a second booster shot, all of the vaccines produced by his company Pfizer in association with BioNTech

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The CDC (Finallys) Aromited the Science on Natural Immunity. Why Did It Take so Long?Monday, August 22, 2022

n August 2021 *Science Magazine*, a peer-reviewed academic journal of the American Association for the Advancement of Science, highlighted groundbreaking research out of Israel that upended the public health establishment. The research, which relied on a database enrolling some 2.5 million Israelis and was led by Tal Patalon, head of the KSM Research and Innovation Center at

Maccabi Healthcare Services, and deputy Sivan Gazit, found that previous infection from Covid-19 conferred considerably stronger and longer-lasting protection against the Delta variant than vaccines. "never-infected people who were vaccinated in January and February were,

in June, July, and the first half of August, Six to 13 times more

likely to get infected than unvaccinated people" who had previously had Covid.

It's not an exaggeration to say that billions of dollars were at stake over whether Covid vaccines were mandatory or voluntary, which might explain why **Pfizer alone spent more than \$10** million lobbying in 2021 and even more in 2020.

Comparing SARS-CoV-2 natural immunity to vaccine-induced immunity: reinfections versus breakthrough infections Sivan Gazit, MD MA1,2*; Roei Shlezing

COVID AND KIDS

Local health stats show unvaxxed kids less likely to have COVID than vaccinated youth

Jul 19, 2022, 6:23 PM

Using figures based on every 100,000 people in King County, **if you have a 5**to 11-year-old and they are fully vaccinated, they are 46% more likely to catch or have COVID than unvaccinated 5to 11-year-olds.

What about local 12- to 15-year-olds? They're the youngest age group eligible for the booster. According to this same data, if your child is 12 to 15 and boosted, they are 16 percent more likely to get COVID compared to unvaccinated kids their same age.

For 16- and 17-year-olds? They're 14 percent more likely to get COVID. This report appears to show that for every age range, children who are vaccinated and potentially boosted are more likely to get COVID than unvaccinated children.

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Catching Covid can age your brain by 20 years and make your IQ drop, study finds People who have had a serious case of Covid may have seen their IQ levels drop by 10 points and had their brain age by 20 years, according to a new study

The study also suggests that these impacts are still noticeable more than six months after having the virus and that what recovery does happen is gradual.



Edward T. Bullmore, James B. Rowe, David K. Menon. **Multivariate profile and** acute-phase correlates of cognitive deficits in a COVID-19 hospitalised cohort. *eClinicalMedicine*, 2022; 47: 101417 DOI: 10.1016/j.eclinm.2022.101417

CDC REPORT ON LONG COVID



Nearly One in Five American Adults Who Have Had COVID-19 Still Have "Long COVID"

<u>New data from the Household Pulse Survey</u> show that more than 40% of adults in the United States reported having COVID-19 in the past, and nearly one in five of those (19%) are currently still having symptoms of "long COVID."

The data were collected from June 1-June 13, 2022 by the U.S. Census Bureau and analyzed by CDC's National Center for Health Statistics (NCHS) Duplication, citation or distribution of this material in whole or in part

NEUROLOGICAL CONSEQUENCES OF COVID SUMMARY

Nervous system consequences of COVID-19

SERENA SPUDICH AND AVINDRA NATH

SCIENCE • 20 Jan 2022 • Vol 375, Issue 6578 •

HIGHLIGHTS:

VERY LITTLE EVIDENCE OF ACTUAL COVID VIRUS PROTEINS FOUND IN THE CNS IE. SPIKE 2 OR NUCLEIC ACID PROTEINS suggesting that SARS-CoV-2 presence in the CNS does not incite classic viral encephalitis.

The most common neurologic conditions are anosmia (loss of smell), stroke, delirium, brain inflammation, encephalopathy, primary psychiatric syndromes, and peripheral nerve syndromes

Autopsy studies of patients with acute COVID-19 show infiltration of macrophages, CD8+ T lymphocytes in perivascular regions, and widespread microglial activation throughout the brain

TO PROTECT AGAINST THIS ACTIVE END STAGE INFLAMMATION DAMAGE (EX. INTERLEUKIN 1B, 6, 12, TNF ETC) AND PROMOTE PROPER IMMUNOREGULATION (EX. IL 4, 10, 13) TREAT THE CNS (TRANSCRANIAL AND VAGAL STIMULATION) WITH YOUR ERCHONIA DEVICE PREVENTATIVELY, DURING ACUTE AND SUB-ACUTE PHASES AND FOR LONG HAULERS



SARS-COV-2 IN NEURONS MAY DAMAGE BRAIN TISSUE

JOURNAL OF EXPERIMENTAL MEDICINE January 12 2021

Neuroinvasion of SARS-CoV-2 in human and mouse brain

A recent three-pronged study concludes that SARS-CoV-2 may infect nerve cells and impede blood flow in the central nervous system.

These findings suggest that the virus has the ability to alter cell metabolism to create an environment in which infected cells thrive and neighboring cells are unable to survive.

The organoid model also showed that the ACE2 receptor allowed the virus to enter brain cells, similarly to the way that this happens in the lungs. The result was surprising, as it had previously been unclear whether cells in the CNS produced ACE2 receptors.

Autopsies of COVID-19 patients

Finally, the researchers examined the brain regions of three patients who had died from severe COVID-19 complications. All had experienced respiratory failure and been admitted to the intensive care unit.

Within the infected brain regions, there were indications of tissue damage and cell death in the form of ischemic infarcts — areas of dead tissue caused by a lack of blood flow.

PROTECT THE BRAIN WITH RED OR VIOLET RED ERCHONIA LLLT ADVANCED BRAIN SETTING TRANSCRANIAL 1-10-40-60 FOLLOWED BY VAGAL STIM PROTOCOL 4 MIN SCM/GUT

without portion of the service of th

Abstract

Laser light with low-power intensity is applied to the surface of the skin to produce local and systemic effects. Based on the clinical experience, peer-reviewed studies, and solid laboratory data in experimental animal models

experience, peer-reviewed studies, and solid laboratory data in experimental animal models, LLLT attenuates cytokine storm at multiple levels and reduces the major inflammatory metabolites. LLLT is a safe, effective, low-cost modality without any side-effects that may be combined with conventional treatment of ARDS. We summarize the effects of LLLT on pulmonary inflammation and we provide a protocol for augmenting medical treatment in

COVID-19 patients. LLLT combined with conventional medical therapy has the potential to prevent the progression of COVID-19, minimize the length of time needed on a ventilator, enhance the healing process, and shorten recovery time.

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>

PMCID: PMC7428000 PMID: 32844112

Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19

Soheila Mokmeli, MD Anesthesiologist and Mariana Vetrici, MD, PhD 22

COVID AND RED LLLT

• LLLT is an affordable modality compared with other treatments and medicines like IL-6 antagonists. LLLT is a safe, effective, low-cost modality without any reported side-effects compared with other approaches. A laser machine costs Can\$35,000.00–200,000.00, and each machine can fully treat 20,000 patients for COVID-19. In comparison, an IL-6 antagonist costs US\$1000.00 per injection, and each patient would need 3–6 injections for complete COVID-19 treatment. Treating 20,000 patients would cost US\$ 60,000,000.00–US\$ 120,000,000.00.

Based on this information, *LLLT will accelerate recovery from COVID-19* and will get patients off ventilator support and out of the ICU more rapidly. This could significantly decompress our severely overburdened health care systems.

Can J Respir Ther. 2020; 56: 25–31. Published online 2020 Jul 23. doi: <u>10.29390/cjrt-2020-015</u>



Low level laser therapy as a modality to attenuate cytokine storm at multiple levels, enhance recovery, and reduce the use of ventilators in COVID-19

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405 NM VIOLET LASER AND CORONAVIRUS

J Photochem Photobiol B

2021 Sep;222:112282. doi: 10.1016/j.jphotobiol.2021.112282. Epub 2021 Aug 8.

Pulsed blue light inactivates two strains of human coronavirus

Chukuka S Enwemeka 1, Violet V Bumah 2, John L Mokili 3

Therefore, we investigated the effect of 405 nm, 410 nm, 425 nm and 450 nm pulsed blue light (PBL) on human alpha coronavirus HCoV-229 E and human beta coronavirus HCoV-OC43

Like SARS-CoV-2, HCoV-229E and HCoV-OC43 are single stranded RNA viruses transmitted by air and direct contact; they have similar genomic sizes as SARS-CoV-2, and are used as surrogates for SARS-CoV-2. Irradiation was carried out either at 32.4 J cm-2 using 3 mW cm-2 irradiance or at 130 J cm-2 using 12 mW cm-2 irradiance. Results: (1) At each wavelength tested, PBL was antiviral against both

coronaviruses. (2) <u>405 nm light gave the best result</u>, yielding 52.3% (2.37 log10) inactivation against HCoV-OC43 (p < .0001), and a significant 1.46 log 10 (44%) inactivation of HCoV-229E (p < .01). HCoV-OC43, which like SARS-CoV-2 is a beta coronavirus, was more susceptible to PBL irradiation than

alpha coronavirus HCoV-229E. The latter finding suggests that PBL is potentially antiviral against multiple coronavirus strains, and that, while its potency may vary from one virus to another, it seems more antiviral against beta coronaviruses, such as HCoV-OC43.



Abstract

A nonrandomized 50-person case study of COVID-19-positive patients was conducted employing (for the first time) a regimen of whole-organ deep-tissue transdermal dynamic photobiomodulation (PBM) as a primary (or exclusive) therapeutic modality

in the treatment of coronavirus. Therapy sessions comprised algorithmically alternating red (650 nm) and near-infrared (NIR; 850 nm) LEDs with an average irradiance of 11 mW/cm2 dynamically sequenced at multiple pulse frequencies. Delivered via 3D bendable polymeric pads maintaining orthogonal optical incidence to body contours over 1,000 cm2, a single 84-minute session concurrently delivered 20 kJ to the sinuses and 15 kJ to each lung at skin temperatures below 42°C. Therapeutic outcomes observed include significant reductions in the duration and severity of disease

symptoms. Acute conditions including fever, body aches (BA) and respiratory distress comprising paroxysmal coughing; lung congestion, dyspnea and hypoxia; sinus congestion; acute eye inflammation; and extreme malaise were eliminated in 41/50 patients within 4 days of commencing PBM treatments with 50/50 patients fully recovering within 3 weeks with no supplemental oxygen requirements. SpO2 concentrations improved as much as 9 points (average 2.5 points) across the entire study population. The PBM sessions required to completely resolve COVID– 19 conditions appears monotonically correlated to the time-to-treatment (TTTx) the delay between the onset of a patient's symptoms and commencing PBM therapy. In contrast, acute inflammatory symptoms were resolved within 4 days

<u>J Biophotonics.</u> 2021 Nov 22 : e202100194. doi: <u>10.1002/jbio.202100194</u> [Epub ahead of print]

PMCID: PMC8646787 PMID: <u>34658147</u> Whole-organ transdermal photobiomodulation (PBM) of COVID-19: A 50-patient case study Richard K. Williams, 1 John Raimondo, 2 David Cahn, 3 Aldon Williams, 4 and Daniel Schell 5



INFECTIOUS DISEASE: SUPPORT FOR ANTI-VIRAL LOW LEVEL LASER THERAPY



"VIRUS SETTING" 20-73-625-787

10 MINUTES TOTAL: 4 MINUTES TRANSCRANIAL 1 MINUTE NASAL/SINUSES 1 MINUTE THROAT, MAXILLARY AND MANDIBULAR GUM LINE 3 MINUTES TRACHEA DOWN TO THYMUS AND LUNG FIELDS 1 MINUTE TO HANDS OR ANY OTHER POTENTIALLY CONTAMINATED BODY SURFACES. GUT

PRE- AND POST-TREAT: PRIOR TO ANY PUBLIC EXPOSURE LIKE FLIGHTS ENTERTAINMENT EVENTS, GYM, MALLS, SUPERMARKET ETC

PERSONAL USE AND PRACTICE



ACUTE PHASE INFECTIOUS DISEASE MANAGEMENT ACUTE PHASE ACTIVE CARE: DAILY TO 3X/WEEK FOR MINIMUM 6 VISITS LASER RENTAL???

ADD TO PREVENTATIVE/MAINTENANCE CARE ON ANY FOLLOW UP VISITS

EVRL HAS MANY OTHER USES OF DAt CLIEARANCES AND USES (VAGAL ETC) without permission of Trevor Berry, DC, DACNB is prohibited.



"405 NM LOW LEVEL LASER HAS THE BENEFITS OF UV LIGHT FOR STERILIZATION WITHOUT THE HARMFUL SIDE EFFECTS. YES, GET YOUR SUN AND VITAMIN D FOR IMMUNE BOOSTING AND STRESS REDUCTION BUT USE LOW LEVEL LASER THERAPY AS A PRIMARY DEFENSE."

-DR. TREVOR BERRY DC, DACNB

ANTI-VIRAL NUTRITIONAL SUPPORT

Israeli-made dietary supplement fights off viruses in lab tests, scientists say

Mixture of 3 food supplements, each approved by the FDA, appears to be 'very capable of inhibiting replication of RNA viruses in a remarkable way,' says researcher behind study

ZINC (PROPRIETARY TRAACS ZINC GLYCINATE FOR OPTIMAL ABSORPTION) COPPER (ARGENTYN - WHOLESCRIPTS) FLAVONOIDS EX. QUERCETIN, CURCUMIN, GREEN TEA EXTRACTS = NRF2 ACTIVATOR



nhibition of Respiratory RNA Viruses by a Composition of Ionophoric Polyphenols with Metal Ions by Topaz Kreiser 1,†,Dor Zaguri 1,†,Shreya Sachdeva 1,Rachel Zamostiano 1,Josef Mograbi 2,Daniel Segal 1,3,Eran Bacharach 1,* andEhud Gazit 1,4,*

with the TAMIN D AND COVID

Israeli study offers strongest proof yet of vitamin D's power to fight COVID

Bolstering previous research, scientists publish 'remarkable' data showing strong link between vitamin deficiency, prevalent in Israel, and death or serious illness among patients

Results

Of 1176 patients admitted, 253 had records of a 25(OH)D level prior to COVID-19 infection. A lower vitamin D status was more common in patients with the severe or critical disease (<20 ng/mL [87.4%]) than in individuals

with mild or moderate disease (<20 ng/mL [34.3%] *p* < 0.001). Patients with vitamin

D deficiency (<20 ng/mL) were 14 times more likely to have severe or critical disease than

patients with $25(OH)D \ge 40 \text{ ng/mL}$ (odds ratio [OR], 14; 95% confidence interval [CI], 4 to 51; p < 0.001).

Pre-infection 25-hydroxyvitamin D3 levels and association with severity of COVID-19 illness



Amiel A. Dror , Nicole Morozov,

Published: February 3, 2022
 https://doi.org/10.1371/journal.pone.0263069
 PLOS One

COVID CONSIDERATIONS

SCIENCE January 18, 2022

Israeli vaccine study finds people still catching Omicron after 4 doses

- Four vaccine shots didn't stop people from catching Omicron, an official from an Israeli trial said.
- The trial compared people with a fourth dose to those with three, and found little difference.

The Israeli trial, launched by the Sheba Medical Center a month ago, is investigating the effect of the Pfizer booster in 154 people and the Moderna booster in 120 people, <u>per Reuters</u>.

It found that there was an "increase in antibodies" among those who got the fourth shot.

But Regev-Yochay said: "we see many infected with Omicron who received the fourth dose. Granted, a bit less than in the control group, but still a lot of infections," per the Times of Israel.



COVID CONSIDERATIONS

Spain's First Study on Omicron Finds Vaccinated People Spread COVID at Same Rate as Unvaccinated

Several governments in Spain scrapped COVID passports following the study's release. Wednesday, February 2, 2022

Secondary Attack Rates, Transmission, Incubation and Serial Interval Periods of first SARS-CoV-2 Omicron variant cases in a northern region of Spain.

Javier Del Águila-Mejía, Reinhard Wallmann, Jorge Calvo-Montes, Jesús Rodríguez-Lozano, Trinidad Valle-Madrazo, Adrian Aginagalde-Llorente

DOI: 10.21203/rs.3.rs-1279005/v1



COVID CONSIDERATIONS

CDC: Natural Immunity Offered Stronger Protection Against COVID Than Vaccines During Delta Wave

Thursday, January 20, 2022

COVID-19 Cases and Hospitalizations by COVID-19 Vaccination Status and Previous COVID-19 Diagnosis — California and New York, May–November 2021 *Early Release / January 19, 2022 / 71 Tomás M. León, PhD*1;

Ivermectin's potential to treat COVID gets a serious look in Duke University study

BY RICHARD STRADLING UPDATED JANUARY 20, 2022 1:03 PM

ACTIV 6 STUDY LOOKING AT 3 DRUGS: IVERMECTIN, FLUVOXAMINE AND All three drugs are already approved for use in humans and have a track record of being safe, Hernandez said. All

three are also easy to use at home and rarely interact with other medications, making them candidates to treat mild to moderate cases of COVID-19.

ANTIBODIES AND COVID



The effect of waning on antibody levels and memory B cell recall following SARS-CoV-2 infection or vaccination

View ORCID Profile David Forgacs, Vanessa S. Moraes, Hannah B. Hanley, View ORCID Profile Jasper L. Gattiker, A doi: https://doi.org/10.1101/2022.03.16.484099



VACCINATION CONSIDERATIONS:

- NOT DEPLETE GLUTATHIONE STORES PRIOR TO VACCINE EXPOSURE (IE. ACETAMINOPHEN)

-PRE- AND POST-TREAT WITH ERCHONIA RED LASER (BRAIN, LIVER, KIDNEY, ARM) AND GLUTATHIONE (S-ACETYL GLUTATHIONE, IV OR TOPICAL CREME) IMMUNE SETTING 20-73-465-728 WITH ERCHONIA LLLT

-ALUMINUM CHELATORS. (MAGNESIUM MALATE, ALUMINA 30 C)

-POST-TREAT	WITH THUJA OCCIDENTALIS 15 OR 30 C	. IF THE
PATIENT CAN	SPOTIFY JOE ROGAN PODCAST #1757 DR. ROBERT MALONE MD	N DILUTE
	DECEMBER 21, 2021	ACCINE
INJURT	http://vaccineimpact.com/2019/ gardasil-vaccine-on-trial-attorney-	Anthony Fauci
	robert-f-kennedy-jr-exposes-merck- Duplication, citation or distribution of this material in whole or in part	Bill Gates, Big Pharma, and the Giobal War on Democracy and Public Health Robert F. Kennedy Jr. New York THES BESTRILING ATHON Children's @

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NUTRITIONAL IMMUNE SUPPORT

DEFENSE BOOSTING MEDPAX:

- **1 OMEGAMONOPURE MD AM / 1 ONCOPLEX ES PM**
- 1 XCELLENT A 3000 AM / 1 XCELLENT C PM
- 1 IMMUNOTIX 250 AM / 1 K2-D3 5000 PM
- 1 PROBIOMAX IG 26 DF 1 AM / 1 DIOVASC PM
- 1 HISTDAO AM / 1 RESVERATIN PLUS PM

PRIMARY LIGAND RECEPTOR BINDING COMPETITION: GLYCYRRHIZIN (LICORICE), QUERCITIN, DIOSMIN, GLUCOSAMINE, CBD

*TAKE IMMUNOTIX, HISTDAO AND OMEGAMONPURE BEFORE BREAKFAST IN AM

BOOST NATURAL IMMUNITY WITH SLEEP HYGIENE, EXERCISE, STRESS REDUCTION, PROPER DIET/INTERMITTENT FASTING, LAURICIDIN

LOW LEVEL LASER PROTOCOLS: BASE BRAIN, VAGAL STIM/ BARRIER SYSTEMS, RESPIRATORY SYSTEM IMMUNE Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.





SUGGESTION - ACUTE PHASE SUPPORT MEDPAX SUMMARY:

ZINC GLYSINATE 1 AM/ 1 PM PRE-ORDER **BOXES OF** THESE MEDPAX NAC 1 AM/ 1 PM TO HAVE **IMMUNOTIX 250 1 AM/ 1 PM** XYMOGEN WAITING IN YOUR OFFICE VIRAGRAPHIS 1 AM/ 1 PM TO SELL TO PATIENTS FOR XCELLENT C 1 AM/ 1 PM (IF OUT: BIO C 1:1) IMMEDIATE ACUTE PHASE **VITAMIN K2-D3 5000 1 AM** SUPPORT

MELATONIN CR 1 PM

SILVER HYDROSOL BID (NASAL SPRAY/NETI POT/GARGLE/ SWALLOW). BID

ERCHONIA EVRL OR RED BASE VIRAL RESPIRATORY SYSTEM PROTOCOL. 1-2X/DAY



ANDROGRAPHIS ANTIVIRAL



Broad-spectrum antiviral properties of andrographolide Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry CKDAVONE' is in childred.

INFECTIOUS DISEASE: PATHOGENS AND NEURODEGENERATION

Appl Nanosci (2014) 4:859-868 DOI 10.1007/s13204-013-0266-1

ORIGINAL ARTICLE

Antibiofilm efficacy of silver nanoparticles against biofilm of extended spectrum b-lactamase isolates of Escherichia coli and Klebsiella pneumoniae

Mohammad Azam Ansari • Haris M. Khan • Aijaz A. Khan • Swaranjit Singh Cameotra • Ruchita Pal



Journal of Industrial and Engineering Chemistry 19 (2013) 614–619 Contents lists available at SciVerse ScienceDirect Journal of Industrial and Engineering Chemistry

journal homepage: www.elsevier.com/locate/jiec



Biofilm-inactivating activity of silver nanoparticles: A comparison with silver ions

Hee-Jin Park^a, Soomin Park^a, Jinkyu Roh^b, Sujin Kim^c, Kyunghee Choi^c, Jongheop Yi^a, Younghun Kim^{b,*}, Jeyong Yoon^{a,**}

A Novel Signaling Network Essential for Regulating Pseudomonas aeruginosa Biofilm Pevelopment DRB20 COUPON CODE

Olga E. Petrova, Karin Sauer* Department of Biological Sciences, Binghamton University, Binghamton, New York, United States of Americ

-



COVID

J Endocrinol Invest

2021 Jun 24;1-16. doi: 10.1007/s40618-021-01614-4. Online ahead of print.

Vitamin D supplementation and clinical outcomes in COVID-19: a systematic review and meta-analysis

R Pal # 1, M Banerjee # 2, S K Bhadada 3, A J Shetty 1, B Singh 4, A Vyas 5

'Reduced Mortality, ICU Admission': Use of Vitamin D To Treat Covid-19 Bores Positive Results, Finds PGIMER Study

Results: We identified 13 studies (10 observational, 3 RCTs) pooling data retrieved from 2933 COVID-19 patients. Pooled analysis of unadjusted data showed that **vitamin D use in COVID-19 was significantly associated with reduced ICU admission/ mortality (OR 0.41, 95% CI: 0.20, 0.81, p = 0.01, I2 = 66%, randomeffects model). Similarly, on pooling adjusted risk estimates, vitamin D was also found to reduce the risk of adverse outcomes** (pooled OR 0.27, 95% CI: 0.08, 0.91, p = 0.03, I2 = 80%, random-effects model). Subgroup analysis showed that vitamin D supplementation was associated with improved clinical outcomes only in patients receiving the drug post-COVID-19 diagnosis and not in those who had received vitamin D before diagnosis.





Role of von Willebrand Factor in COVID-19 Associated Coagulopathy.

Mei ZW, van Wijk XMR, Pham HP, Marin MJ. J Appl Lab Med. 2021 Apr 30:jfab042. doi: 10.1093/jalm/jfab042. Online ahead of print. PMID: 33930144

The high levels of both vWF antigen and activity have been clinically correlated with worse outcomes. Furthermore, the severity of a COVID-19 infection appears to reduce molecules that regulate vWF level and activity such as ADAMT-13 and high density lipoproteins (HDL). Finally, studies have suggested that patients with blood group O (a blood group with lower than baseline levels of vWF) have a lower risk of infection and disease severity compared to other blood groups; however, more studies are needed to elucidate the role of vWF.

Ann Lab Med. 2017 Mar; 37(2): 155–158. Published online 2016 Dec 20. doi: <u>10.3343/alm.2017.37.2.155</u> Effect of Vitamin D Status on Von Willebrand Factor and ADAMTS13 in Diabetic Patients on Chronic Hemodialysis

Keren Cohen-Hagai, M.D.,1 Gloria Rashid, Ph.D.,2 Yael Einbinder, M.D.,1,3 Meital Ohana, M.S.,1 Sydney Benchetrit, M.D.,1,3 and Tali Zitman-Gal, Ph.D.1,3



LOW LEVEL LASER THERAPY AND ORGAN SUPPORT

PRE AND POST INFECTIOUS DISEASE SUPPORT: STUDIES SHOW PRE-TREATING ORGANS WITH LLLT CAN BE TISSUE AND ORGAN PROTECTIVE (EX. BRAIN IN STROKE, HEART IN MI ETC)



LARGE INTESTINE

2 MIN



ASER YOUR VASCULAR SYSTEM GLOBALLY AND LUNG FIELDS IMMUNE :20-73-465-728 without permission of Trevor Berry, DC, DACNB is prohibited.

STERNAL/LEFT
DYSAUTONOMIA AND COVID

Source: Journal of Physiology

August 9, 2021

New research published in the *Journal of Physiology* found that otherwise healthy young people diagnosed with COVID-19, regardless of their symptom severity, have problems with their nervous system when compared with healthy control subjects.

Specifically, the system that oversees the fight-or-flight response, the sympathetic nervous system, seems to be abnormal (overactive in some instances and underactive in others) in those recently diagnosed with COVID-19.

Clinical considerations dysautonomia:

- -Tilt table to sub ANS, OVAR
- -Erchonia Red (Vagal or Ad Brain to inhibit sympathetics), Violet (to promote sympathetics). SCM vagus, GI (Percussor), Ethmoid (combo smell tx), throat
- -Cerebellar purkinje activation (isometrics)
- -VORs (Fixation with "No-No's" for parasympathetics, "Yes-Yes" for sympathetics
- -OPK (Horizontal, vertical, side posture with decline in phase and counter phase
- -Watch for other common pathogen triggers (Mold, Lyme etc)
- -Hydration. Adrenal Support (Underactive Addison's and Polyglandular AI) Vs Overactive (Rule out tumor pathology)

EVRL VIOLET-RED LASER: AUTONOMIC CONSIDERATIONS HEART RATE VARIABILITY

ASSESSING THE AUTONOMIC EFFECT OF VAGAL NERVE STIMULATION WITH LOW LEVEL LASERS BY HEART RATE VARIABILITY MACHADO ET AL.

ABSTRACT: VAGUS NERVE STIMULATION (VNS) HAS BEEN APPROVED TO TREAT REFRACTORY EPILEPSY, AND FOR OTHER CONDITIONS. THE INVASIVE NATURE OF THE ELECTRICAL STIMULUS, WHICH REQUIRES SURGICAL IMPLANTATION OF ELECTRODES AROUND THE CERVICAL VAGUS NERVE, IS A TECHNICAL LIMITATION. FIFTEEN NORMAL PARTICIPANTS FROM 22 TO 46 YEARS DIVIDED INTO THREE GROUPS OF 5 SUBJECTS EACH, PAIRED IN AGE AND GENDER, WERE STUDIED APPLYING VNS USING LLLT BY LASERS OF DIFFERENT FREQUENCIES; RED LASER (5 SUBJECTS), VIOLET LASER (5 SUBJECTS) AND RED-VIOLET COMBO (5 SUBJECTS). THE STUDY INCLUDED 3 EXPERIMENTAL CONDITIONS; BASAL RECORD (10 MIN), VNS (10 MIN), AND POST-VNS (10 MIN). THE LF/HF RATIO WAS CONSIDERED BECAUSE IT PROVIDES A MEASUREMENT OF PARA-SYMPATHETIC BALANCE. 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.**

INSPIRATION IS SYMPATHETIC TONE, EXPIRATION PARASYMPATHETIC (1:2) ACH BATH ON VAGAL TONE. COMA PROGNOSIS/THERAPEUTIC WINDOW, 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 AND NOT OTHER LIGHT DEVICES



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 LLLT MAY EFFECTIVELY TARGETS DISEASED CELLS

IMMUNOLOGY OVERVIEW

3 LAYERS OF PROTECTION

1. BARRIER SYSTEMS: PHYSICAL BARRIER PLUS EXTRACTION SKIN - SHEDDING, EYES - TEARS, RESPIRATORY SYSTEM - CILIA, SNEEZE, COUGH, GUT - ACID, ENZYMES, CELL TURNOVER, DEFECATION, VOMITING, GENITOURINARY - URINATION. TECHNICALLY PART OF INNATE SYSTEM

2. INNATE IMMUNE SYSTEM = BORN WITH IT. NON-SPECIFIC REACTION TO FOREIGN INTRUDERS. CELL TYPES (WBC/LEUKOCYTES): MACROPHAGES/MONOCYTES IN BLOOD - BOUNCERS, LOCAL OR MIGRATING NEUTROPHILS - PHAGOCYTOSIS, PRIMARY WBC RESPONSE, ELEVATED RATIOS BACTERIA FOCUS, FIRST RESPONDERS THAT CAN INFILTRATE TISSUES EOSINOPHILS - MULTICELLULAR PARASITES, WORMS (PATHOGENS TOO BIG FOR PHAGOCYTOSIS). SEEN IN MOLD AND LYME ALSO. WATCH FOR INCREASED ENVIRONMENTAL ALLERGIES, MUCOUS PRODUCTION, CLEARING THROAT MAST CELLS - MUCOUS LINING, HISTAMINE RESPONSES, WOUND HEALING BASOPHILS - LEAST COMMON, PREVENT CLOTTING (CONTAIN HEPARIN), SOME ALLERGY AND PARASITE FUNCTION DENDRTIC CELLS - SKIN AND LININGS, BECOME ANTIGEN PRESENTING CELLS TO LYMPHOCYTES NK (NATURAL KILLER) CELLS - DESTROY OUR OWN CELLS THAT HAVE BECOME DEFECTIVE SUCH AS CANCER CELLS AND OUR OWN CELLS THAT HAVE BEEN



IMMUNOLOGY OVERVIEW

INNATE SYSTEM CONTINUED...

COMPLIMENT SYSTEM: APPROXIMATELY 20 PROTEINS IN CIRCULATION THAT COMPLIMENT THE ANTIBODY SYSTEMS IN DESTROYING PATHOGENS. WHEN AN ANTIBODY BINDS TO A PATHOGEN IT TRIGGERS THE COMPLIMENT SYSTEM TO PROMOTE POTENT LOCAL INFLAMMATORY REACTIONS AND AMPLIFIES THE DEFENSE RESPONSES

ADAPTIVE/ACQUIRED IMMUNE SYSTEM:

THE DENDRITIC/MACROPHAGE ANTIGEN PRESENTING CELL CIRCULATES TO B AND T cell REGIONS (IE. LYMPH TISSUE). THIS ACTIVATES B cell ANTIBODY RESPONSE WITH MHC2 COMPLEX TO START REPLICATING ANTIBODIES SPECIFIC TO THAT ONE ANTIGEN (FOREIGN INVADER) MAKING EFFECTOR (ANTIBODY PRODUCING FACTORIES) CALLED PLASMA CELLS, AND MEMORY CELLS. THIS SYSTEM ALSO WORKS WITH T HELPER CELLS WITH CD4+ BINDING TO MHC2 COMPLEX TO PROMOTE THE SPECIFIC ANTIBODY RESPONSE AND AMPLIFY THE CYOTOXIC CELL RESPONSE. THINK TH2

THE "SWAT TEAM" TH1/CD8+ CELL KNOWN AS THE CYOTOXIC CELL KICKS IN TO ATTACK THE PATHOGEN INVADED OR CANCER CELL (KILLS BAD CELLS). THESE ARE ATTRACTED MHC1 COMPLEX. THINK TH1

TH17/IL17: INVOLVED IN CELL MEDIATED IMMUNITY AND FUNGAL INFECTIONS BUT CHRONIC POLARIZATION INVOLVED IN MANY AUTOIMMUNE AND CHRONIC INFLAMMATORY CONDITIONS

T CELL POLARIZATION

-IF A PATHOGEN GETS PAST THE BARRIER SYSTEMS/INNATE IMMUNE RESPONSE, THE ANTIGEN PRESENTING SYSTEM (IE. DENDRITIC/MACROPHAGE CELLS) WILL STIMULATE B cells TO PROMOTE ANTI-BODY PRODUCTION AND T cells TO FACILITATE B ANTI-BODY PRODUCTION AND SEND OUT CYTOTOXIC T cells

-IF THE PATHOGEN PRESENTED IS A VIRUS OR BACTERIA, IL-12 WILL BE SECRETED AND A TH1 CELL WILL BE MADE. THIS ALSO HELPS REGULATE MACROPHAGES AND MONOCYTES

-IF IT'S ALLERGY, PARASITE OR WORMS, IL-4 WILL BE SECRETED AND A TH2 CELL WILL BE MADE. THIS ALSO HELPS REGULATE EOSINOPHILS, BASOPHILS AND MAST CELLS

-IF IT'S CHRONIC INFLAMMATION AND TISSUE DEBRIS, IL-6, IL-21 AND IL-23 WILL BE SECRETED AND A TH17 CELL WILL BE MADE

-IN ALL THREE OF THESE SCENARIOS T REG CELLS, IL-10 AND TGF-B WILL BE SECRETED IN AN ATTEMPT TO PREVENT AN OVER EXUBERANT RESPONSE. THIS IS ONE OF THE MANY STRENGTHS OF INVOLVING LOW LEVEL LASER FOR PROPER IMMUNE REGULATION IN PATHOGEN EXPOSURE AND PROPER RESOLUTION



IMMUNOLOGY: LASERS AND IMMUNOREGULATION

Most studies have shown that low-power lasers can affect $\mathrm{TGF}\beta1~\mathrm{signaling}$ which is the most important signaling in the treatment of renal fibrosis.

Lasers Med Sci. 2020 Spring; 11(2): 220–225. Published online 2020 Mar 15. doi: 10.34172/jlms.2020.36

PMCID: PMC7118498 PMID: <u>32273966</u>

The Effect of Low-Power Laser Therapy on the TGF/β Signaling Pathway in Chronic Kidney Disease: A Review

Behnaz Ahrabi, 1 Maryam Bahrami,

Front. Immunol., 02 February 2022 | https://doi.org/10.3389/fimmu.2021.789426 Photobiomodulation Therapy Restores IL-10 Secretion in a Murine Model of Chronic Asthma: Relevance to the Population of CD4+CD25+Foxp3+ Cells in Lung

Aurileia Aparecida de Brito1, Tawany Gonçalves Santos2,



Abstract

IMMUNOLOGY AND LILT

As low-level laser therapy immune cells responses are not always clarified, this study aimed to evaluate cytokines and immune cells profile after low-level laser therapy (LLLT) on arthritis-induced model. Arthritis was induced in C57BL/6 mice divided into five groups: euthanized 5 hours after inflammation induction; untreated; dexamethasone treated; LLLT at 3 Jcm-2; LLLT at 30 Jcm-2. Cytokine measurements by enzyme-linked immunosorbent assay and mRNA cytokine relative levels by real-time quantitative polymerase chain reaction were performed

with arthritic ankle (IL-1β, IL-6, TNF-α, IL-10 and TGF-β). Macrophages, dendritic cells, natural killer cells, lymphocytes CD4+, CD8+, Treg and costimulatory proteins were quantified in proximal lymph node by flow cytometry. Data showed decrease in all cytokine levels after LLLT and alteration in mRNA relative levels, depending on the energy density used. LLLT was able to increase of immune cell populations analyzed in the lymph node as well as costimulatory proteins expression on macrophages and dendritic cells. Treg TCD4+ and TCD8+ population enrichment were observed in LLLT at 3 and 30 Jcm-2 groups, respectively. Furthermore, Treg TCD8+ cells expressing higher levels of CD25 were observed at LLLT at 30 Jcm-2 group. Our results indicate that LLLT could change the inflammatory course of arthritis, tending to accelerate its resolution through immune cells photobiostimulation.

J Biophotonics

2019 Feb;12(2):e201800120. doi: 10.1002/jbio.201800120. Epub 2018 Oct 18.

Modulation of immune response to induced-arthritis by lowlevel laser therapy

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*T REG CELLS RELEASE TGF-B AND IL-10 FOR EVERY TYPE OF T cell RESPONSE TO PREVENT THEM FROM BECOMING TOO DOMINANT