# WHAT IS THE FIRST THING YOU SHOULD DO IN THE EVENT OF A TRAUMATIC BRAIN INJURY OR STROKE???

# *PRE-TREAT!*



## PRE-TREATING WITH TRANSCRANIAL LOW LEVEL LASER THERAPY TO IMPROVE BRAIN INJURY OUTCOMES

Dose Response. 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.**

Agrawal T1, Gupta GK2, Rai V3, Carroll JD4, Hamblin MR5.

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 Low-level 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.***

# PRECONDITIONING WITH TRANSCRANIAL LOW 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!

Magnes Res. 2017 Aug 1;30(3):88-97. doi: 10.1684/mrh.2017.0427. Magnesium enhances the beneficial effects of NK1 antagonist administration on blood-brain barrier permeability and motor outcome after traumatic brain injury. Ameliorate JL1, Ghabriel MN1, Vink R2.



## TBI

**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

IN SUMMARY: ASIDE FROM PRE-TREATING THE BRAIN FOR BETTER OUTCOMES, IF YOU OR YOUR PATIENT BASE HAS SUFFERED EVEN A MILD TRAUMATIC BRAIN INJURY YOU ARE AT SERIOUS RISK OF LEAKY BARRIER SYSTEMS (GUT AND BBB) AS WELL AS DEMENTIA.
AGGRESSIVE TREATMENT SCHEDULES WITH YOUR ERCHONIA LASERS USING THE MASTER BRAIN AND VAGAL PROTOCOLS SHOULD BE PURSUED!

**Association of Mild Traumatic Brain Injury With and Without Loss of Consciousness With Dementia in US Military Veterans**

Deborah E Barnes 1 2 3, Amy L Byers 1 2 3, Raquel C Gardner 1 4, Karen H Seal 1 2 5, W John Boscardin 1 5, Kristine Yaffe 1 2 3 4

- PMID: 29801145 PMCID: PMC6143113 DOI: 10.1001/jamaneurol.2018.0815





# ACUTE PHASE TBI

## Summary: Acute Phase TBI Management (<4 Weeks)


**24 HOUR WATER FAST ON DAYS 7, 14, 21 AND 28**

**AM:**
**1 OMEGA MONOPURE CURCUMIN EC**
**1 S-ACETYL GLUTATHIONE**
**2 ACTIV NUTRIENTS WITHOUT COPPER AND IRON**
**1 MEMORALL**
**1 ALAMAX CR**

**PM:**
**1 MELATONIN CR**
**1 NRF2 ACTIVATOR**
**1 K2-D3**
**2 HEMP MONOPURE**
**1 RESVERATIN PLUS**

**1 SCOOP OPTIMAG NEURO DAILY**

*** 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!**

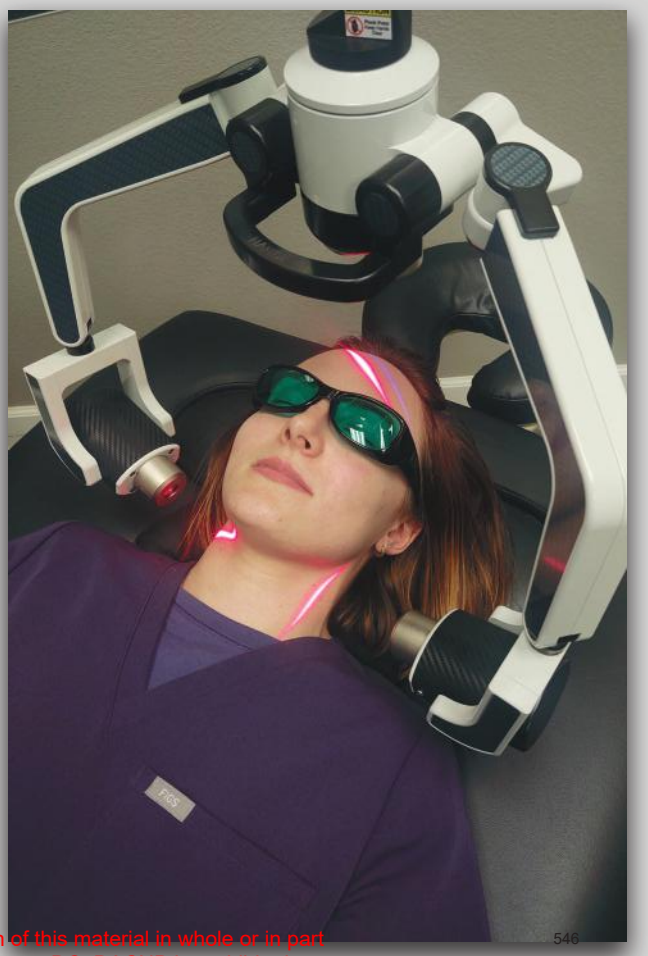


## ACUTE PHASE TBI ERCHONIA FX APPLICATION

**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**

* WATCH 'STROBE" EFFECT OVER EYES
* IF CONCERN OVER METABOLIC FATIGUE DO VAGAL STIM ONLY OR DOSE TO 2
* MINUTES FOR INITIAL TREATMENT TRIAL

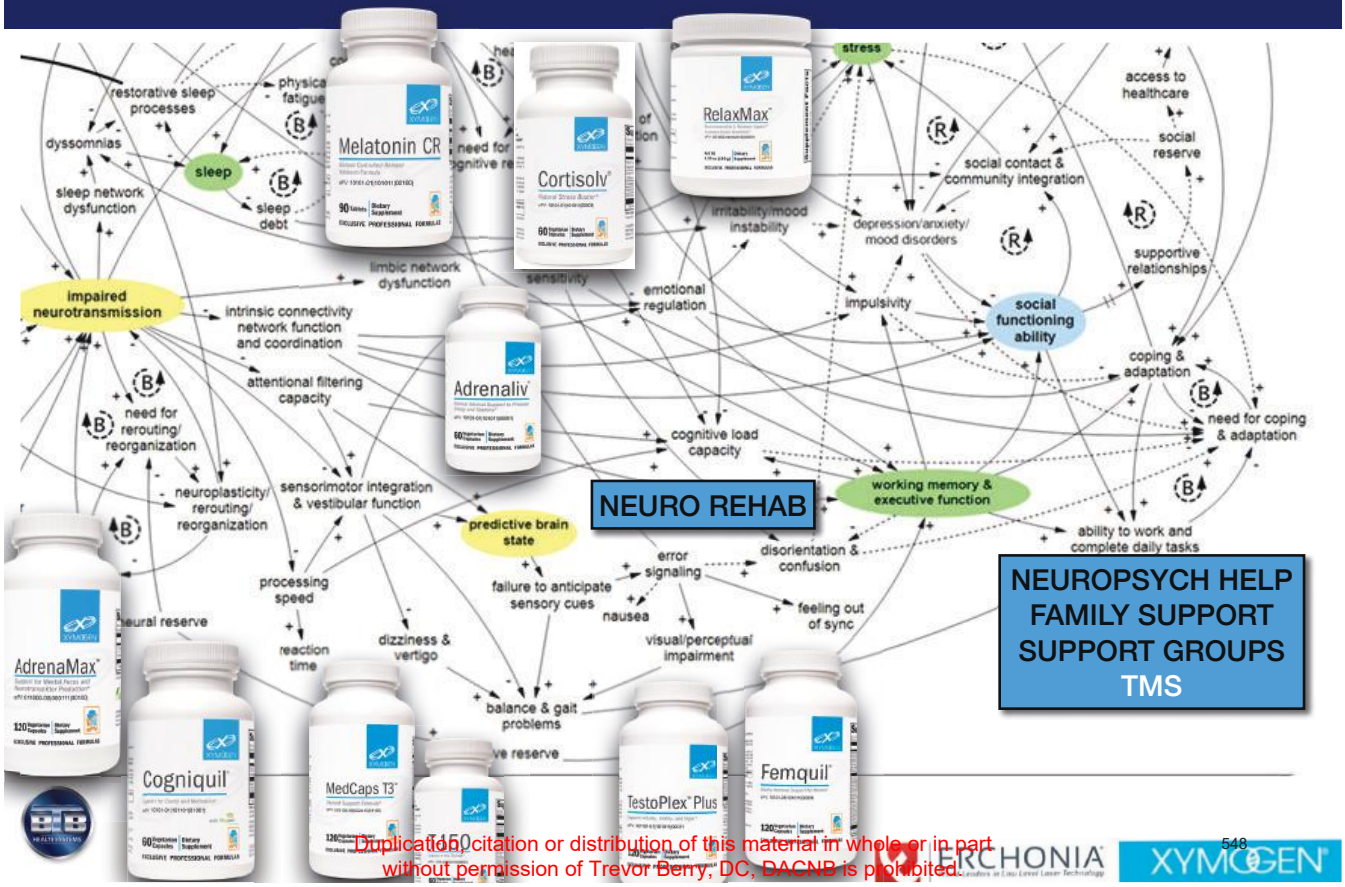


# ABBOTT RECEIVES FDA 510(K) CLEARANCE FOR THE FIRST RAPID HANDHELD BLOOD TEST FOR CONCUSSIONS

- The test to help evaluate mild traumatic brain injury (TBI), commonly known as concussion, produces **a result within 15 minutes after a plasma sample is inserted and will run on Abbott's i-STAT™ Alinity™ handheld device**
- Having a blood test available could help eliminate wait time in the emergency room and **could reduce the number of unnecessary CT scans by up to 40%**
- The test **simultaneously measures biomarkers UCH-L1 (Ubiquitin C-terminal hydrolase - L1) and GFAP, proteins found in the blood after a concussion or head trauma**
- Building on this initial clearance, Abbott is also working on a test that would use whole blood on i-STAT at the point of care, and developing a test for its Alinity™ i and ARCHITECT® core laboratory instruments under FDA breakthrough designation



## THE DYNAMICS OF CONCUSSION



# EXERCISE POST MTBI

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, activation of alternative inflammatory responses and enhancement of neurogenesis. In contrast, early initiation of exercise

**CLINICAL TAKE HOME: PHYSICAL EXERCISE CAN BE AN INDICATED INTERVENTION POST TBI PROMOTING CELLULAR MECHANISMS SUCH AS INDUCING BDNF AND NEUROIMMUNOREGULATION, PROMOTING NEUROCHEMISTRY OUTPUT SUCH AS DOPAMINE AND SEROTONIN AND MUCH MORE. HOWEVER, IT IS CRITICAL TO MONITOR AUTONOMICS AND SYMPTOM STATUS TO NOT EXCEED METABOLIC THRESHOLD!**

**LOW LEVEL LASER THERAPY CAN DRAMATICALLY IMPROVE METABOLIC THRESHOLD**



# MRI AND TBI

## Study Finds Brain Lesions on MRI Linked to Years of Playing Football

Featured Neurology Neuroscience · November 26, 2021

***Summary:*** *White matter hyperintensities were more common in athletes who played more contact sports or had more head injuries and concussions during their sporting careers.*

***Source:*** *AAN*

**Certain markers of injury to the brain's white matter, called white matter hyperintensities, can be seen on brain scans. A new study finds that brain scans taken during the lifetimes of athletes in contact sports, compared to changes in their brains at autopsy, showed that white matter hyperintensities were associated with neuropathological changes.**

The research is published in *Neurology*.

# Massey TBI Grand Challenge

Thanks to funding from the Joyce and Don Massey Family Foundation, MCIRCC hopes to improve treatment and survival rates for patients that experience a TBI. The Massey family had their own experience with TBI after a car accident injured mother and wife, Joyce Massey.

**The funding also allows MCIRCC to host the Joyce Massey TBI Summit each fall. The conference brings together the nation's leading experts and researchers to discuss the most pressing challenges facing TBI care and how to work together to find innovative solutions.**

**THE FIVE FINALISTS THAT RECEIVED FUNDING:**
- **VALPROIC ACID**
- **POINT OF CARE DEVICE TO MONITOR BLOOD BIOMARKERS IN REAL TIME**
- **ACUTE PHASE INTRANASAL INSULIN THERAPY**
- **DEVICE FOR CONSTANT REAL TIME SYSTOLIC BLOOD PRESSURE MONITORING**

**-USING LIGHT THERAPY WAVELENGTHS TO TARGET CELLS**





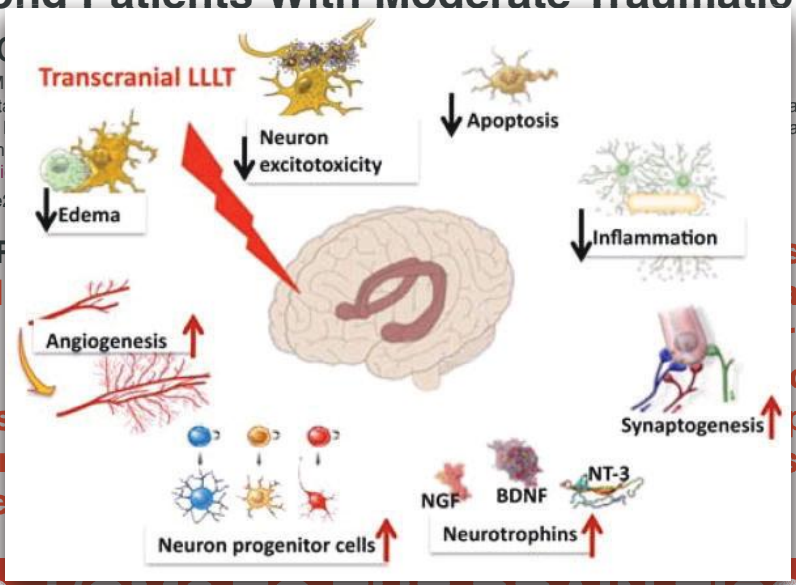
September 14, 2020

## Effect of Transcranial Low-Level Light Therapy vs Sham Therapy Among Patients With Moderate Traumatic Brain Injury

A Randomized

Maria Gabriela Figueiro Longo, M
Jonathan Welt, BS[4]; Arman Avesta
Calero, PhD[1]; Blair A. Parry, BA[1];
Michael Hamblin, PhD[1]; Benjamin
amati, PhD[1]; Isabel Chico-
ael Lev, MD[1]; Jarone Lee, MD[1];
Author Affiliations Article Informati

*JAMA Netw Open.* 2020;3(9):e




**Conclusions and**
**s feasible in all patients and did ltered multiple diffusion tensor in the late subacute stage. o date that light therapy engages physiologic factors of moder s the biomarker of therapeutic re**

**1.3 J/CM2 TO THE BRAIN TISSUE**

# VAGAL NERVE STIMULATION IN TBI

Neurocrit Care. 2016 Apr;24(2):308-19. doi: 10.1007/s12028-015-0203-0.

## Vagus Nerve Stimulation and Other Neuromodulation Methods for Treatment of Traumatic Brain Injury.

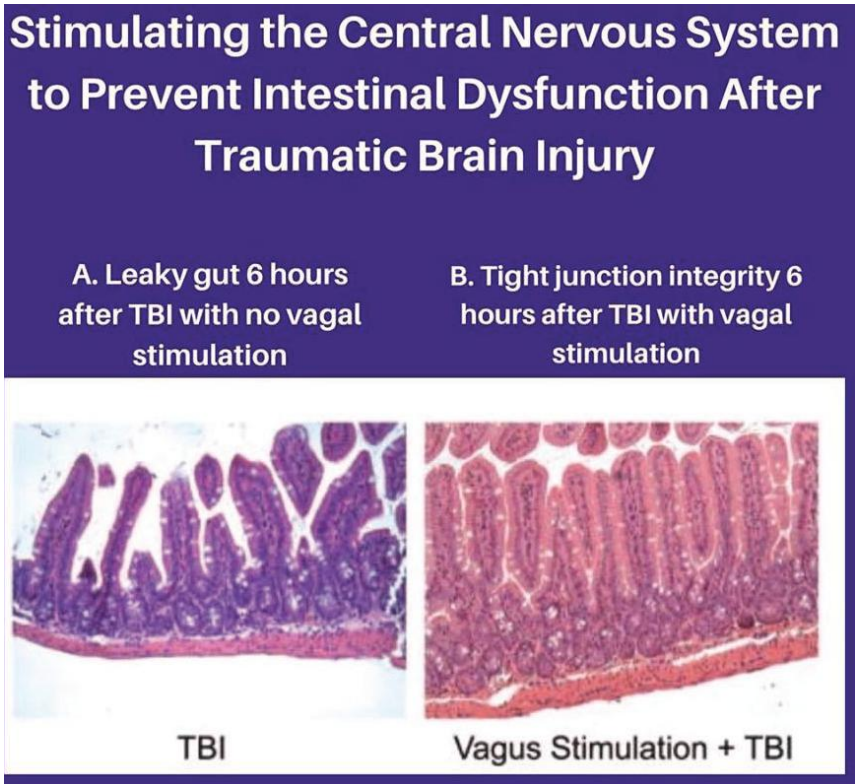
Neren D1,2, Johnson MD3, Legon W4, Bachour SP1, Ling G5, Divani AA6,7,8.
**Author information Abstract**

The objective of this paper is to review the current literature regarding the use of vagus nerve stimulation (VNS) in preclinical models of traumatic brain injury (TBI) as well as discuss the potential role of VNS along with alternative neuromodulation approaches in the treatment of human TBI. Data from previous studies have demonstrated VNS-mediated improvement following TBI in animal models. In these cases, ***VNS was observed to enhance motor and cognitive recovery, attenuate cerebral edema and inflammation, reduce blood brain barrier breakdown, and confer neuroprotective effects.*** Yet, the underlying mechanisms by which VNS enhances recovery following TBI remain to be fully elucidated. Several hypotheses have been offered including: a noradrenergic mechanism, reduction in post-TBI seizures and hyper-excitability, anti-inflammatory effects, attenuation of blood-brain barrier breakdown, and cerebral edema. We present other potential mechanisms by which VNS acts including enhancement of synaptic plasticity and recruitment of endogenous neural stem cells, stabilization of intracranial pressure, and interaction with the ghrelin system. In addition, alternative methods for the treatment of TBI including deep brain stimulation, transcranial magnetic stimulation, transcranial direct current stimulation and focused ultrasound stimulation are discussed. Although the primary... these findings can be tra...

**VNS improves TBI outcomes**



VAGAL NERVE STIMULATION IN TBI



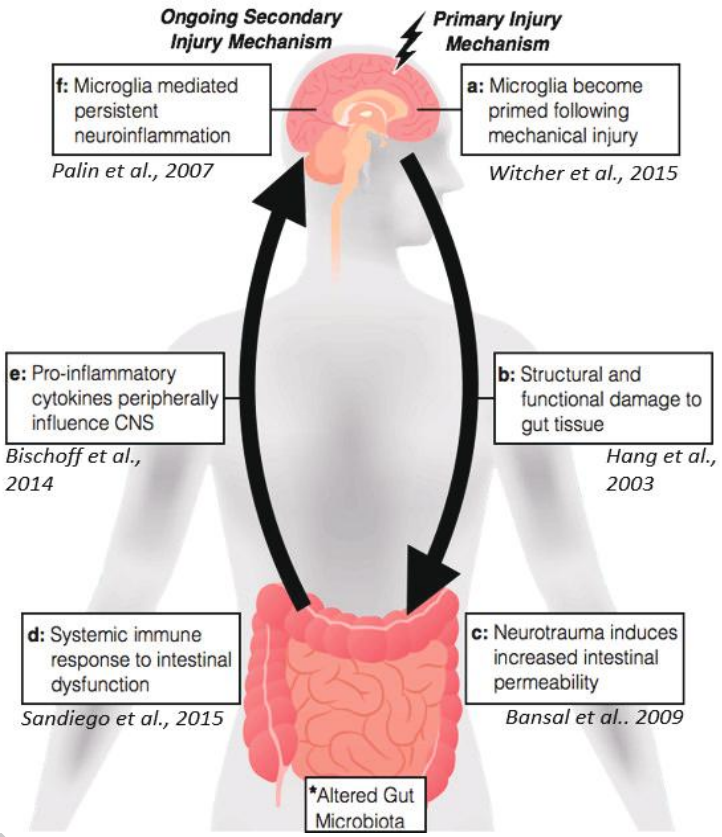

**Stimulating the central nervous system to prevent intestinal dysfunction after traumatic brain injury.**

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 TBI-induced intestinal permeability. Furthermore, vagal stimulation increased enteric glial activity and may represent the pathway for central nervous system regulation of intestinal permeability.**

# BRAIN GUT AXIS NEGATIVE FEEDBACK LOOP FOLLOWING TRAUMATIC BRAIN INJURY




**TBI AND NEURODEGENERATIVE CONDITIONS ARE LEADING TO LEAKY GUT AND BBB. BREAK THE CYCLE WITH YOUR BRAIN AND VAGAL STIM PROTOCOLS**

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# Management of a "leaky gut"

- 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 MICRO 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 (1-2 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.**

# LEAKY BLOOD BRAIN BARRIER BASE PROTOCOL

**ERCHONIA RED LASER BASE BRAIN 1-10-40-60. MINIMUM 6 (12+) VISITS TRANSCRANIAL BASE PROTOCOLS (UP TO 12 MINUTES HAND HELD/10 MINUTES FX)**

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

**XYMOGEN SUPPORT (2 MONTH MINIMUM):**

- **MEMORALL 1 DAILY**
- **GASTRACID 1 DAILY (BEFORE PROTEIN MEALS)**
- **OPTIMAG NEURO 1 SCOOP DAILY**
- **C0QMAX OMEGA 100 MG 2 DAILY**

**60 DAY GLUTALOEMINE**

**ONCOPLEX ES 1 DAILY**

**DIOVASC 1 DAILY**

**CURCUPLEX-95 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 (NEXT PAGE)...**



# CLINICAL CONSIDERATIONS CNS TIGHT JUNCTION BARRIER AS SUMMARIZED BY "HOW TO FIX A LEAKY BLOOD BRAIN BARRIER"

**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)**

**SUPPLEMENTS TO PROTECT AND REPAIR:**

- **ALCAR ( ACETYL L CARNITINE) MEMORALL**
- **ALPHA LIPOIC ACID. ALAMAXCR**
- **ALPHA GPC (GLYCEROPHOPHOCHOLINE) NO XYMOGEN**
- **ANGELICA (DONG QUAI) FEMALE SUPPORT PRODUCTS**
- **ASTRALAGUS. NO XYMOGEN**
- **ASTAXANTHIN. OMEGAPURE KRILL**
- **APIGENIN. NO XYMOGEN**
- **METHYLATED B'S. B ACTIV (LOWER HOMOCYSTEINE)**
- **BERBERINE. BERBEMYCIN**
- **BITTER MELON. GLUTALOEMINE, GASTRACID, MEDCAPS**
- **BUTYRATE. KETONX**

Expert Rev Mol Diagn. 2018 Feb;18(2):165-180. doi: 10.1080/14737159.2018.1428089. Epub 2018 Jan 23.

# An update on diagnostic and prognostic biomarkers for traumatic brain injury.

Wang KK1, Yang Z1, Zhu T1, Shi Y2, Rubenstein R3, Tyndall JA4, Manley GT5,6.
**Author information**

**Abstract**

Traumatic brain injury (TBI) is a major worldwide neurological disorder of epidemic proportions. To date, there are still no FDA-approved therapies to treat any forms of TBI. Encouragingly, there are emerging data showing that biofluid-based TBI biomarker tests have the potential to diagnose the presence of TBI of different severities including concussion, and to predict outcome. Areas covered: The authors provide an update on the current knowledge of TBI biomarkers, **including protein biomarkers for neuronal cell body injury (UCH-L1, NSE), astroglial injury (GFAP, S100B), neuronal cell death (αII-spectrin breakdown products), axonal injury (NF proteins), white matter injury (MBP), post-injury neurodegeneration (total Tau and phospho-Tau), post-injury autoimmune response (brain antigen-targeting autoantibodies)**, and other emerging non-protein biomarkers. The authors discuss biomarker evidence in TBI diagnosis, outcome prognosis and possible identification of post-TBI neurodegernative diseases (e.g. chronic traumatic encephalopathy and Alzheimer's disease), and as theranostic tools in pre-clinical and clinical settings. Expert commentary: A spectrum of biomarkers is now at or near the stage of formal clinical validation of their diagnostic and prognostic utilities in the management of TBI of varied severities including concussions. TBI biomarkers could serve as a theranostic tool in facilitating drug development and treatment monitoring.





PROGNOSTIC BIOMARKERS FOR TBI

**Mol Neurodegener. 2018** Apr 4;13(1):17. doi: 10.1186/s13024-018-0249-5.

# Apolipoprotein E4 impairs spontaneous blood brain barrier repair following traumatic brain injury.

Main BS1, Villapol S1, Sloley SS1, Barton DJ1, Parsadanian M1, Agbaegbu C1, Stefos K1, McCann MS1, Washington PM1, Rodriguez OC2, Burns MP3,4.
**Author information Abstract**

**BACKGROUND:**

Traumatic Brain Injury (TBI) is a major cause of disability and mortality, to which there is currently no comprehensive treatment. Blood Brain Barrier (BBB) dysfunction is well documented in human TBI patients, yet the molecular mechanisms that underlie this neurovascular unit (NVU) pathology remains unclear. The apolipoprotein-E (apoE) protein has been implicated in controlling BBB integrity in an isoform dependent manner, via suppression of Cyclophilin A (CypA)-Matrix metallopeptidase-9 (MMP-9) signaling cascades, however the contribution of this pathway in TBI-induced BBB permeability is not fully investigated.

**METHODS:**

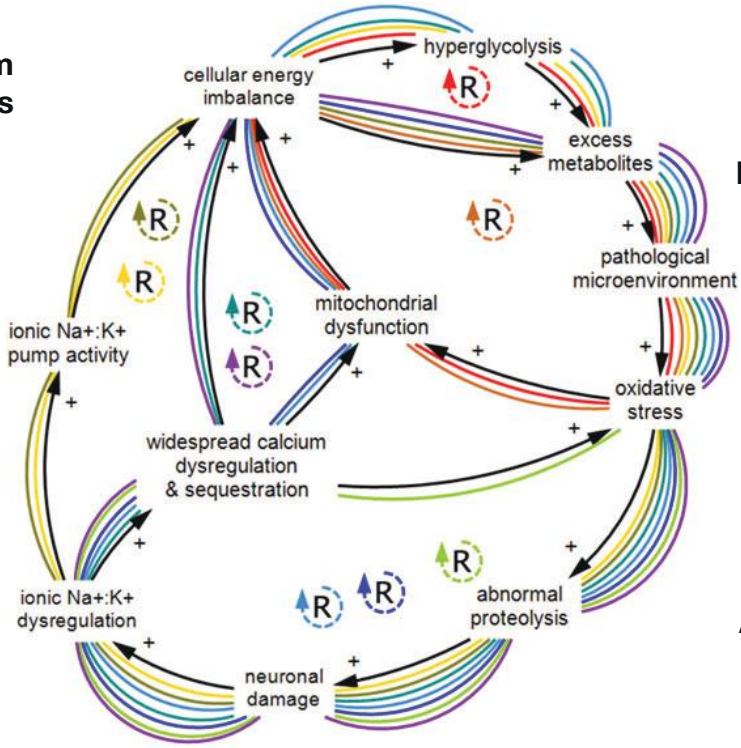
We exposed C57Bl/6 mice to controlled cortical impact and assessed NVU and BBB permeability responses up to 21 days post-injury. We pharmacologically probed the role of the CypA-MMP-9 pathway in BBB permeability after TBI using Cyclosporin A (CsA, 20 mg/kg). Finally, as the apoE4 protein is known to be functionally deficient compared to the apoE3 protein, we used humanized APOE mice as a clinically relevant model to study the role of apoE on BBB injury and repair after TBI.

**RESULTS:**

**These data confirm apoE as an important modulator of spontaneous BBB stabilization following TBI, and highlights the APOE4 allele as a risk factor for poor outcome after TBI.**

# CELLULAR SCALE OF CONCUSSION: ACUTE PHASE SUPPORT

**Diet:**
**High in medium chain and EFA's**
**Avoid NMDA Excitotoxins**




**Anti-oxidants/ Perfusion and Mitochondrial Support:**
**N-Acetyl Carnitine**
**Alpha Lipoic Acid**
**Glutathione**
**Resveratrol**

**Magnesium L-Threonate**
**NMDA "Plug"**

**FASTING:**
**AUTOPHAGY**
**MITOPHAGY**

**LOW LEVEL LASER THERAPY**



# CELLULAR SCALE OF CONCUSSION: ACUTE PHASE SUPPORT

**Diet:**
**High in medium chain and EFA's**
**Avoid NMDA Excitotoxins**



**2 Daily**

**After Day 3 Fast:**
**2 BID 2 weeks**
**Then daily until bottle is gone**

**2 Daily acute then 1 daily**


**Anti-oxidants/ Perfusion and Mitochondrial Support:**
**N-Acetyl Carnitine**
**Alpha Lipoic Acid**
**Glutathione**
**Resveratrol**

**Magnesium L-Threonate**
**NMDA "Plug"**


**1 Scoop (Dose?)**

**FASTING:**
**AUTOPHAGY**
**MITOPHAGY**
**24 hour water fast**
**Days 3, 7, 14, 21**

**LOW LEVEL LASER THERAPY**
**ERCHONIA Frequency 1-1-1-1 or 10. 3x/Week**
**TCTFX 10 min/Hand/Head 12**

# TBI NUTRITIONAL SUPPORT

***START NUTRITIONAL SUPPORT 24-48 HOURS POST TBI***

**PRIMARY OBJECTIVES: STABILIZE NEURONAL MEMBRANES AND MINIMIZE APOPTOSIS**
**MITIGATE DAMAGING EFFECTS OF ISCHEMIA AND VASCULAR INJURY**
**MAINTAIN CELL METABOLISM AND MINIMIZE ROS DAMAGE**
**MAINTAIN CA2+ HOMEOSTASIS**
**POST INITIAL RESPONSE, REDUCE LONG TERM INFLAMMATION/GLIAL PRIMING**

**ALPHA LIPOIC ACID -** LIPID PEROXIDASE INHIBITORS ARE MORE EFFECTIVE IN NEURONAL PROTECTION THAN DIRECT FREE RADICAL SCAVENGERS. LIPOIC ACID ALSO HELPS MITIGATE NEURONAL DEATH POST TBI AND PROMOTES GLUTATHIONE AND SUPEROXIDE DISMUTASE ANTI-OXIDANT ACTIVITY. WATCH WITH LIVER DISEASE/ ETOH, BLOOD SUGAR REGULATION, THYROID DISORDERS, THIAMINE DEFICIENCY

**ZINC, MAGNESIUM, VITAMIN D AND E -** ALL HAVE BEEN SHOWN TO IMPROVE OUTCOMES PRE- AND/OR POST-TBI. ZINC IS A CO-FACTOR FOR SUPEROXIDE DISMUTASE FREE RADICAL PROTECTION, AND HAS BEEN SHOWN TO HELP WITH APOPTOSIS, AUTOPHAGY AND INFLAMMATION REDUCTION POST TBI. IT HAS ALSO SHOWN EVIDENCE IN HELPING POST TBI DEPRESSION AND ANXIETY.

Nutr Neurosci. 2018 Feb; 21(2): 79–91.
Published online 2016 Oct 5. doi: 10.1080/1028415X.2016.1236174

PMCID: PMC5491366
NIHMSID: NIHMSID818643
PMID: 27705610



# TBI NUTRITIONAL SUPPORT

**DHA OMEGA 3 FATTY ACID -** STABILIZES NEURONAL MEMBRANES, MITIGATES EXCITOTOXICITY AND NEURONAL DEATH (APOPTOSIS). IT HELPS MINIMIZE AMYLOID BURDEN AND HIGHER PRE-INJURY DHA LEVELS IMPROVE TBI OUTCOMES. DHA PROMOTES GLUTATHIONE REDUCTION OF ROS FREE RADICAL DAMAGE. HIGH SPM'S PROMOTES LONG TERM INFLAMMATION REDUCTION AND GLIAL RESOLUTION.

**RESVERATROL -** REDUCES ROS DAMAGE, LIPID PEROXIDATION, INFLAMMATION AND EXCITOXICITY. IT ALSO HELPS PROTECT THE BBB AND DECREASES THE LESION SIZE AND SPREADING EFFECT.

**SULFORAPHANES -** PROTECT AND REPAIR THE BBB, REDUCE BRAIN INFLAMMATION AND EDEMA, IMPROVES COGNITION POST TBI. NRF2!

**CURCUMINS -** MODULATE NF-KB REDUCING INFLAMMATION. PROMOTES GLIAL RESOLUTION AND BDNF OUTPUT, INCREASES VAGAL TONE, SHOWN TO IMPROVE MOTOR AND LEARNING POST TBI.

Nutr Neurosci. 2018 Feb; 21(2): 79–91.
Published online 2016 Oct 5. doi: 10.1080/1028415X.2016.1236174

PMCID: PMC5491366
NIHMSID: NIHMSID818643
PMID: 27705610

# TBI

**VITAMIN B2 (RIBOFLAVIN) -** MINIMIZE GLUTAMATE EXCESS IN BLOOD, REDUCES INFARCT SIZE AND EDEMA AND IMPROVES COGNITION AND SENSORIMOTOR.

**MELATONIN -** PROMOTES IMPROVED COGNITIVE AND BEHAVIORAL OUTCOMES AND MITIGATES PATHOLOGICAL EFFECTS OF TBI SUCH AS CEREBRAL EDEMA, BBB DAMAGE

**CANNABINOIDS -** MINIMIZES EXCITOXICITY AND PROMOTES CELLULAR HOMEOSTASIS. HELPS WITH MANY POST TBI COGNITIVE AND BEHAVIORAL FINDINGS SUCH AS DEPRESSION, ANXIETY, SLEEP, PTSD AND MUCH MORE. HELPS WITH THE "BRAIN-GUT AXIS" AND MAINTAINING HEALTHY COMMUNICATION WITH THE GUT MICROBIOME INCLUDING PAIN MECHANISMS ASSOCIATED WITH GABA AND OPIOID RECEPTOR SYSTEMS.

**Progesterone -** Progesterone treatment leads to reduced edema, neuroinflammation, neuronal excitotoxicity, and apoptosis after TBI in both animal studies and initial clinical trials.

***MONITOR THE ENTIRE HP AXIS POST TBI AND POST-CONCUSSION SYNDROME***

Biomedicines. 2020 Oct; 8(10): 389.
Published online 2020 Sep 29. doi: 10.3390/biomedicines8100389

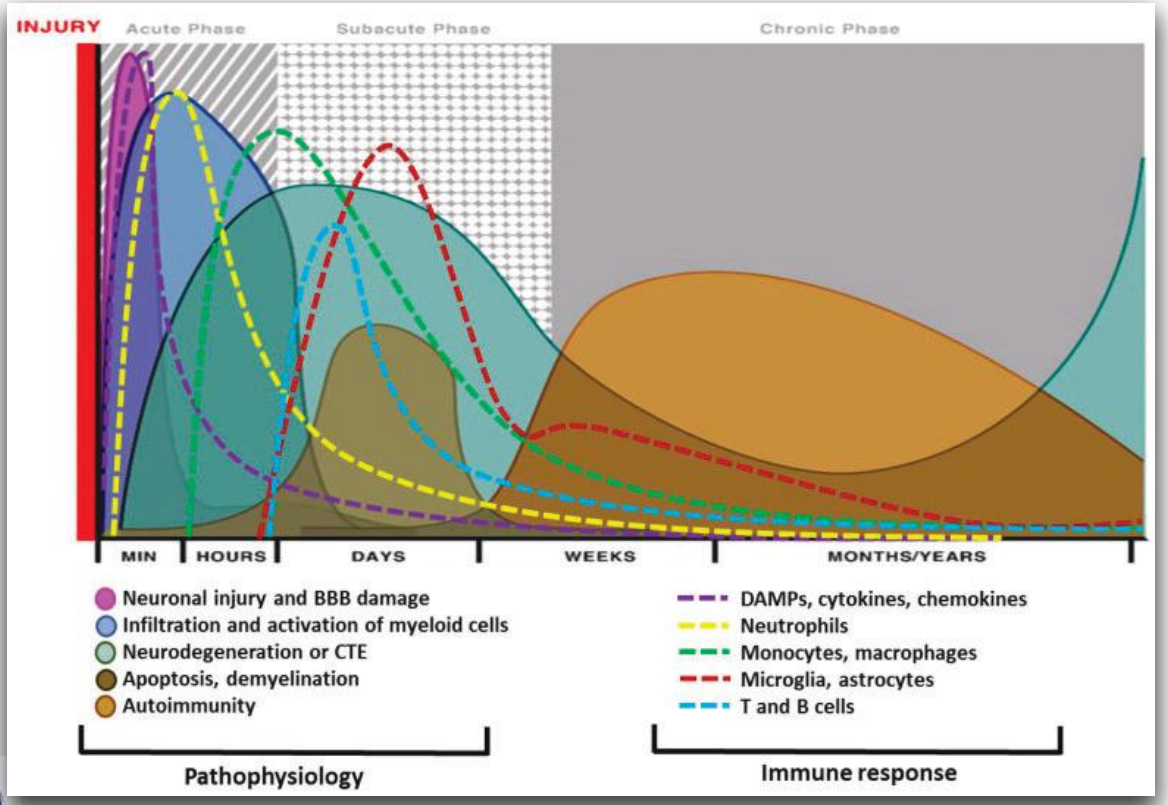
PMCID: PMC7601301
PMID: 33003373

Revisiting Traumatic Brain Injury: From Molecular Mechanisms to Therapeutic Interventions

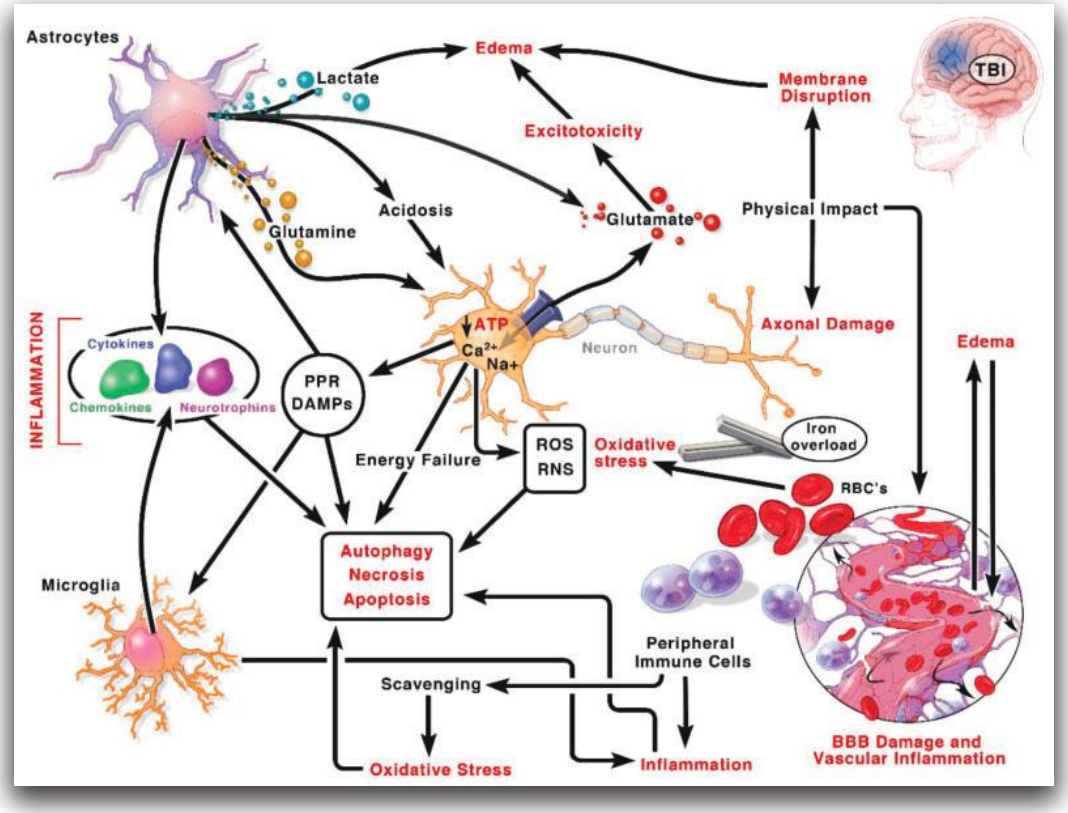
Abbas Jarrahi,1,† Molly Braun,1,2,



# TBI



# TBI






# MELATONIN FOR TBI

J Neurotrauma. 2018 Jun 14. doi: 10.1089/neu.2018.5752. [Epub ahead of print]

## Melatonin as a treatment after Traumatic Brain Injury: A systematic review and meta-analysis of the pre-clinical and clinical literature.

Barlow K1,2, Esser MMJ3, Veidt M4, Boyd R5.

**Author information Abstract**
Traumatic brain injury is common and yet effective treatments of the secondary brain injury are scarce. Melatonin is a potent, non-selective neuroprotective and anti-inflammatory agent that is showing promising results in neonatal brain injury. The aim of this study was to systematically evaluate the pre-clinical and clinical literature for the effectiveness of Melatonin to improve outcome after TBI. Using the systematic review protocol for animal intervention studies (SYRCLE) and Cochrane methodology for clinical studies, a search of English articles was performed. Eligible studies were identified and data was extracted. Quality assessment was performed using the SYRCLE risk of bias tool. Meta-analyses were performed using standardized mean differences (SMD). Seventeen studies (15 pre-clinical, 2 clinical) met inclusion criteria. There was heterogeneity in the studies, and all had moderate-to-low risk of bias. Meta-analysis of pre-clinical data revealed an overall positive effect on neurobehavioural outcome with SMD of 1.51 (95% CIs: 1.06-1.96). Melatonin treatment had a favorable effect on the neurological status, by a SMD of 1.35 (95% CI: 0.83-1.88) and cognition by a SMD of 1.16 (95% CIs: 0.4-1.92). Melatonin decreased the size of the contusion by a SMD of 2.22 (95% CI: 0.84-3.59) and cerebral oedema by SMD of 1.91 (95% CI: 1.08-2.74). Only two clinical studies were identified. They were of low quality, used for symptom management, and were of uncertain significance. In conclusion, ***there is evidence that Melatonin treatment after TBI significantly improves both behavioural outcomes and pathological outcomes***, but significant research gaps exist esp in clinical populations. Key Words: Traumatic Brain Injury, Melatonin, Systematic Review.

# RELAXMAX


- **Supports Relaxed Mood***
- **Supports Inhibitory Neurotransmitter and Second Messenger**
- **Functions***
- **Supports Neurotransmitter Balance and Neuronal Stabilization***
- **May Support Blood Pressure Already Within Normal Range***
- **Addresses Brain Osmotic Regulation, Glial Cell Function, and**
- **Effective Neuronal Transmission***

**Magnesium**

**L-Theanine (N-ethyl-L-glutamine) L-Theanine, provided as Suntheanine®: Inhibits glutamate toxicity and reduces neuronal overstimulation.**

**Inositol: A precursor for the second-messenger phosphatidyl- inositol system, which affects mood status differently than precursors for neurotransmitters.[2]**

**GABA: Increases the production of alpha waves (related to a relaxed, yet mentally focused state) while decreasing beta waves (associated with hyperactivity, nervousness, and fleeting thoughts). Sufficient GABA results in the smooth, calming, regular rhythmic flow of electrical impulses in the brain needed for emotional well-being.[5] Supplementation in humans has shown support for the maintenance of healthy cortisol and secretory IgA levels while under stress.*[6]**

**Taurine: Considered neuroprotective, taurine modulates the ability of mitochondria to buffer intracellular calcium during glutamate depolarization and excitotoxicity and, thereby, may prevent cell death.[7] In addition to its antioxidant and cytokine-regulation. It is important in neurotransmission.**



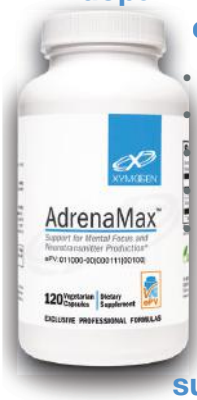
# FOCUS/ATTENTION/COGNITION/DRIVE MOTOR/WORKING MEMORY


- **Supports Healthy Brain Magnesium Levels***
- **Promotes Concentration, Mental Clarity, and Focus***
- **Supports Cognitive Health***
- **Promotes Mental and Physical Energy and Motivation to Exercise***

**MagteinTM (Patented Magnesium L-Threonate) and Activated B12**

**TeaCrine® (Theacrine): Similar alkaloid derivative in Tea and coffee but without habituating properties.**

**Theacrine is a dopamine D1 and D2 receptor agonist, and its actions help increase dopamine signaling associated with attention, movement, task initiation and completion, mood, learning, and the brain's "reward center."***




- **Maintain Healthy Levels of Dopamine, Norepinephrine and Epinephrine***
- **Supports Memory Under Stressful Conditions***
- **Supports Mental Focus and Alertness***
- **Supports Individuals with Polymorphism in Dopamine Receptors***
- **Supports Healthy Mood***

**Tyrosine: non-essential amino acid that can be synthesized in the body from phenylalanine, is converted into dopamine, epinephrine, and norepinephrine. Improves working memory under stress. Tyrosine also supports adrenal and pituitary function, and may increase thyroid hormone**

**Although increased dopamine may be beneficial in some circumstances, excessive synthesis of this neurotransmitter generates hydoxy radicals that stress glutathione levels. N-acetyl cysteine (NAC), a derivative of the amino acid, L- cysteine, is the precursor to glutathione.**