The Role of Nutrition and the Reduction of Infection in the Promotion of Cognitive Health

Cindy M. Howard, DC, DABCI, DACBN, FIAMA, FICC

UVCA
April 26, 2025

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Current:

Board Certified Chiropractic Internist and Nutritionist

Fellowship International Academy of Medical Acupuncture

Fellowship International College of Chiropractors

Owner Innovative Health & Wellness Center

VP of operations, Inguardia Health

Medical Advisory Board, Fullscript

DC Consulting, Owner

Illinois Delegate American Chiropractic Association

ACA Committee member: Nominating , Professional Development, Guidelines and Membership

Past President, College of Pharmacology and Toxicology

Executive Board Member Chiropractic Defense Council

Medical Advisory Board Functional Medicine University

Post graduate instructor for DABCI diplomate

Mom of three amazing kids

Past History:

Original Director of Functional Medicine Aligned Modern Health, Chicago, Illinois

Director of Functional Nutrition for Neurosport Elite, Davie, Florida

Past President of the Council on Diagnosis and Internal Disorders

Past President of the ACA College of Pharmacology and Toxicology

Medical Advisory Board Integrative Therapeutics

Executive Board Member Doc:s

Board member Frankfort Falcons Youth Football Association Team Chiropractic Physician for Dreamz Elite Cheer

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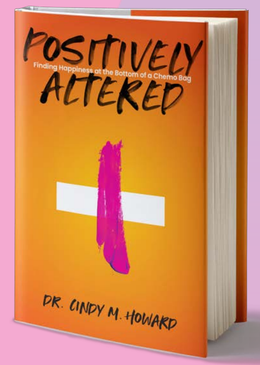

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Objectives:

Understand the role of the gut, mouth, brain and its relationship to infection and injury to the brain

Understand tools for testing and treating common gut and oral infections

Learn nutritional recommendation for infections, inflammation and associated Neurotransmitter dysfunction

Have a new clinical pearl for Monday morning

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Daniel

DR. Cindy!

History/Physical

19YOM
WT: 237 HT 6’1” P: 85, R 18, T 99.0, BP seated left 141/70, Right 142/66
EENT WNL, Chest, Lungs, Abdomen WNL
Concussed on the field
Evaluated by athletic trainer
Returned to play after headaches vanished and Baseline levels of Scat 5 were obtained
Presents with elevated blood pressure and sleep disruption
Difficulty focusing in school
Stress Level ranges from 4-7/10
Takes over an hour to fall asleep
Waking at 3 am
T, TH up at 5 am for training, other days at 8
Alcohol consumption
Headaches
Chest pain
Heart races
Anxiety
EKG WNL
Light headed when skipping meals
Afternoon fatigue
Difficulty gaining weight

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THE HOT DOG

VS

THE PIZZA PUFF

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Doritos
Spicy Sweet CHILI
Funyuns
Lay's
Classic
Classique
Cheetos
Flamin' Hot
Fritos
Pinch of Salt
CORN CHIPS
FRUIT BY THE FOOT
ultimate
boarding
giveaway!
STRAWBERRY
Pocky
OREO
Cakesters
Smartfood
WHITE CHEDDAR
Chips Ahoy!
NEW!

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Concussion

A Clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma

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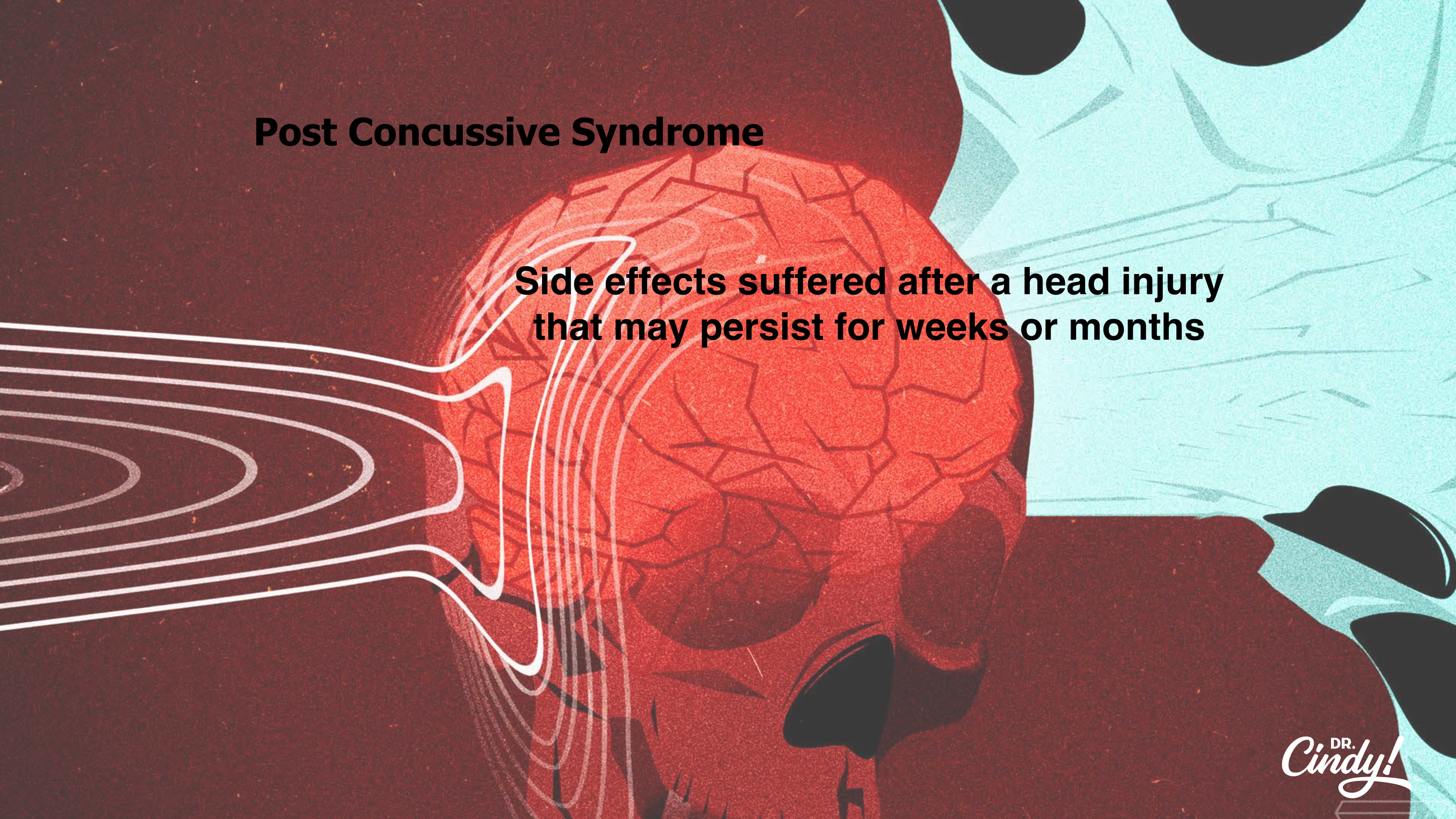
Post Concussive Syndrome

Side effects suffered after a head injury
that may persist for weeks or months



Incidence of TBI

- Estimated: One million TBIs in US every year
- Every 15 seconds
- 5.3 millions people living in US with permanent disabilities from TBI

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Why Post Concussion Nutrition?

Rest is not enough

Reduce inflammation

Decrease chemical changes

Affect neuronal function/provide neuroprotection

Blood vessel and tissue repair

Speed up recovery

Prevent further damage

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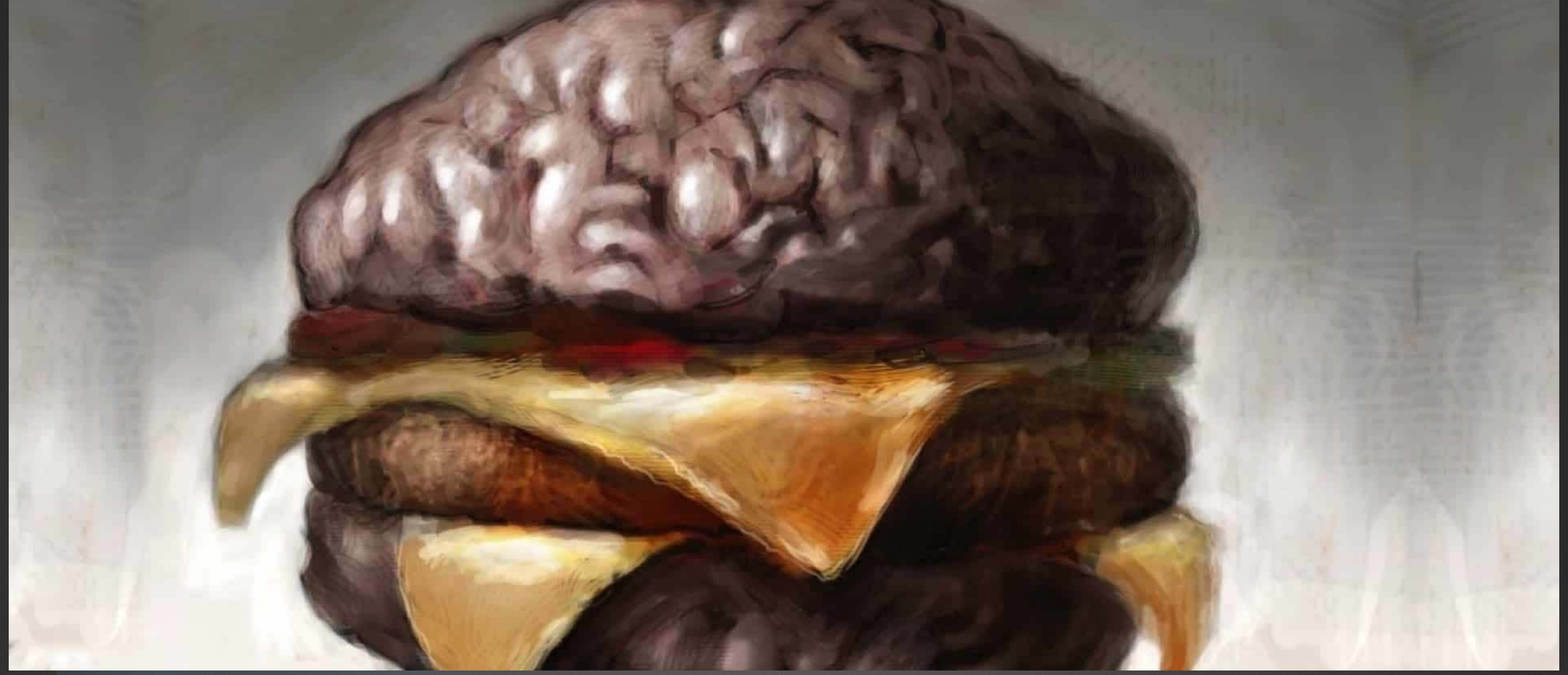

Considerations Post Concussion (and Pre Too!!!!)

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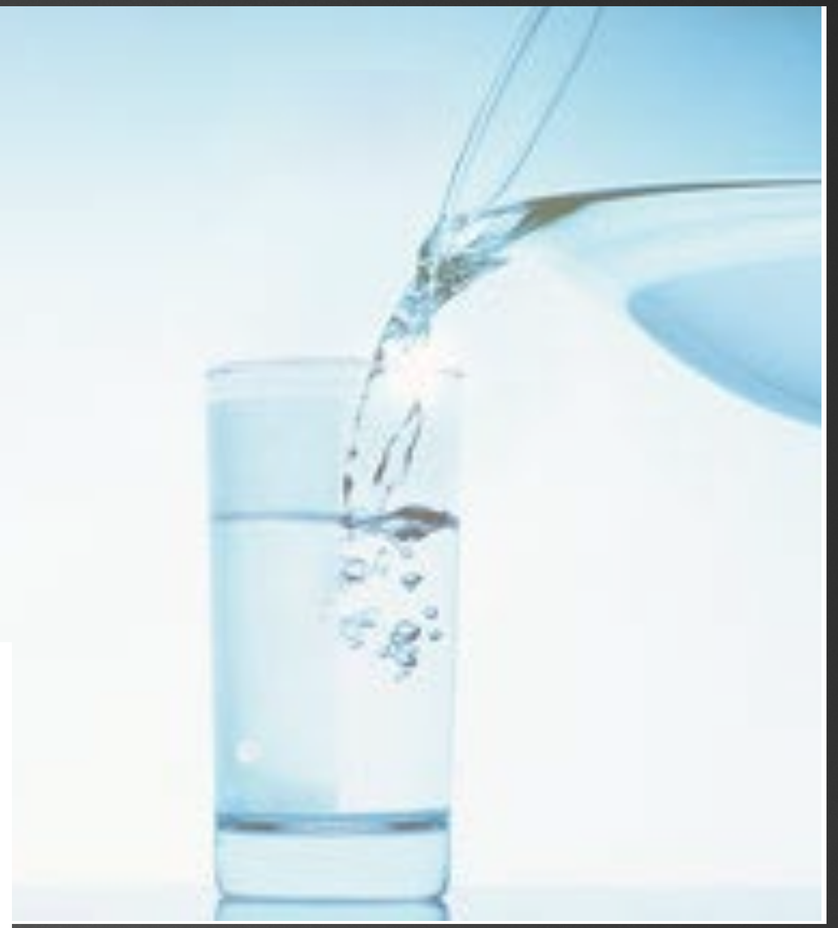
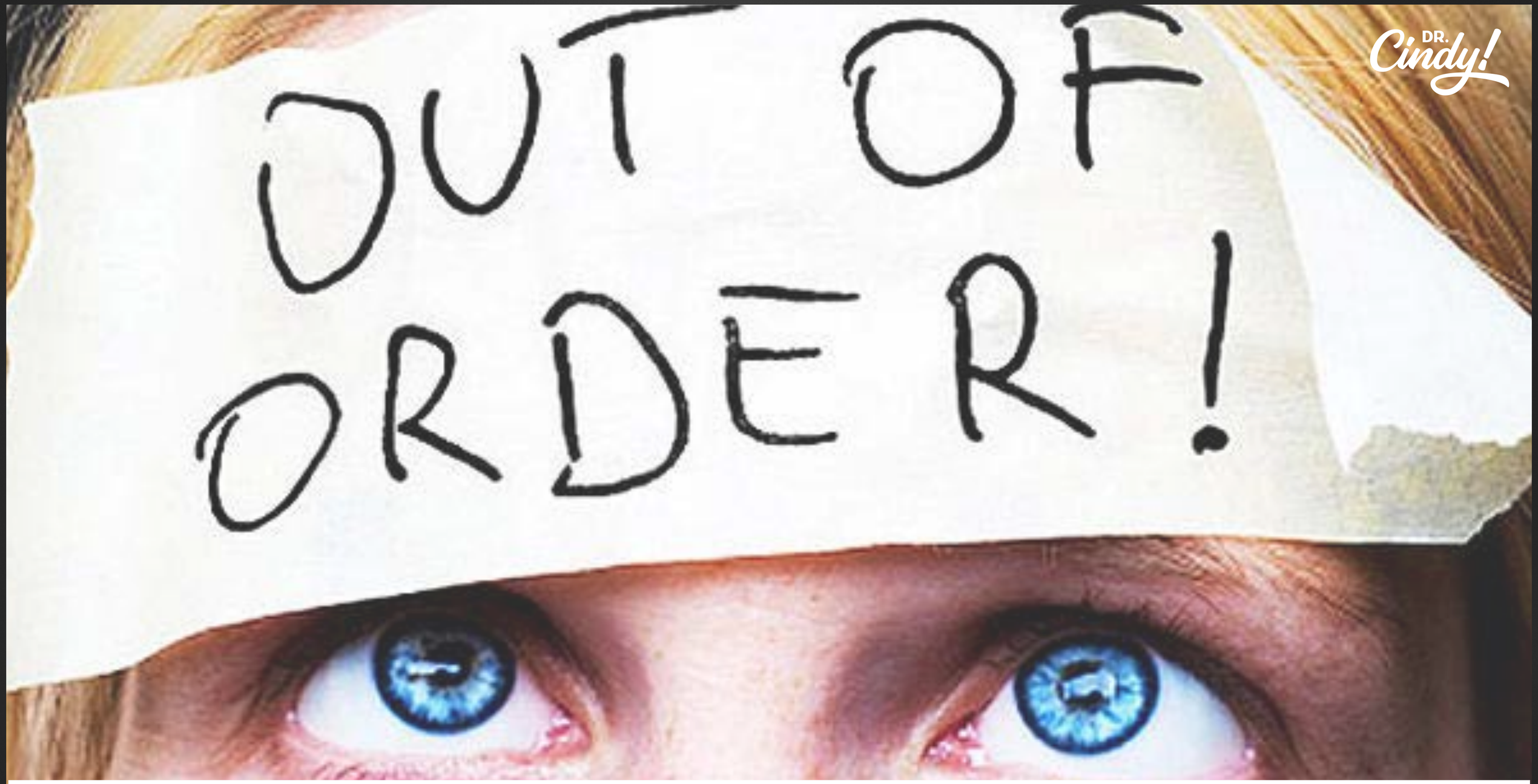
Hydration and Recovery



Moderate dehydration significantly influenced the self-report of symptoms commonly associated with concussion



Deterioration of visual memory and increases in the self-report of fatigue.

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Study challenges idea of mandatory water intake

+SHARE

10 October 2016

A multi-institute study led by Monash University has revealed for the first time the mechanism that regulates fluid intake in the human body and stops us from over-drinking, which can cause potentially fatal water intoxication. The study challenges the popular eight glasses of water a day for health.

The study showed that a 'swallowing inhibition' kicks in after excess liquid is consumed, helping maintain tightly calibrated volumes of water in the body.

Associate Professor Michael Farrell from the Monash Biomedicine Discovery Institute oversaw the work by University of Melbourne PhD student Pascal Saker as part of a collaboration with several Melbourne institutes.

"If we just do what our body demands us to we'll probably get it right – just drink according to thirst rather than an elaborate schedule," Associate Professor Farrell said.

Three fold increase in effort after over drinking.

Building on a previous study, the researchers asked participants to rate the amount of effort required to swallow water under two conditions; following exercise when they were thirsty and later after they were persuaded to drink an excess amount of water.

Drinking too much can put the body in danger of water intoxication or hyponatremia.

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Calories

Raise BDNF

Reduce Saturated

The interplay between oxidative stress and brain-derived neurotrophic factor modulates the outcome of a saturated fat diet on synaptic plasticity and cognition

Aiguo Wu, Zhe Ying, Fernando Gomez-Pinilla

First published: 8 April 2004 Full publication history

DOI: 10.1111/j.1460-9568.2004.03246.x View/save citation

Cited by (CrossRef): 182 articles Check for updates | Citation tools

Altmetric score 45

: Dr Fernando Gomez-Pinilla, 1Department of Physiological Science, as above.
E-mail: fgomezpi@ucla.edu

View Issue TOC
Volume 19, Issue 7
April 2004
Pages 1699–1707

Abstract

cognition caused by consumption of the HF diet. Male adult rats were maintained on a HF diet for 2 months with or without 500 IU/kg of vitamin E. Supplementation of the HF diet with vitamin E dramatically reduced oxidative damage, normalized levels of BDNF, synapsin I and cyclic AMP-response element-binding protein (CREB), caused by the consumption of the HF diet. In addition, vitamin E supplementation preserved the process of activation of synapsin I and CREB, and reversed the HF-impaired cognitive function. It is known that BDNF facilitates the synapse by modulating synapsin I and CREB, which have been implicated in synaptic plasticity associated to learning and memory. These results show that oxidative stress can interact with the BDNF system to modulate synaptic plasticity and cognitive function. Therefore, studies appear to reveal a mechanism by which events classically related to the maintenance of energy balance of the cell, such as oxidative stress, can interact with molecular events that modulate neuronal and behavioural plasticity.

Vitamin E used with HF diet reduced oxidative damage, normalized levels of BDNF

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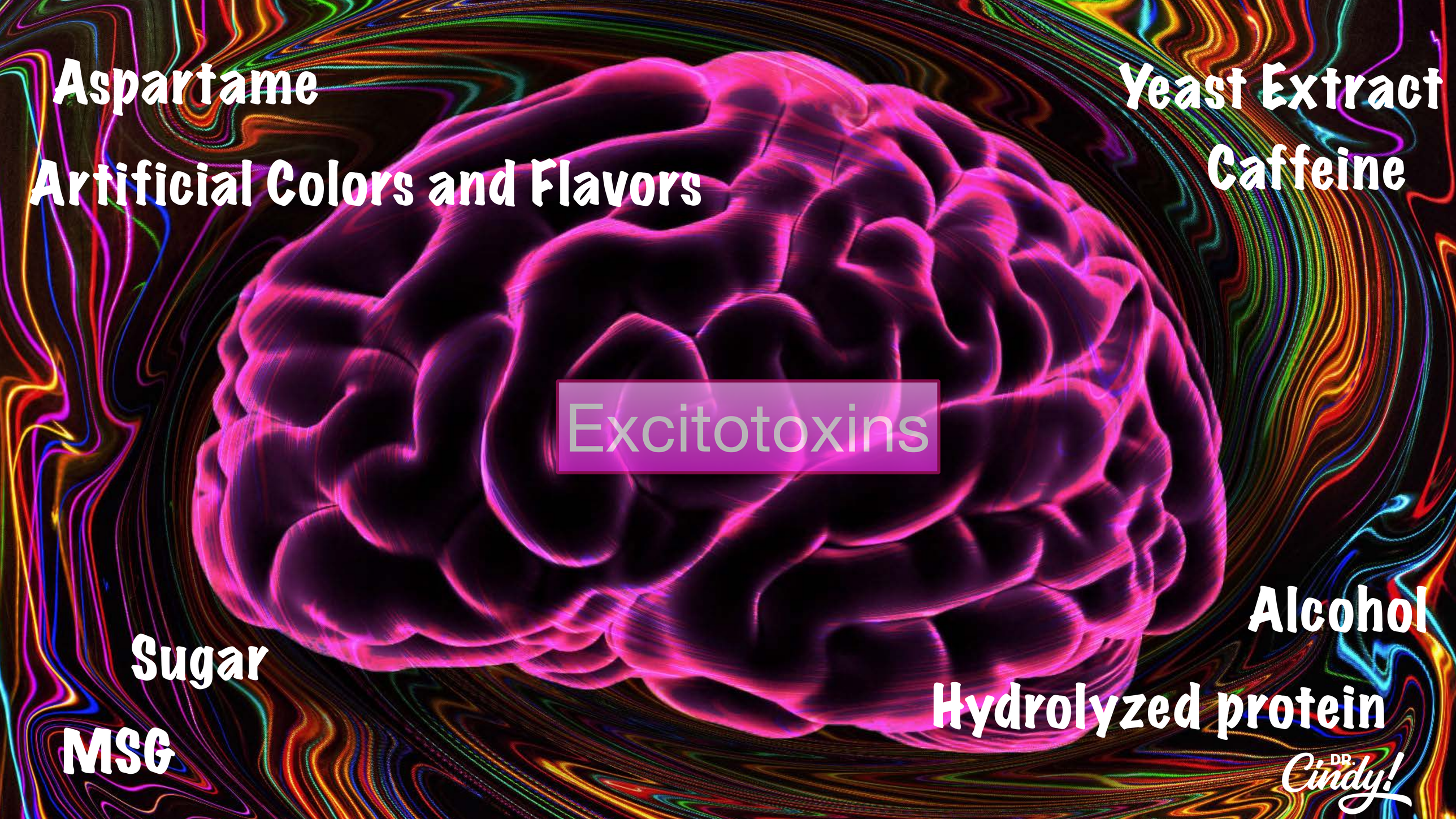
Aspartame
Artificial Colors and Flavors
Yeast Extract
Caffeine
Excitotoxins
Alcohol
Sugar
Hydrolyzed protein
MSG
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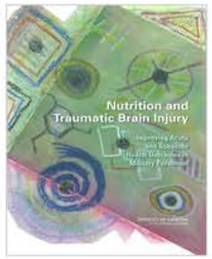

Nutrition and Traumatic Brain Injury: Improving Acute and Subacute Health Outcomes in Military Personnel.

< Prev Next >

Show details

Institute of Medicine (US) Committee on Nutrition, Trauma, and the Brain; Erdman J, Oria M, Pillsbury L, editors.
Washington (DC): National Academies Press (US); 2011.

Contents Hardcopy Version at National Academies Press

Search this book

6 Energy and Protein Needs During Early Feeding Following Traumatic Brain Injury

Several Cochrane reviews have established a reasonable basis for early and adequate feeding following traumatic brain injury (TBI), although the number and size of the trials supporting this recommendation are limited (Perel et al., 2006; Yanagawa et al., 2002). Improvements in mortality and neurological outcome have been suggested, with a relative risk for mortality of 0.67 [illegible] 5 (0.50–1.11) for death and disability (Perel et al., [illegible] al illness, including studies in TBI requiring admission to an intensive care unit (ICU), and using an intent-to-treat analysis, total parenteral nutrition (TPN) was found superior to enteral nutrition in reducing mortality, although it significantly increased the risk of infection (Doig et al., 2008). However, this improvement in mortality was related to the early and adequate feeding, because of patients who were fed adequately plus early by either enteral nutrition or TPN both did better than those receiving late enteral feeding (Doig et al., 2008). Although there are other meta-analyses that did not demonstrate any difference in mortality between parenteral and enteral feeding in the critically ill (Gramlich et al., 2004; Koretz et al., 2007; Mazaki and Ebisawa, 2008; Peter et al., 2005), only Doig et al. (2008) evaluated

Nutrition within 24 hours of injury for best results

1-1.5 g/kg of protein for the following 2 weeks

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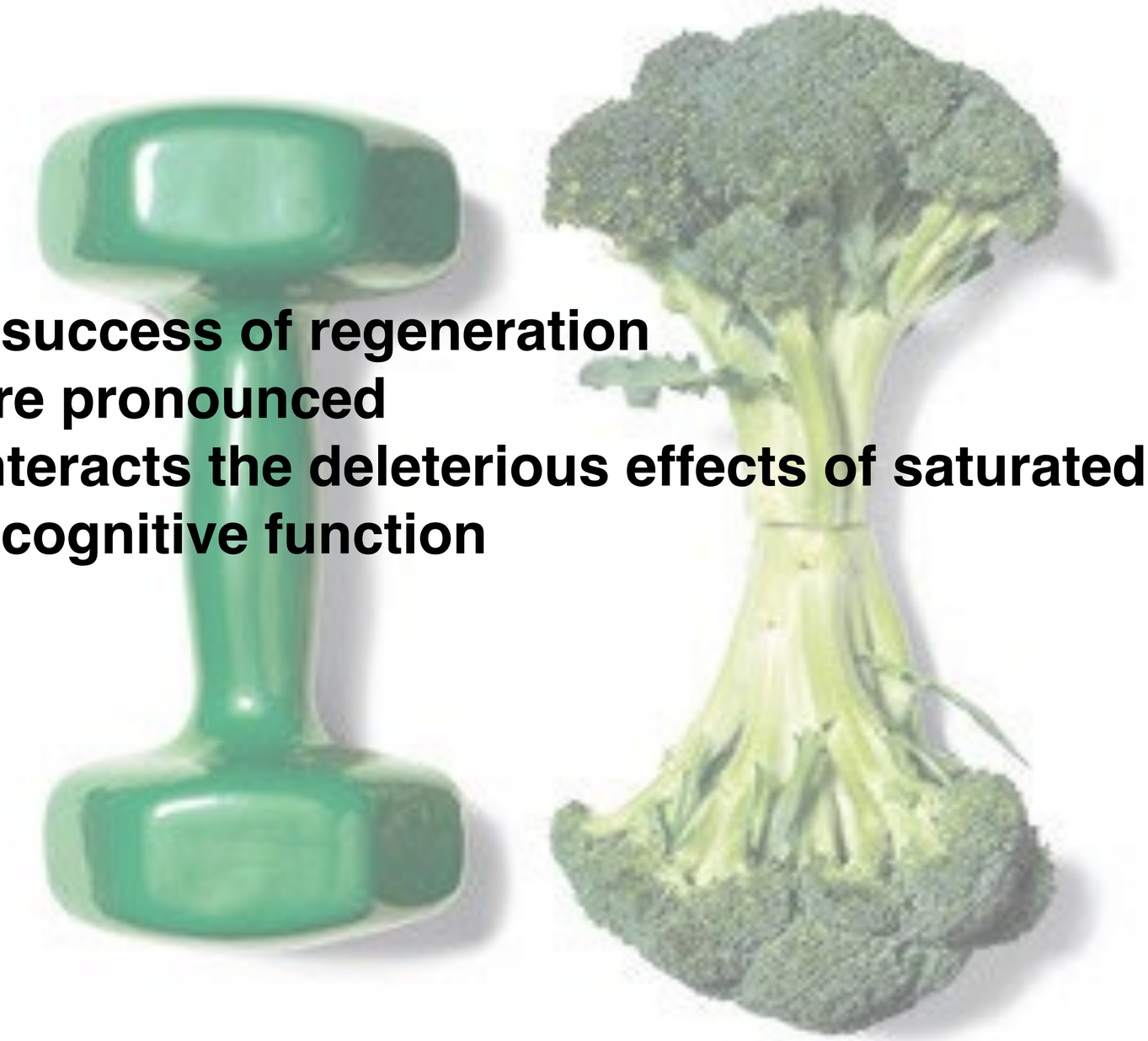
Walking
DR.
Cindy!

Diet and Exercise: Combined effect!



Increases the success of regeneration
Healing is more pronounced
Exercise counteracts the deleterious effects of saturated fat on synaptic plasticity and cognitive function

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Brain Res Rev. 2009 Mar;59(2):293-315. doi: 10.1016/j.brainresrev.2008.09.002. Epub 2008 Sep 25.

The neuroprotective properties of calorie restriction, the ketogenic diet, and ketone bodies.

Maalouf M[1], Rho JM, Mattson MP.

Author information

Abstract

Both calorie restriction and the ketogenic diet possess broad therapeutic potential in various clinical settings and in various animal models of neurological disease. Following calorie restriction or consumption of a ketogenic diet, there is notable improvement in mitochondrial function, a decrease in the expression of apoptotic and inflammatory mediators and an increase in the activity of neurotrophic factors. However, despite these intriguing observations, it is not yet clear which of these mechanisms account for the observed neuroprotective effects. Furthermore, limited compliance and concern for adverse effects hamper efforts at broader clinical application. Recent research aimed at identifying compounds that can reproduce, at least partially, the neuroprotective effects of the diets with less demanding changes to food intake suggests that ketone bodies might represent an appropriate candidate. Ketone bodies protect neurons against multiple types of neuronal injury and are associated with mitochondrial effects similar to those described during calorie restriction or ketogenic diet treatment. The present review summarizes the neuroprotective effects of calorie restriction, of the ketogenic diet and of ketone bodies, and compares their putative mechanisms of action.

Ketones can prevent neuron cell death

Helps ATP depletion, reactive oxygen species production and inflammation

More effective in children since younger brains are better at transporting and utilizing ketone bodies

Favors GABA production which can help with an increase in Glutamate

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J Lipid Res. 2014 Dec;55(12):2450-7. doi: 10.1194/jlr.R046706. Epub 2014 Apr 10.

The collective therapeutic potential of cerebral ketone metabolism in traumatic brain injury.

Prins ML[1], Matsumoto JH[2].

Author information

Abstract

The postinjury period of glucose metabolic depression is accompanied by adenosine triphosphate decreases, increased flux of glucose through the pentose phosphate pathway, free radical production, activation of poly-ADP ribose polymerase via DNA damage, and inhibition of glyceraldehyde dehydrogenase (a key glycolytic enzyme) via depletion of the cytosolic NAD pool. Under these post-brain injury conditions of impaired glycolyt More Efficient energy than Glucose?! one bodies are the only known natural alternative substrate ted that other fuels (pyruvate, lactate, and acetyl-L-carnitine) can be metabolized by the brain, ketones are the only endogenous fuel that can contribute significantly to cerebral metabolism. Preclinical studies employing both pre- and postinjury implementation of the ketogenic diet have demonstrated improved structural and functional outcome in traumatic brain injury (TBI) models, mild TBI/concussion models, and spinal cord injury. Further clinical studies are required to determine the optimal method to induce cerebral ketone metabolism in the postinjury brain, and to validate the neuroprotective benefits of ketogenic therapy in humans.

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Intestinal Permeability

Connection of brain and enteric system

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Bacterial Cytotoxins
Cytoskeletal Proteins
SIBO

Traumatic brain injury causes intestinal damage, study shows

Two-way brain-gut interactions may worsen outcome after TBI

Date: December 6, 2017

Source: University of Maryland School of Medicine

Trauma can cause disruption of cortico-pontine integrations leading to intestinal permeability that leads to inflammation and malabsorption leading to GI dysfunction

University of Maryland School of Medicine (UMSOM) researchers have found a two-way link between traumatic brain injury (TBI) and changes in the gastrointestinal tract. Two-way brain-gut interactions may contribute to worsening outcomes in these patients, and may also worsen brain damage.

Two way link between TBI and Intestinal changes
Leads to increased infection and worsen brain damage.

This is the first study to find that TBI in mice can trigger delayed, long-term changes in the colon and that subsequent bacterial infections in the gastrointestinal system can increase posttraumatic brain inflammation and associated tissue loss. The findings were published recently in the journal Brain, Behavior, and Immunity.

"These results indicate strong two-way interactions between the brain and the gut that may help explain the increased incidence of systemic infections after brain trauma and allow new treatment [illegible]earcher, Alan Faden, MD, the David [illegible]n the Departments of Anesthesiology, Anatomy & Neurobiology, Psychiatry, Neurology, and Neurosurgery at UMSOM, and director of the UMSOM Shock, Trauma

Intestinal permeability after trauma for a month after TBI

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Bacterial Overgrowth of the Small Intestine

Breath Test

12/16

63 Zillicoa Street
Asheville, NC 28801

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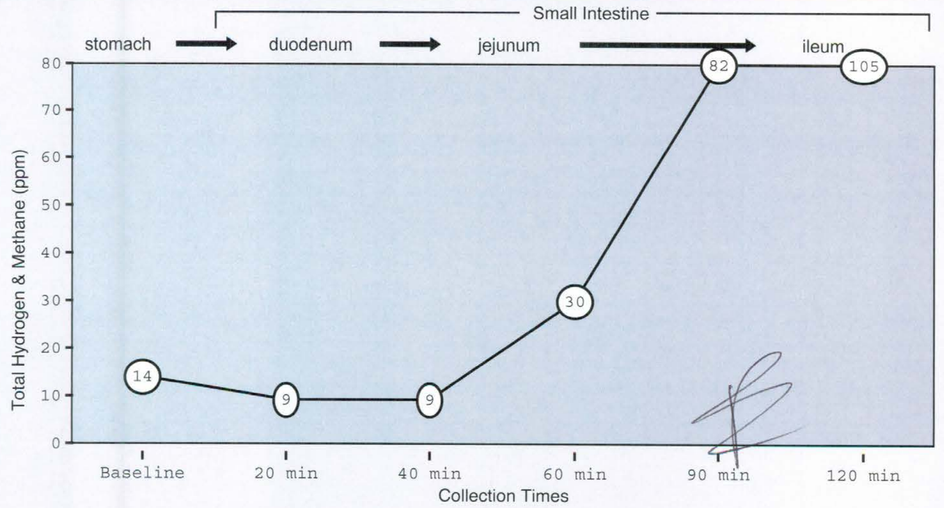
18309 Distinctive Dr
Orland Park, IL 60467

Total Hydrogen and Methane Breath Gases



Hydrogen & Methane (ppm)

Minutes	Base-line	20	40	60	90	120
Hydrogen (H_2)	11	6	8	29	79	102
Methane (CH_4)	3	3	1	1	3	3
Total	14	9	9	30	82	105

Increase over Baseline

Change in H_2 & CH_4

ppm

Normal <= 11 | Mild 12 - 32 | Severe >= 33

(Highest result value minus the baseline value)

Baseline Evaluation

Baseline Level

ppm

Normal <= 20 | Elevated >= 21

This test was developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

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Bacterial Overgrowth of the Small Intestine

Breath Test

2/17
2 months later

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Orland Park, IL 60467

Total Hydrogen and Methane Breath Gases

Hydrogen & Methane (ppm)

Minutes	Base-line	20	40	60	90	120
Hydrogen (H_2)	15	9	9	19	30	54
Methane (CH_4)	0	0	0	0	0	0
Total	15	9	9	19	30	54

Increase over Baseline

Change in H_2 & CH_4 ppm

(Highest result value minus the baseline value)

Baseline Evaluation

Baseline Level ppm

This test was developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

TBO63 RMS 1040 Rev 2

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Bacterial Overgrowth of the Small Intestine

Breath Test

5 months later

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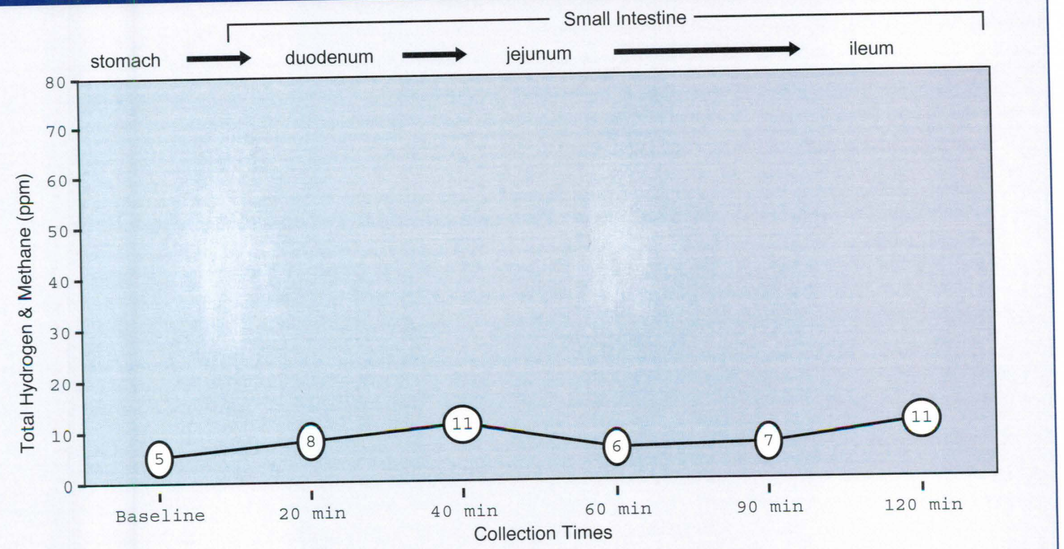
Total Hydrogen and Methane Breath Gases



Hydrogen & Methane (ppm)

Minutes	Base-line	20	40	60	90	120
Hydrogen (H_2)	5	6	11	6	7	9
Methane (CH_4)	0	2	0	0	0	2
Total	5	8	11	6	7	11

Increase over Baseline

Change in H_2 & CH_4

ppm

Normal <= 11 | Mild 12 - 32 | Severe >= 33

(Highest result value minus the baseline value)

Baseline Evaluation

Baseline Level

ppm

Normal <= 20 | Elevated >= 21

This test was developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

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PMCID: PMC4640931
NIHMSID: NIHMS731364

Glutamate and GABA imbalance following traumatic brain injury

Réjean M. Guerriero,[1,2,3] Christopher C. Giza,[4,5] and Alexander Rotenberg[1,2,3]

Author information ► Copyright and License information ►

Abstract

Traumatic brain injury (TBI) leads to multiple short and long term ch
ultimately conclude with an imbalance of cortical excitation and inhi
concentrations, receptor populations and specific cell survival are im
these changes occur gradually, which may explain the vulnerability c
alterations in neuroplasticity, and delays in the presentation of post-traumatic epilepsy. In this review we provide an overview of normal glutamate and GABA homeostasis, and describe acute, subacute and chronic changes that follow injury. We conclude by highlighti
in this paradigm.

Elevation of Glutamate after TBI

Causes surrounding neurons to take in too much calcium for neuron to function normally

Prevention of neuron from producing the energy it needs to function

Theanine 100-200mg
Taurine 1500-3000 mg
Valerian 600mg

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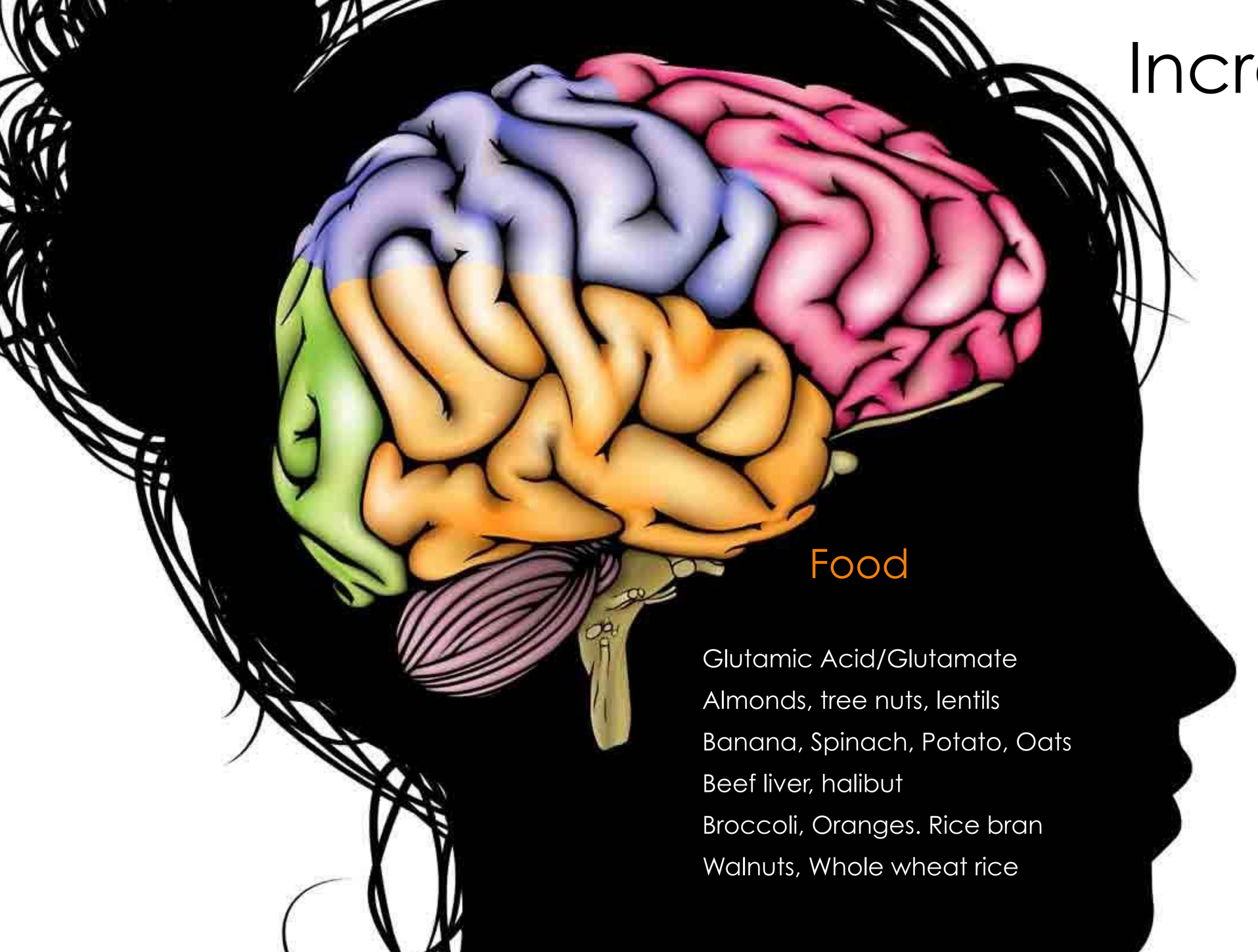
Increase GABA



Food

Glutamic Acid/Glutamate

Almonds, tree nuts, lentils

Banana, Spinach, Potato, Oats

Beef liver, halibut

Broccoli, Oranges. Rice bran

Walnuts, Whole wheat rice

Supplements

Magnolia bark (dopamine Agonist)

Inositol

Vitamin B6 (as P-5-P) 6.8 mg

Magnesium (succinate) 10 mg

Zinc (glycinate) 5 mg

Manganese (succinate) 10 mg

L-Taurine 150 mg

Valerian Root extract (standardized to 0.8 % valerenic acid) 100 mg

Passion Flower extract (standardized to 3.5 % flavonoids) 100 mg

L-Theanine 15 mg

Lithium (orotate) 10 mg

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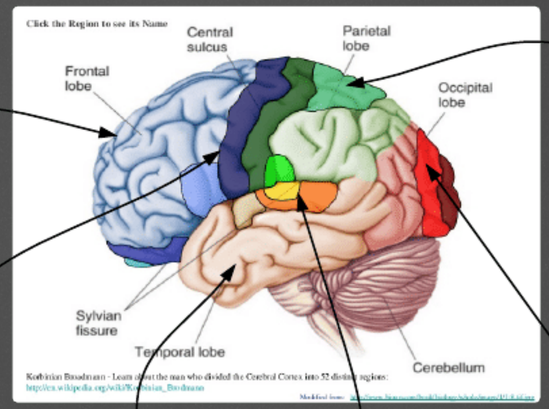
Nutrition Based on Site of Injury

Nutrition for Concussions Based on Site of Injury



Prefrontal Cortex: Dopamine, judgement, concentration, working memory, attention span, impulse control, organization, planning, language

Nutrition: Avoid gluten, increase protein intake, B6, magnesium, folate, zinc, copper, DHA, vitamin C, probiotics

Parietal Lobes: Sensory information, directions, symbols, reading, writing, mathematics

Nutrition: Currently Unknown

Anterior Cingulate Gyrus: Serotonin, Relaxation, flexibility, future plans and goal setting.

Nutrition: B6, B12, folate, vitamin D, magnesium, DHA, inositol

Temporal Lobes: Common area of head injury, GABA, anxiety, amnesia, hallucinations, aggression, dyslexia, depression, suicidal thoughts, poor social cues, sense of smell

Nutrition: Increased protein intake, vitamin C, zinc, DHA, vitamin D, B6, magnesium, choline, bifidobacterium

Limbic System: Norepinephrine, dopamine, serotonin, sleep, appetite cycles, bonding, social connectedness

Nutrition: B6, magnesium, folate, zinc, vitamin C, DHA

Occipital Lobes: Visual processing

Nutrition: Currently Unknown

Alex J. Swanson MS

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Published online 2015 Dec 23. doi: 10.1016/j.brainres.2015.12.030
PMCID: PMC4870112
NIHMSID: NIHMS750661

Vitamins and Nutrients as Primary Treatments in Experimental Brain Injury: Clinical Implications for Nutraceutical Therapies

Cole Vonder Haar,[1,*] Todd C. Peterson,[2,*] Kris M. Martens,[1] and Michael R. Hoane[3,#]

Mechanistic targets for nutritional therapies. Larger marks indicate increased effects.

		Excitotoxicity	Oxidative Stress	Energy Supplementation (mitochondria function, ATP, etc)	Cell Death	Edema	Plasticity & Neuromodulation	Inflammation
Vitamins	B_2		X					
	B_3		X	X				
	B_6	X		X				
	B_9				X			
	C		X					
	D		X					X
	E		X					
Herbs	Ginseng		X					X
	Ginkgo	X				X		
Flavonoids	Luteolin		X					X
	Quercetin		X					X
	Baicalein		X					
	Puerarin		X					
	Formononetin		X					
	7,8-DHF						X	
	Wogonin							X
	Flavopiridol							X
Other Nutrients	Magnesium	X	X					
	Zinc		X					
	Carnitine			X				
	Omega-3 Acids	X	X				X	X

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BRAIN INJURY

http://informahealthcare.com/bij
ISSN: 0269-9052 (print), 1362-301X (electronic)

Brain Inj, 2013; 27(12): 1454–1460
 DOI: 10.3109/02699052.2013.825009

informa
healthcare

ORIGINAL ARTICLE

The effect of Boswellia Serrata on neurorecovery following diffuse axonal injury

Payam Moein[1], Salman Abbasi Fard[2], Ali Asnaashari[3], Hajar Baratian[1], Majid Barekatain[4], Naser Tavakoli[5], & Houshang Moein[6]

[1]Behavioral Sciences Research Center, Noor Hospital, Isfahan University of Medical Sciences, Isfahan, Iran, [2]Departments of Neurosurgery, Saint Al-Zahra Hospital, Isfahan University of Medical Sciences, Isfahan, Iran, [3]Departments of Neurosurgery, Kashani Hospital, Isfahan University of Medical Sciences, Isfahan, Iran, [4]Department of Psychiatry, Noor Hospital, Isfahan University of Medical Sciences, Isfahan, Iran, [5]Departments of Pharmacology, Isfahan University of Medical Sciences, Isfahan, Iran, and [6]Department of Neurosurgery, Al-Zahra Hospital, Isfahan, Iran

38 patients with pure DAI were enrolled in a 12 week double bind, randomized, cross over study

Significant increase in cognitive function during the periods they were on Boswellia Serata

ability to self-care' during the second 6 weeks in group A on BS compared to an insignificant spontaneous recovery in group B during the same period on placebo. Moreover, both groups experienced a close-to-significant increase in the cognitive function-related items of the DRS during the periods they were on BS. The reported adverse events were all of mild quality and had similar frequency between the groups.

Conclusion: These results suggest that BS resin does not significantly affect general outcome, but may enhance the cognitive outcome of patients with DAI.

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N-Acetyl Cysteine

- July 2000 issue of the "British Journal of Pharmacology"
- N-acetyl cysteine **protects brain cells from excessive damage and death**, especially after ischemic injuries
- Association between N-acetyl cysteine and glutathione
- Glutathione levels drop in the brain after injury, free radicals wreak havoc and worsen brain injury

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N-Acetyl cysteine

- Early post-injury treatment with N-Acetyl Cysteine (NAC) **reversed the behavioral deficits associated with the TBI**
- Direct scavenging of radicals or stimulation of glutathione peroxidase activity suggesting that N-acetyl cysteine may be useful for treatment of oxygen free radical-mediated brain injury

Ellis EF1, Dodson LY, Police RJ. Restoration of cerebrovascular responsiveness to hyperventilation by the oxygen radical scavenger n-acetylcysteine following experimental traumatic brain injury. J Neurosurg. 1991 Nov;75(5):774-9.

Eakin K1, Baratz-Goldstein R2, Pick CG2, Zindel O2, Balaban CD3, Hoffer ME4, Lockwood M1, Miller J1, Hoffer BJ5 Efficacy of N-acetyl cysteine in traumatic brain injury. PLoS One. 2014 Apr 16;9(4):e90617. doi: 10.1371/journal.pone.0090617. eCollection 2014.

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Fish Oil

- Omega-3 fatty acids reduce oxidative stress
- Conserving and activating the brain's own protection mechanisms responsible for maintaining the integrity of your brain cells
- The brain tissue analysis of TBI models supplemented with omega-3 polyunsaturated fatty acids (PUFAs) showed significantly reduced lipid peroxidation, nucleic acid and protein oxidation, promoting neuronal and glial cell survival
- July publication of The Journal of Neurosurgery, Dr. Julian Bailes and Dr. Barry Sears: "Animals receiving the daily fish oil supplement for 30 days post concussion had a greater than 98 percent reduction in brain damage compared with the animals that did not receive the supplement."

Kumar PR1, Essa MM1, Al-Adawi S2, Dradekh G1, Memon MA3, Akbar M4, Manivasagam T5. Omega-3 Fatty acids could alleviate the risks of traumatic brain injury. J Tradit Complement Med. 2014 Apr;4(2):89-92. doi: 10.4103/2225-4110.130374.

DR. Cindy!

Exp Neurol. 2006 Feb;197(2):309-17. Epub 2005 Dec 20.

Dietary curcumin counteracts the outcome of traumatic brain injury on oxidative stress, synaptic plasticity, and cognition.

Wu A[1], Ying Z, Gomez-Pinilla F.

Author information

Abstract

The pervasive action of oxidative stress on neuronal function and plasticity after traumatic brain injury (TBI) is becoming increasingly recognized. Here, we evaluated the capacity of the powerful antioxidant curry spice curcumin ingested in the diet to counteract the oxidative damage encountered in the injured brain. In addition, we have examined the possibility that dietary curcumin may favor the injured brain by interacting with molecular mechanisms that maintain synaptic plasticity and cognition. The analysis was focused on the BDNF system based on its action on synaptic plasticity and cognition by modulating synapsin I and CREB. Rats were exposed to a regular diet or a diet high in saturated fat, with or without 500 ppm curcumin for 4 weeks (n = 8/group), before a mild fluid percussion injury (FPI) was performed. The high-fat diet has been shown to exacerbate the effects of TBI on synaptic plasticity and cognitive function. Supplementation of curcumin in the diet dramatically reduced oxidative damage and normalized levels of BDNF, synapsin I, and CREB that had been altered after TBI. Furthermore, curcumin supplementation counteracted the cognitive impairment caused by TBI. These results are in agreement with previous evidence, showing that oxidative stress can affect the injured brain by acting through the BDNF system to affect synaptic plasticity and cognition. The fact that oxidative stress is an intrinsic component of the neurological sequel of TBI and other insults indicates that dietary antioxidant therapy is a realistic approach to promote protective mechanisms in the injured brain.

Curcumin

- Systemic anti inflammatory
- Treats central nervous system injury, inflammation, subarachnoid hemorrhage and TBI
- Improves patient outcome by reducing acute activation of microglia/macrophages and neuronal apoptosis (Journal of Neuroinflammation, 2014)
- Neurorehabilitation and Neural Repair reported that CNB-001 dramatically reversed the behavioral deficits in both locomotion and memory that accompany the brain injury (2010)
- A study presented in the Experimental Neurology Journal 2006 which revealed that curcumin counteracted the outcome of traumatic brain on oxidative stress, synaptic simplicity and cognition

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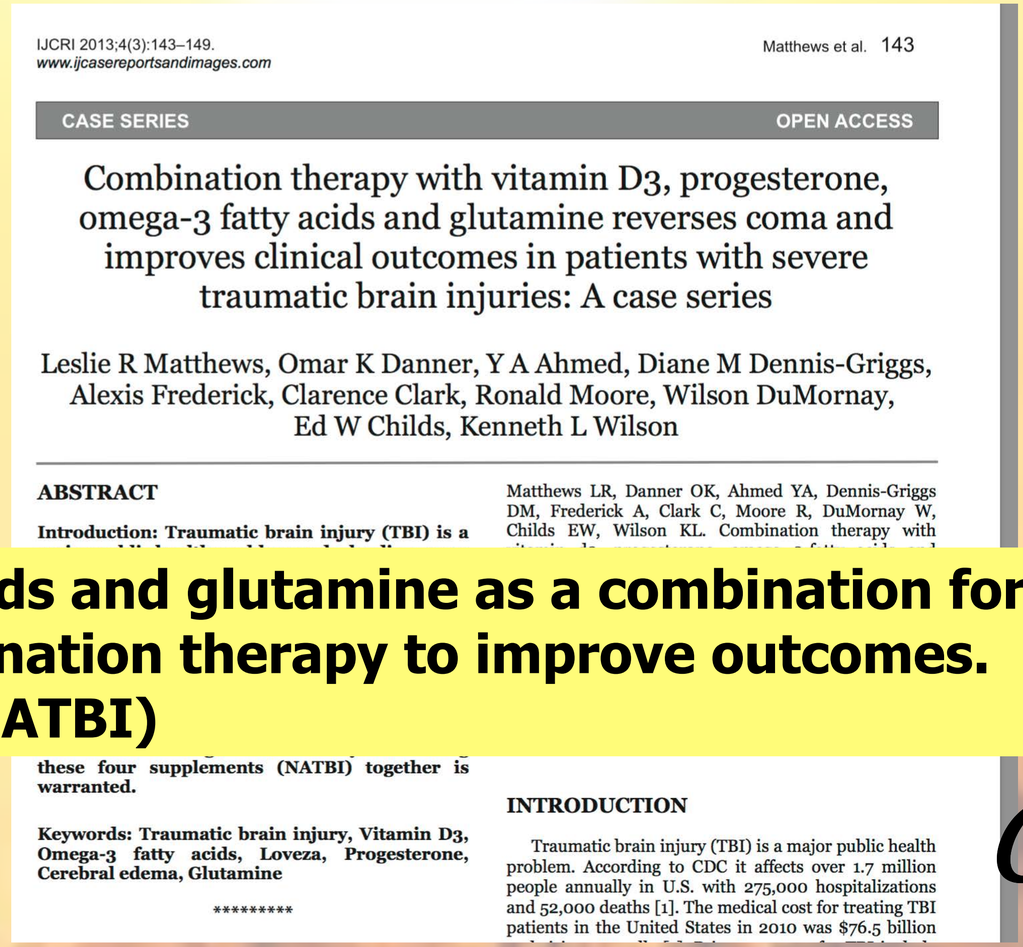
Vitamin D

6000 iu/day
Increases resilience to TBI
Deficiency may increase inflammatory damage and behavior impairment



IJCRI 2013;4(3):143–149.
www.ijcasereportsandimages.com

CASE SERIES OPEN ACCESS

Combination therapy with vitamin D3, progesterone, omega-3 fatty acids and glutamine reverses coma and improves clinical outcomes in patients with severe traumatic brain injuries: A case series

Leslie R Matthews, Omar K Danner, Y A Ahmed, Diane M Dennis-Griggs, Alexis Frederick, Clarence Clark, Ronald Moore, Wilson DuMornay, Ed W Childs, Kenneth L Wilson

ABSTRACT

Introduction: Traumatic brain injury (TBI) is a

these four supplements (NATBI) together is warranted.

Keywords: Traumatic brain injury, Vitamin D3, Omega-3 fatty acids, Loveza, Progesterone, Cerebral edema, Glutamine

****

Matthews LR, Danner OK, Ahmed YA, Dennis-Griggs DM, Frederick A, Clark C, Moore R, DuMornay W, Childs EW, Wilson KL. Combination therapy with

INTRODUCTION

Traumatic brain injury (TBI) is a major public health problem. According to CDC it affects over 1.7 million people annually in U.S. with 275,000 hospitalizations and 52,000 deaths [1]. The medical cost for treating TBI patients in the United States in 2010 was $76.5 billion

Vitamin D3, progesterone, omega-3 fatty acids and glutamine as a combination for moderate and severe TBI works as a combination therapy to improve outcomes. (NATBI)

DR. Cindy!

Vitamin D aided progesterone in reducing traumatic brain injury – RCT Dec 2012

Comparison of the administration of progesterone versus progesterone and vitamin D in improvement of outcomes in patients with traumatic brain injury: A randomized clinical trial with placebo group

Vitamin D and Progesterone

Adv Biomed Res 2012, 1:58

Bahram Aminmansour 1, Hossein Nikbakht 1, Abbas Ghorbani 2, Majid Rezvani 1, Paiman Rahmani 1, Mostaffa Torkashvand 1, Mohammadamin Nourian1, Mehran Moradi 1

1 Department of Neurosurgery, Isfahan University Of Medical Sciences, Isfahan, Iran

2 Department of Neurology, Al-zahra Hospital, Isfahan University Of Medical Sciences, Isfahan, Iran

Date of Submission 27-Apr-2012; Date of Acceptance 08-Jul-2012; Date of Web Publication 28-Aug-2012

Hossein Nikbakht, Neurosurgery Department, Al-Zahra Hospital, Isfahan University Of Medical Sciences, Isfahan; Iran

Recovery rate from severe brain trauma supplemented with progesterone and vitamin D was higher than with just progesterone or a placebo.

Background: Due to the heterogeneity of traumatic brain injur t of progesterone drug on severe brain injuries has been identifie ffects on animal samples in some studies. This study was conducted to

Materials and Methods: This study was performed on patients with severe brain trauma (Glasgow Coma Scale (GCS) ≤ 8) from April to September, 2011. The patients were divided to 3 groups (placebo, progesterone, progesterone-vitamin D), each with 20 people. Upon the patients' admission, their GCS and demographic information were recorded. After 3 months, they were reassessed, and their GCS and GOS (Glasgow outcome scale) were recorded. The collected data were analyzed using SPSS 18 software (SPSS Inc., Chicago IL, USA).

Results: Before intervention, GCS mean of the placebo, progesterone, and progesterone-vitamin D groups were 6.3 ± 0.88, 6.31 ± 0.87, and 6 ± 0.88, respectively. They increased to 9.16 ± 1.11, 10.25 ± 1.34, and 11.27 ± 2.27, respectively 3 months after intervention. There was a significant difference among GCS means of the 3 groups (P-value = 0.001). GOS was classified to 2 main categories of favorable and unfavorable recovery, of which, favorable recovery in **placebo, progesterone, and progesterone-vitamin D was 25%, 45%, and 60%,** respectively which showed a statistical significant difference among the groups (P-value = 0.03).

Conclusion: The results showed that recovery rate in patients with severe brain trauma in the group receiving progesterone and vitamin D together was significantly higher than that of progesterone group, which was in turn higher than that of placebo group.

DR. Cindy!

Combination treatment with progesterone and vitamin D hormone may be more effective than monotherapy for nervous system injury and disease.

Front Neuroendocrinol. 2009 Jul;30(2):158-72. Epub 2009 Apr 24; Cekic M, Sayeed I, Stein DG.
Department of Emergency Medicine, Emory University School of Medicine, **Atlanta**, Georgia 30322, USA.

2 decades of research

More than two decades of pre-clinical research and two recent clinical trials have shown that progesterone (PROG) and its metabolites exert beneficial effects after **traumatic brain injury** (TBI) through a number of metabolic and physiological pathways that can reduce damage in many different tissues and organ systems. Emerging data on 1,25-dihydroxyvitamin D(3) (VDH), itself a steroid hormone, have begun to provide evidence that, like PROG, it too is neuroprotective, although some of its actions may involve different pathways. Both agents have high safety profiles, act on many different injury and pathological mechanisms, and are clinically relevant, easy to administer, and inexpensive. Furthermore, vitamin D deficiency is prevalent in a large segment of the population, especially the elderly and institutionalized, and can significantly affect recovery after CNS injury. The combination of PROG and VDH in pre-clinical and clinical studies is a novel and compelling approach to TBI treatment.

PMID: 19394357 CLICK HERE for PDF

Traumatic brain injury and aging: is a combination of progesterone and vitamin D hormone a simple solution to a complex problem?

Neurotherapeutics. 2010 Jan;7(1):81-90; Cekic M, Stein DG; Emory University School of Medicine, Department of Emergency Medicine, **Atlanta**, Georgia 30322, USA.

Although progress is being made in the development of new clinical treatments for traumatic brain injury (TBI), little is known about whether such treatments are effective in older patients, in whom frailty, prior medical conditions, altered metabolism, an…ain injury. In this review we consider TBI to be a complex, highly variable, and systemic disorder that may require a new pharma…eat the many components of the injury cascade. We review some recent research on the role of vitamin D hormone and vitamin D def…esterone, the only treatment for TBI that has shown clinical effectiveness. Progesterone is now in phase III multicenter trial…isms and pathways through which the combination of hormones may work, singly and in synergy, to enhance survival and recovery afte…

PMID: 20129500 **PDF is attached at the bottom**

High safety profiles
Clinically relevant
Easy to administer
Inexpensive

DR. Cindy!

Enhancement of Learning and Memory by Elevating Brain Magnesium

Inna Slutsky,[3,6,7] Nashat Abumaria,[1,7] Long-Jun Wu,[5] Chao Huang,[1] Ling Zhang,[1] Bo Li,[1] Xiang Zhao,[1] Arvind Govindarajan,[2,3,4] Ming-Gao Zhao,[5] Min Zhuo,[5] Susumu Tonegawa,[2,3,4] and Guosong Liu[1,3,4,*]
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[3]Department of Brain and Cognitive Sciences
[4]Department of Biology
Massachusetts Institute of Technology, Cambridge, MA 02139, USA
[5]Department of Physiology, Faculty of Medicine, University of Toronto, Toronto, ON M5S 1A8, C
[6]Department of Physiology and Pharmacology, Faculty of Medicine, Tel Aviv University, Tel Aviv
[7]These authors contributed equally to this work
*Correspondence: liu.guosong@gmail.com
DOI 10.1016/j.neuron.2009.12.026

Magnesium

Enhances short term synaptic facilitation and long term potentiation and improves learning and memory functions

SUMMARY

Learning and memory are fundamental brain functions affected by dietary and environmental factors. Here, we show that increasing brain magnesium using a newly developed magnesium compound (magnesium-L-threonate, MgT) leads to the en-
h
a
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s
with memory improvement. Functionally, magnesium increased the number of functional presynaptic release sites, while it reduced their release probability. The resultant synaptic reconfiguration enabled selective enhancement of synaptic transmission for burst inputs. Coupled with concurrent upregulation of NR2B-containing NMDA receptors and its downstream signaling, synaptic plasticity induced by correlated inputs was enhanced. Our findings suggest that an increase in brain magnesium enhances both short-term synaptic facilitation and long-term potentiation and improves learning and memory functions.

INTRODUCTION

tions, as well as the number of available connections. Therefore, number of synapses should be critical for learning and memory too. Indeed, loss of synapses is correlated with age-dependent memory decline in rats (for review, see Burke and Barnes, 2006; Chen et al., 1995; Smith et al., 2000; Wilson et al., 2006), while hormones and neuropeptides, such as estrogen (Li et al., 2004), neurotophins (Vicario-Abejón et al., 2002), insulin/IGF (Lichten-
00), and ghrelin (Diano
nd improve memory.
tal factors, has a crucial
(for review, see Gómez-
dietary components that
of synapses might yield
and memory functions.
Magnesium (Mg^{2+}), the fourth most abundant ion in body and a cofactor for more than 300 enzymes, is essential for the proper functioning of many tissues and organs, including the cardiovascular, neuromuscular, and nervous systems. In brain, one major action of Mg^{2+} is modulating the voltage-dependent block of NMDA receptors (NMDAR), controlling their opening during coincidence detection that is critical for synaptic plasticity (Mayer et al., 1984; Nowak et al., 1984). Our previous study suggests that Mg^{2+} is a positive regulator of synaptic plasticity; increasing Mg^{2+} concentration in the extracellular fluid ($[Mg^{2+}]_o$) within the physiological range leads to permanent enhancement of synaptic plasticity in networks of cultured hippocampal neurons in vitro (Slutsky et al., 2004). Therefore, it is tempting to investigate whether the increase in brain Mg^{2+} content will enhance cognitive function in vivo.

Mg^{2+} concentration is higher in the cerebrospinal fluid than in

DR. Cindy!

Magnesium

- “Spark plug” for the adrenals and energy systems
- Works great in combo with Vitamin C and pantothenic acid
- Take it with other minerals or acidic food or drink, or digestive aids
- Malate and aspartate supports energy production
- Citrate is great for constipation
- Glycinate for muscles
- Threonate for brain health
- 150-800mg QD

DR. Cindy!

J Diet Suppl. 2018 Jan 2;15(1):1-10. doi: 10.1080/19390211.2017.1304486. Epub 2017 May 3.

Impact of Zinc Supplementation on the Clinical Outcomes of Patients with Severe Head Trauma: A Double-Blind Randomized Clinical Trial.

Khazdouz M[1], Mazidi M[2], Ehsaei MR[3], Ferns G[4], Kengne AP[5], Norouzy AR[1].

Author information

Abstract

To determine the effects of zinc supplementation on clinical outcomes of patients with severe head trauma, this double-blind clinical trial randomly allocated 100 patients with severe head trauma, aged between 18 to 65 years, to receive placebo or 120 mg zinc via a nasogastric tube for 15 days. Plasma zinc and copper, 24-hour urinary zinc excretion, Sequential Organ Failure Assessment (SOFA) were assessed on days 1, 7, and 16. Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and white blood cell (WBC) count were measured on days 1 and 16. Glasgow outcome score (GOS), mortality rate on day 28, and length of stay (LOS) were compared between groups. There were no significant differences in baseline data between groups (all $p > .05$). Mean plasma zinc concentration was significantly higher in the zinc group than the placebo group on day 7 (119.5 vs. 81.7 μg/dl, $p < .001$) and day 16 (124.1 vs. 101.1 μg/dl, $p < .001$). The SOFA, GOS, and inflammation factors were significantly better in the zinc-supplemented group by day 16 (all $p < .05$). The LOS was shorter (52 vs. 65 days, $p = .043$) and mortality rate on day 28 was borderline lower (17% vs. 22%, $p = .507$) in zinc versus placebo groups. Zinc supplementation in the study had favorable effects on GOS, SOFA score, and inflammatory markers in patients with severe head injury.

100 patients with severe head trauma aged 18-65
Placebo vs. 120 mg. of zinc
Positive effect on inflammatory markers

DR. Cindy!

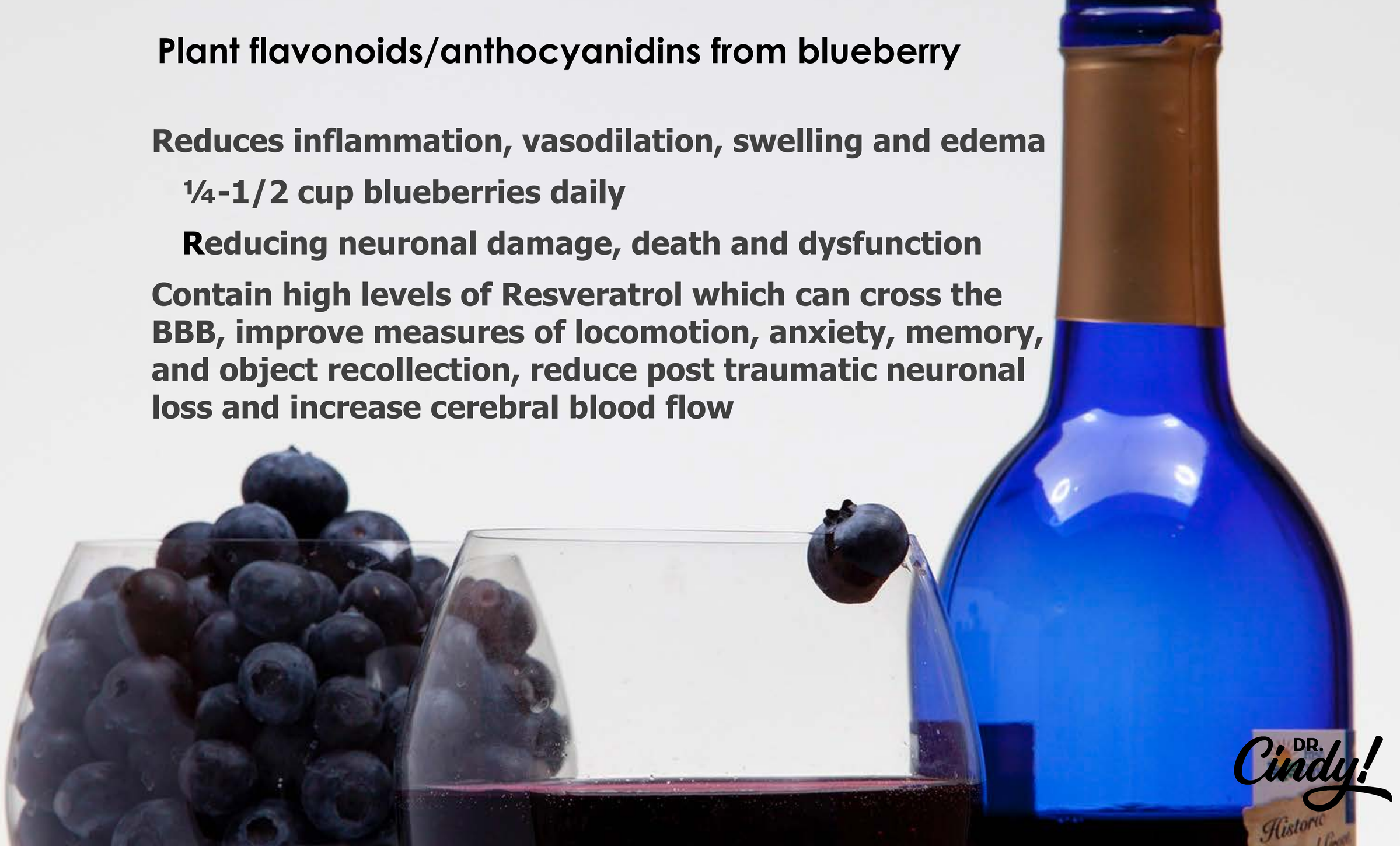
Plant flavonoids/anthocyanidins from blueberry

Reduces inflammation, vasodilation, swelling and edema

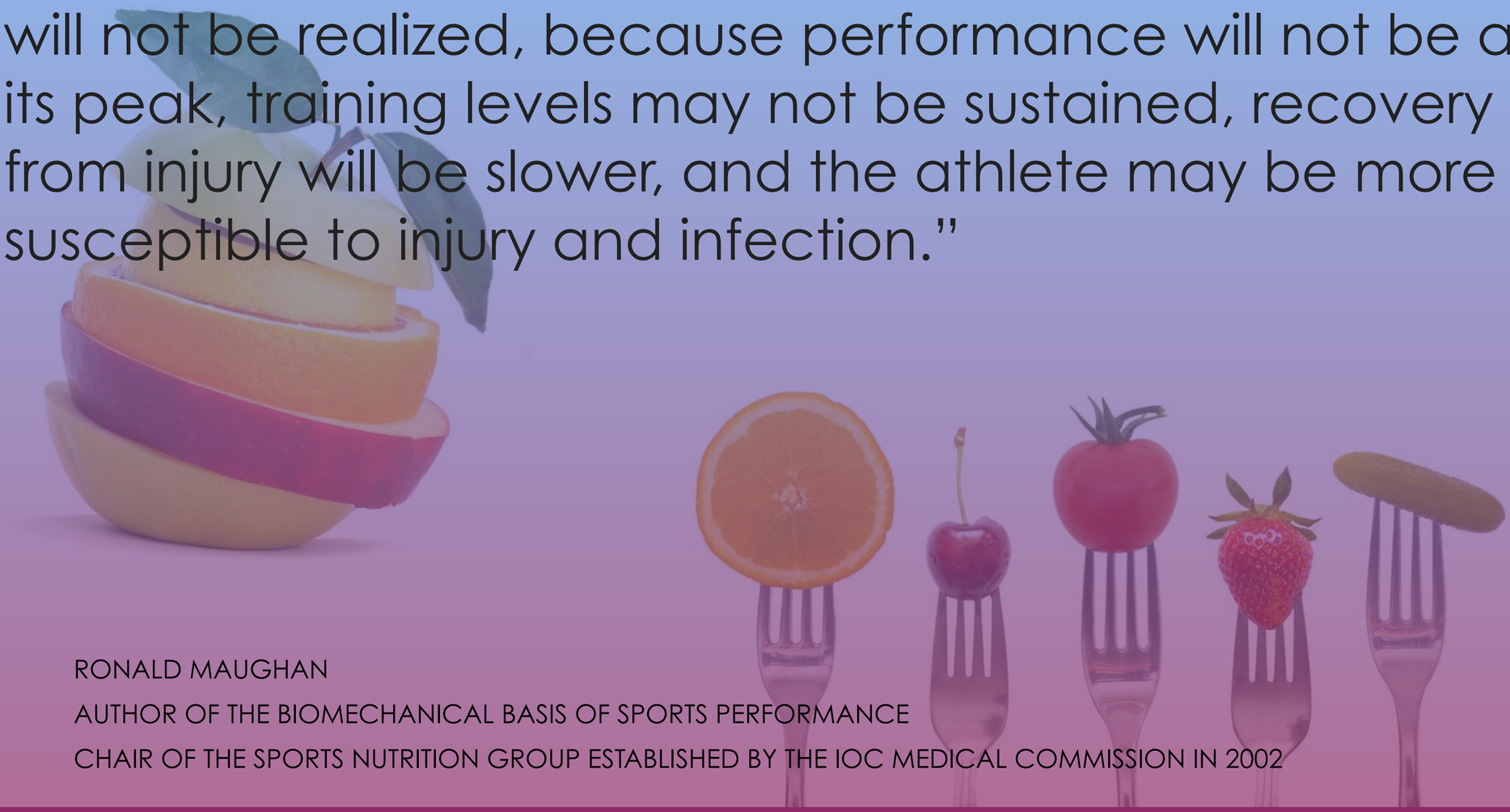
¼-1/2 cup blueberries daily

Reducing neuronal damage, death and dysfunction

Contain high levels of Resveratrol which can cross the BBB, improve measures of locomotion, anxiety, memory, and object recollection, reduce post traumatic neuronal loss and increase cerebral blood flow



"Without proper nutrition, the full potential of the athlete will not be realized, because performance will not be at its peak, training levels may not be sustained, recovery from injury will be slower, and the athlete may be more susceptible to injury and infection."



RONALD MAUGHAN

AUTHOR OF THE BIOMECHANICAL BASIS OF SPORTS PERFORMANCE

CHAIR OF THE SPORTS NUTRITION GROUP ESTABLISHED BY THE IOC MEDICAL COMMISSION IN 2002

DR. Cindy!

Head injuries may spark immune system attack

Consequences of Repeated Blood-Brain Barrier Disruption in Football Players

Nicola Marchi, Jeffrey J. Bazarian, Vikram Puvenna, Mattia Janigro, Chaitali Ghosh, Jianhui Zhong, Tong Zhu, Eric Blackman, Desiree Stewart, Jasmina Ellis, Robert Butler, Damir Janigro

Published: March 6, 2013 • https://doi.org/10.1371/journal.pone.0056805

Published in journal ***PLOS ONE***, the research suggests a new way of thinking about concussions: That the brain degeneration observed among professional football players (including the much-publicized chronic traumatic encephalopathy) could result from an out-of-control immune response, similar to what multiple sclerosis patients experience.

Reader Comments (1)
Media Coverage (0)
Figures

game interviews. S100B serum levels and auto-antibodies against S100B were measured and correlated by direct and reverse immunoassays (n=15 players; 5 games). A subset of players underwent DTI scans pre- and post-season and after a 6-month interval (n=10). Cognitive and functional assessments were also performed. After a game, transient BBB damage measured by serum S100B was detected only in players experiencing the greatest number of sub-concussive head hits. Elevated levels of auto-antibodies against S100B were elevated only after repeated sub-concussive events characterized by BBBD. Serum levels of S100B auto-antibodies also predicted persistence of MRI-DTI abnormalities which in turn correlated with cognitive changes. Even in the absence of concussion, football players may experience repeated BBBD and serum surges of the potential auto-antigen S100B. The correlation of serum S100B, auto-antibodies and DTI changes support a link between repeated BBBD and future risk for cognitive changes.

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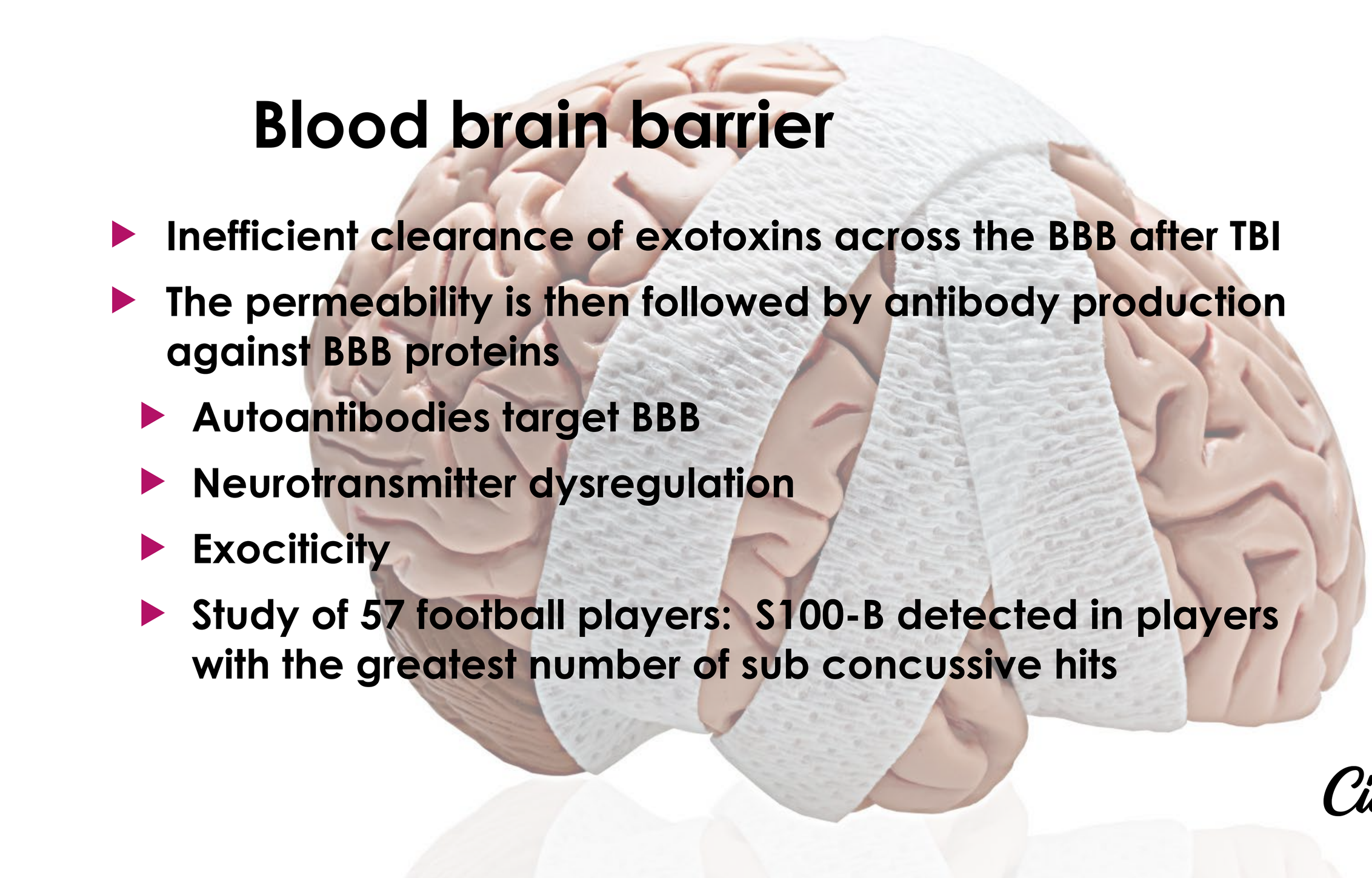
Blood brain barrier



- Inefficient clearance of exotoxins across the BBB after TBI
- The permeability is then followed by antibody production against BBB proteins
  - Autoantibodies target BBB
  - Neurotransmitter dysregulation
  - Exociticity
  - Study of 57 football players: S100-B detected in players with the greatest number of sub concussive hits

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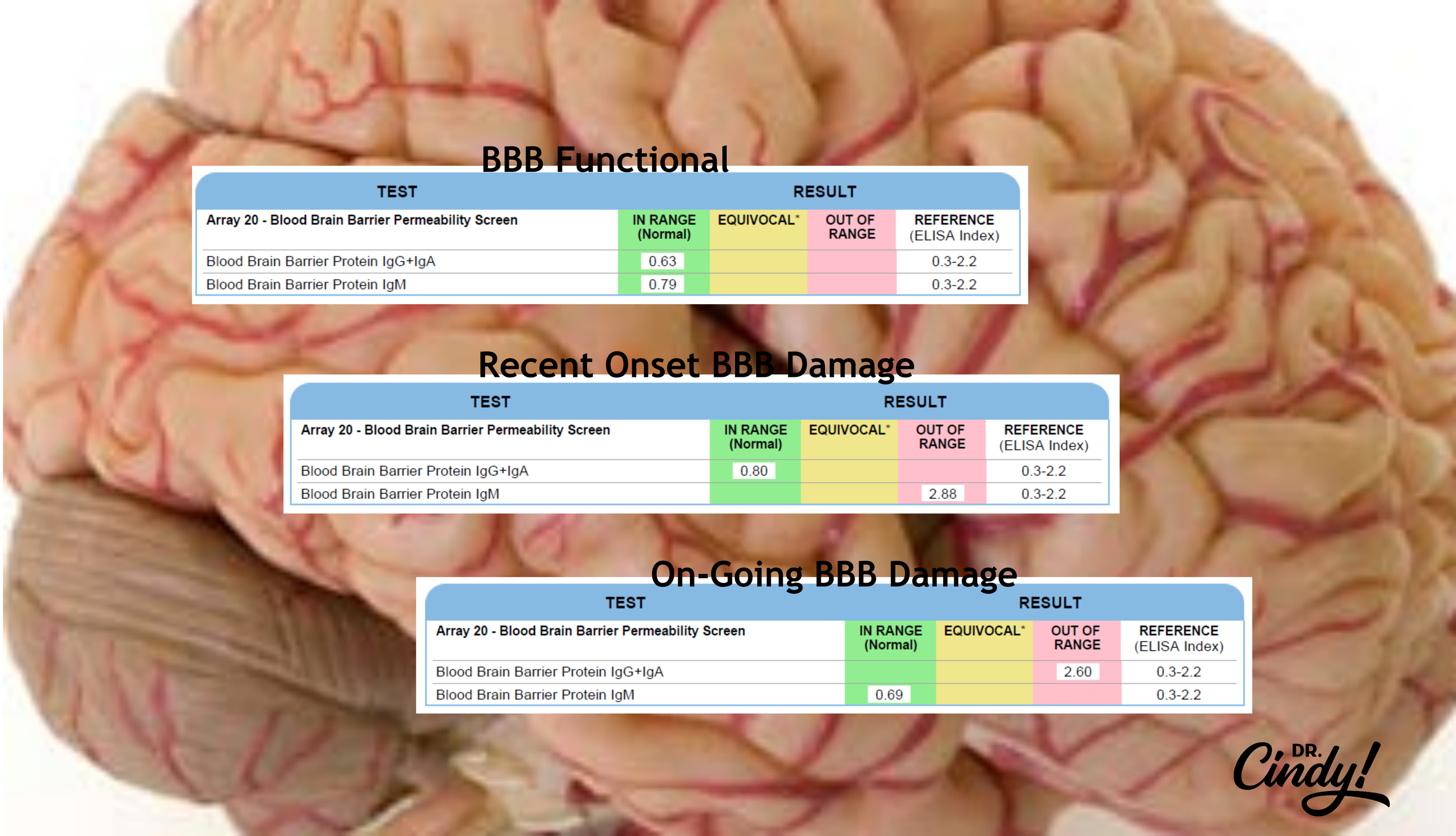

BBB Functional

TEST	RESULT			
Array 20 - Blood Brain Barrier Permeability Screen	**IN RANGE (Normal)**	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE** (ELISA Index)
Blood Brain Barrier Protein IgG+IgA	0.63			0.3-2.2
Blood Brain Barrier Protein IgM	0.79			0.3-2.2

Recent Onset BBB Damage

TEST	RESULT			
Array 20 - Blood Brain Barrier Permeability Screen	**IN RANGE (Normal)**	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE** (ELISA Index)
Blood Brain Barrier Protein IgG+IgA	0.80			0.3-2.2
Blood Brain Barrier Protein IgM			2.88	0.3-2.2

On-Going BBB Damage

TEST	RESULT			
Array 20 - Blood Brain Barrier Permeability Screen	**IN RANGE (Normal)**	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE** (ELISA Index)
Blood Brain Barrier Protein IgG+IgA			2.60	0.3-2.2
Blood Brain Barrier Protein IgM	0.69			0.3-2.2

DR. Cindy!

Transl Stroke Res. Author manuscript; available in PMC 2012 Jan 30.
Published in final edited form as:
Transl Stroke Res. 2011 Dec; 2(4): 492–516.
doi: 10.1007/s12975-011-0125-x

PMCID: PMC3268209
NIHMSID: NIHMS349424

Blood-brain barrier pathophysiology in traumatic brain injury

Adam Chodobski, Brian J. Zink, and Joanna Szmydynger-Chodobska

Author information ▶ Copyright and License information ▶

Abstract

Go to:

The blood-brain barrier (BBB) is formed by tightly connected cerebrovascular endothelial cells, but its normal function also depends on paracrine interactions between the brain endothelium and closely located glia. There is a growing consensus that brain injury, whether it is ischemic, hemorrhagic, or traumatic, leads to dysfunction of the BBB. Changes in BBB function observed after injury are thought to contribute to the loss of neural tissue and to affect the r
considering the entire gliovascular unit, rath
cellular and molecular responses to traumati
breakdown in TBI, the role of blood-borne f
changes in BBB permeability and post-traur
factors associated with TBI that may contrib
neuroinflammation and the possible effect o
described. Finally, the potential role of the E
of normal BBB function after injury and/or by harnessing the cerebrovascular endothelium to produce neurotrophic growth factors will be discussed.

Normally functioning BBB is key to restore brain homeostasis and to create an optimal microenvironment for neuronal repair.

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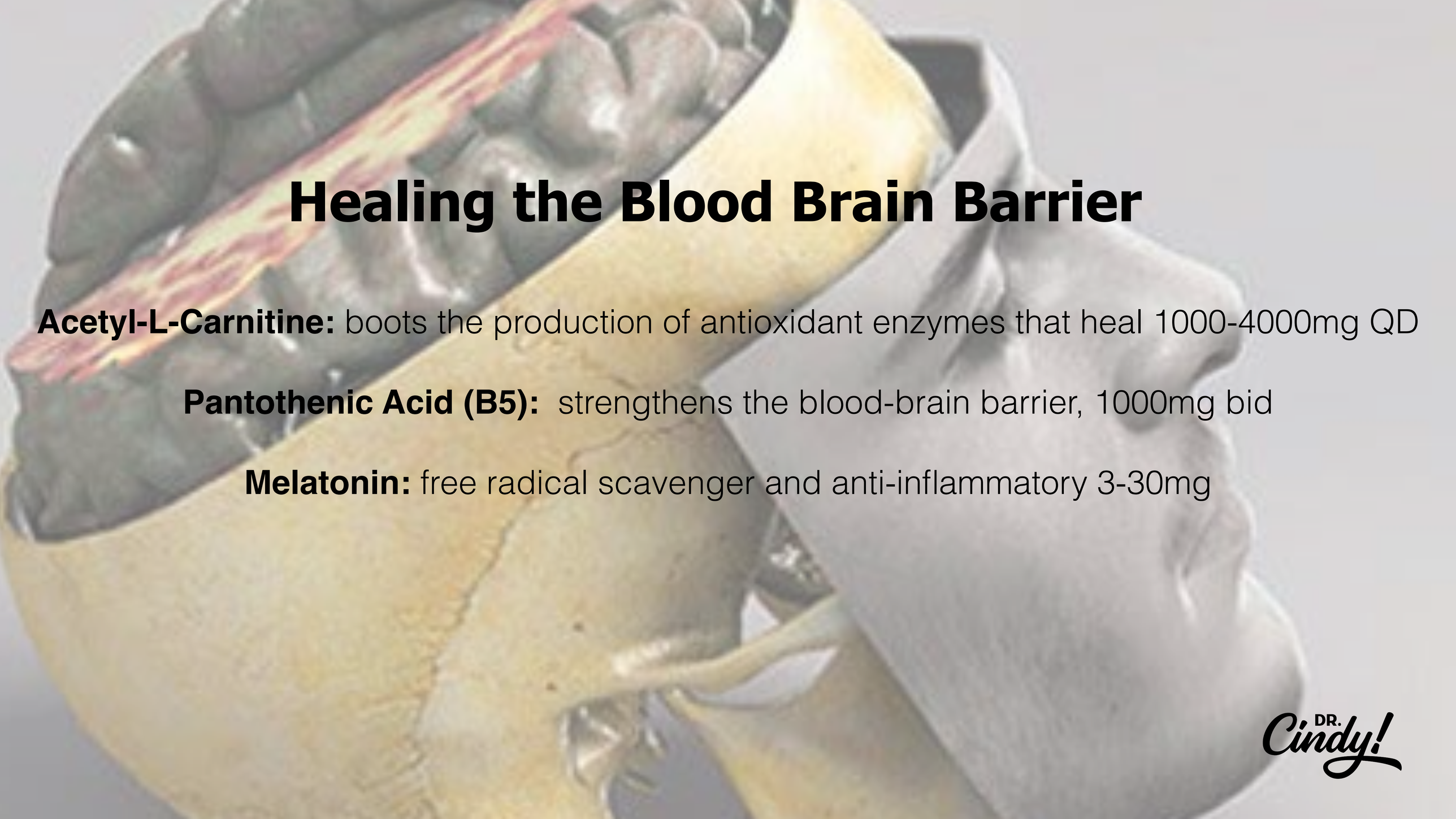


Healing the Blood Brain Barrier

Acetyl-L-Carnitine: boots the production of antioxidant enzymes that heal 1000-4000mg QD

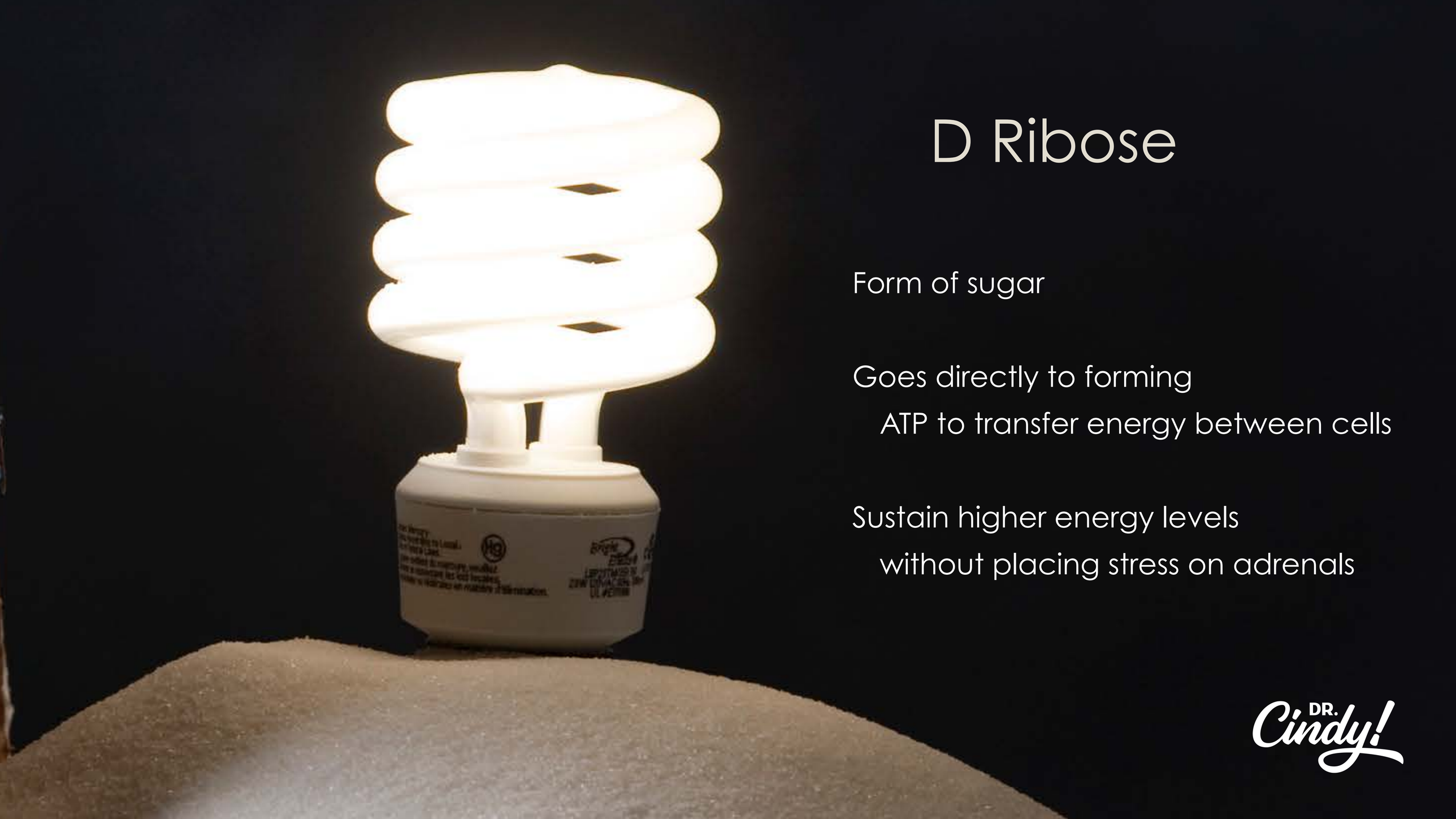
Pantothenic Acid (B5): strengthens the blood-brain barrier, 1000mg bid

Melatonin: free radical scavenger and anti-inflammatory 3-30mg

DR. Cindy!

Acetyl-L-Carnitine

- Increases the production of Acetyl Choline
- Moves fatty acids into mitochondria to produce energy
- Penetrates the BBB
- Helps with alertness
- Improves concentration



D Ribose

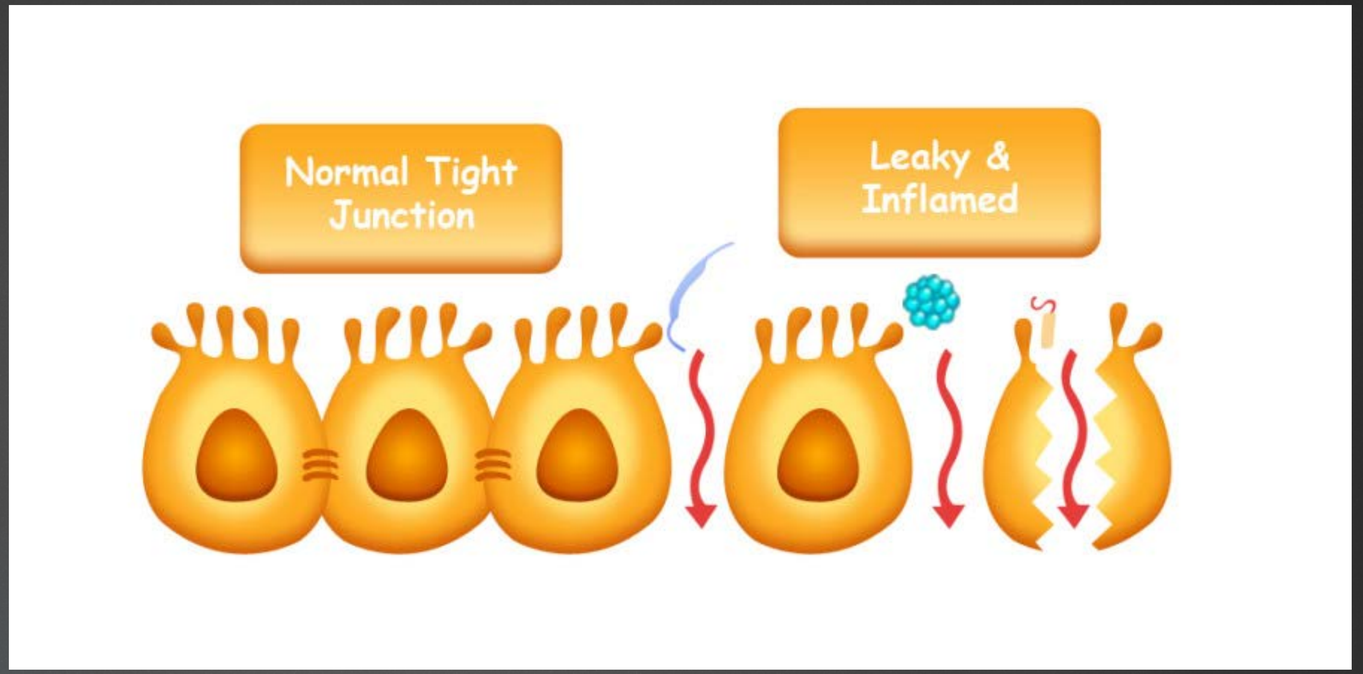
Form of sugar

Goes directly to forming
ATP to transfer energy between cells

Sustain higher energy levels
without placing stress on adrenals

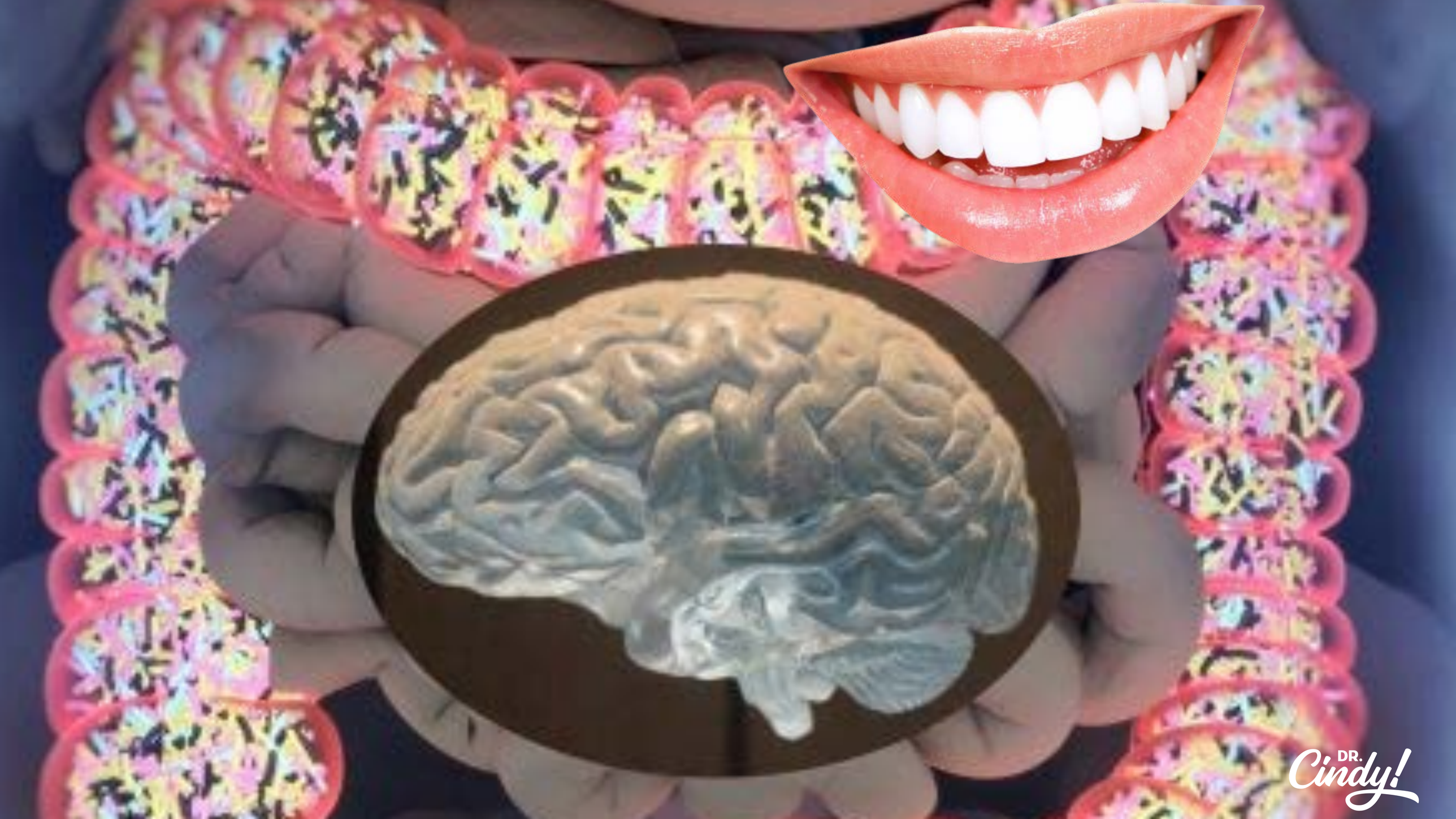
DR. Cindy!

Intestinal Permeability screen



TEST	RESULT			
Array 2 – Intestinal Antigenic Permeability Screen	**IN RANGE (Normal)**	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE** (ELISA Index)
Actomyosin IgA **	7.65			0.0-20
Occludin/Zonulin IgG			5.13	0.2-1.5
Occludin/Zonulin IgA	1.05			0.1-1.8
Occludin/Zonulin IgM			3.90	0.1-2.1
Lipopolysaccharides (LPS) IgG		1.21		0.1-1.6
Lipopolysaccharides (LPS) IgA	0.88			0.1-1.8
Lipopolysaccharides (LPS) IgM	1.49			0.1-2.0

DR. Cindy!


DR.
Cindy!

Caries are the most common infectious disease in the mouth.

J Clin Microbiol. 2005 Nov; 43(11): 5721–5732.
doi: 10.1128/JCM.43.11.5721-5732.2005

PMCID: PMC1287824
PMID: 16272510

Defining the Normal Bacterial Flora of the Oral Cavity

Jørn A. Aas,[1,2,*] Bruce J. Paster,[1,3] Lauren N. Stokes,[1] Ingar Olsen,[2] and Floyd E. Dewhirst[1,3]

Author information ► Article notes ► Copyright and License information ► Disclaimer

ABSTRACT

Go to:

More than 700 bacterial species or phylotypes, of which over 50% have not been cultivated, have been detected in the oral cavity. Our purposes were (i) to utilize culture-independent molecular techniques to ealthy human oral cavity, including not-oject specificity of bacterial colonization. Nine sites from five clinically healthy subjects were analyzed. Sites included tongue dorsum, lateral sides of tongue, buccal epithelium, hard palate, soft palate, supragingival plaque of tooth surfaces, subgingival plaque, maxillary anterior vestibule, and tonsils. 16S rRNA genes from sample DNA were amplified, cloned, and transformed into *Escherichia coli*. Sequences of 16S rRNA genes were used to determine species identity or closest relatives. In 2,589 clones, 141 predominant species were detected, of which over 60% have not been cultivated. sites belonged to the genera *Gemella*, *Gran* were subject specific and detected in most 30 different predominant species, and the n ranged from 34 to 72. Species typically associated with periodontitis and caries were not detected. There is a distinctive predominant bacterial flora of the healthy oral cavity that is highly diverse and site and subject specific. It is important to fully define the human microflora of the healthy oral cavity before we can understand the role of bacteria in oral disease.

The Gut brain mouth connection

700+ bacterial species have been detected in the oral cavity

DR. Cindy!

DNA CONNEXIONS

Patient with Root Canal 33 Pathogens Detected

PATIENT: [redacted]

DOCTOR: Dr. John Rothchild

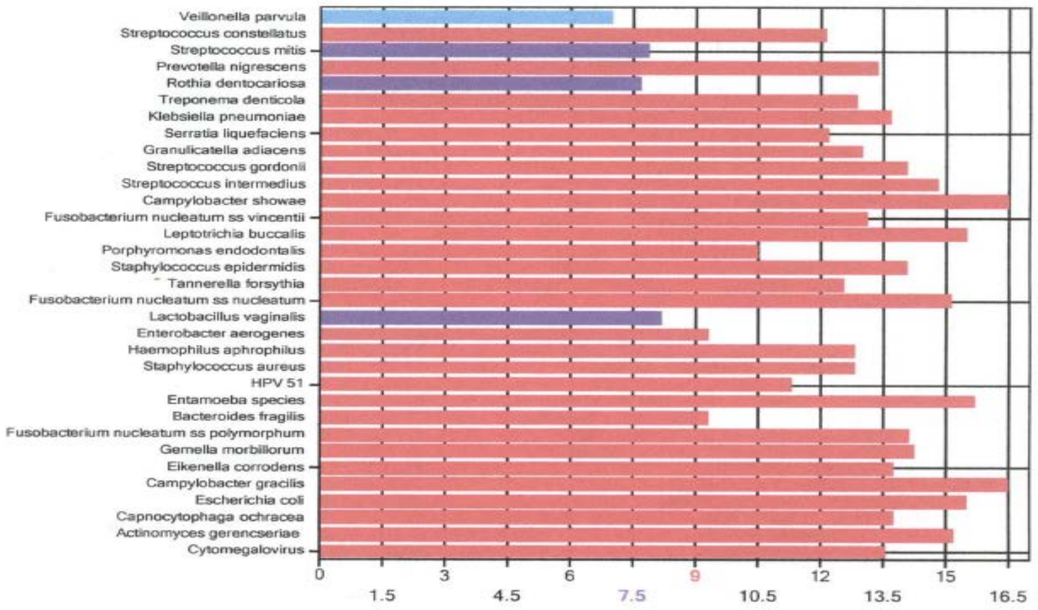
Test ID: 05605

Full View Test

Sample Collected	Sample Received	Sample Tested	Test Reported
02/13/2017	02/16/2017	04/27/2017	04/27/2017

Sample Type: #19 Root Canal Tooth

The following microbes were detected in the sample that was submitted for testing:



After 8 weeks of broad spectrum antimicrobial

PATIENT: [redacted]

DOCTOR: Dr. John Rothchild

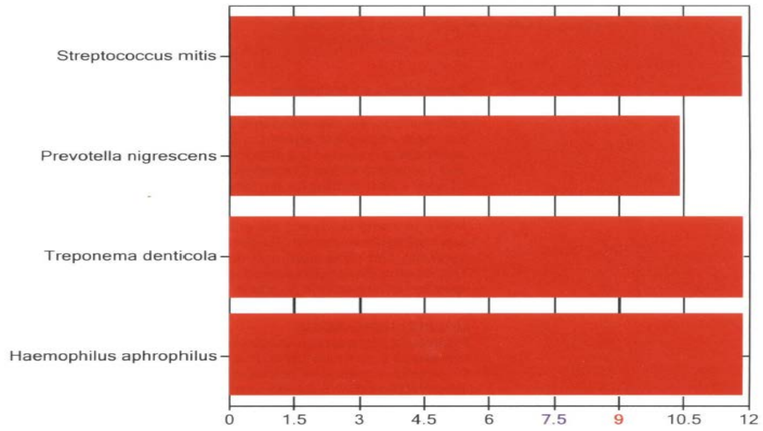
Test ID: 05834

Full View Test

Sample Collected	Sample Received	Sample Tested	Test Reported
04/26/2017	05/01/2017	05/26/2017	06/01/2017

Sample Type: #19 Paper Points (2 Month Post-op, 1 Month Biocydin)

The following microbes were detected in the sample that was submitted for testing:



DR. Cindy!

Periodontitis

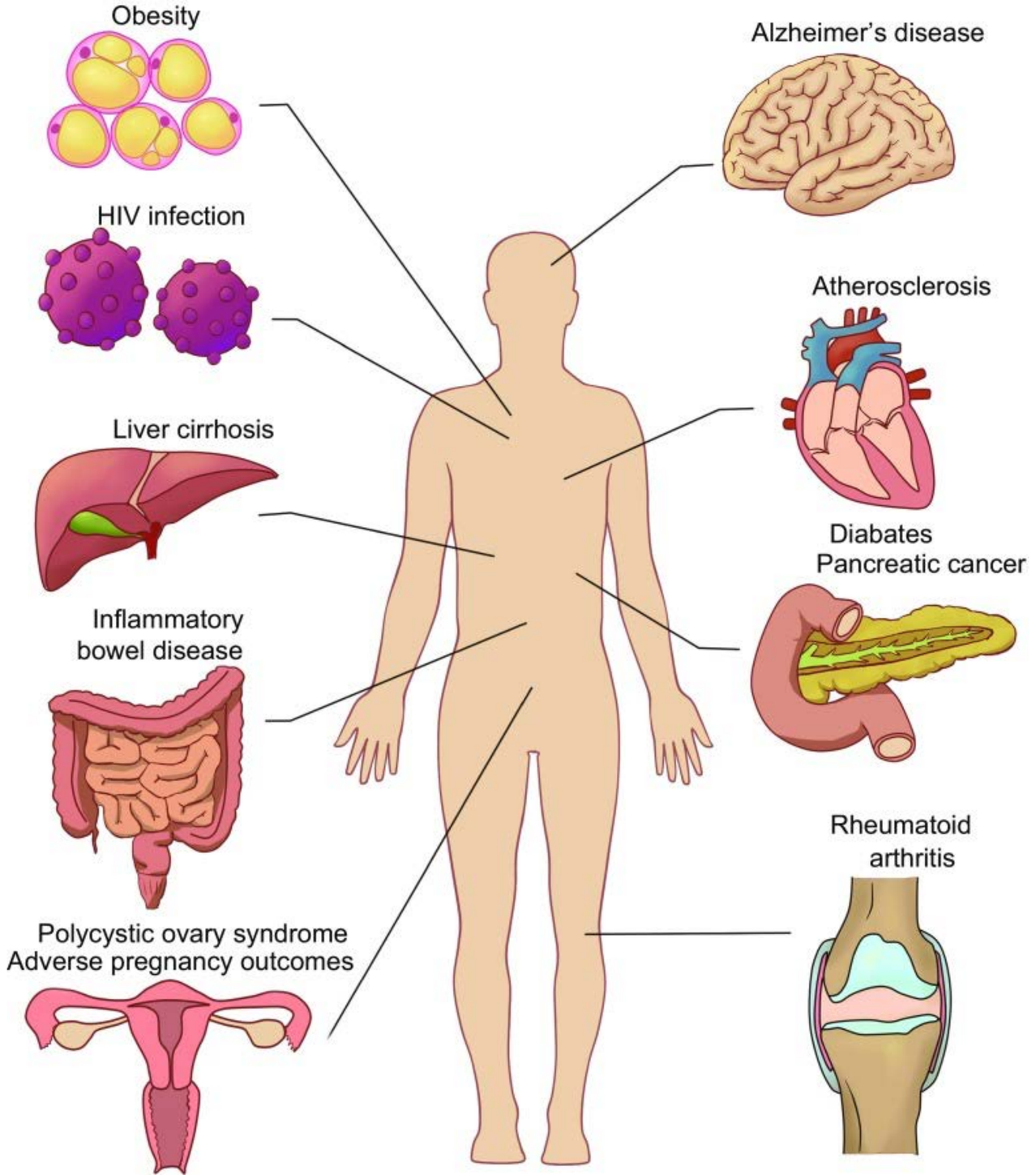
Caries

Peri-implantitis

Microbiome
and
oral diseases

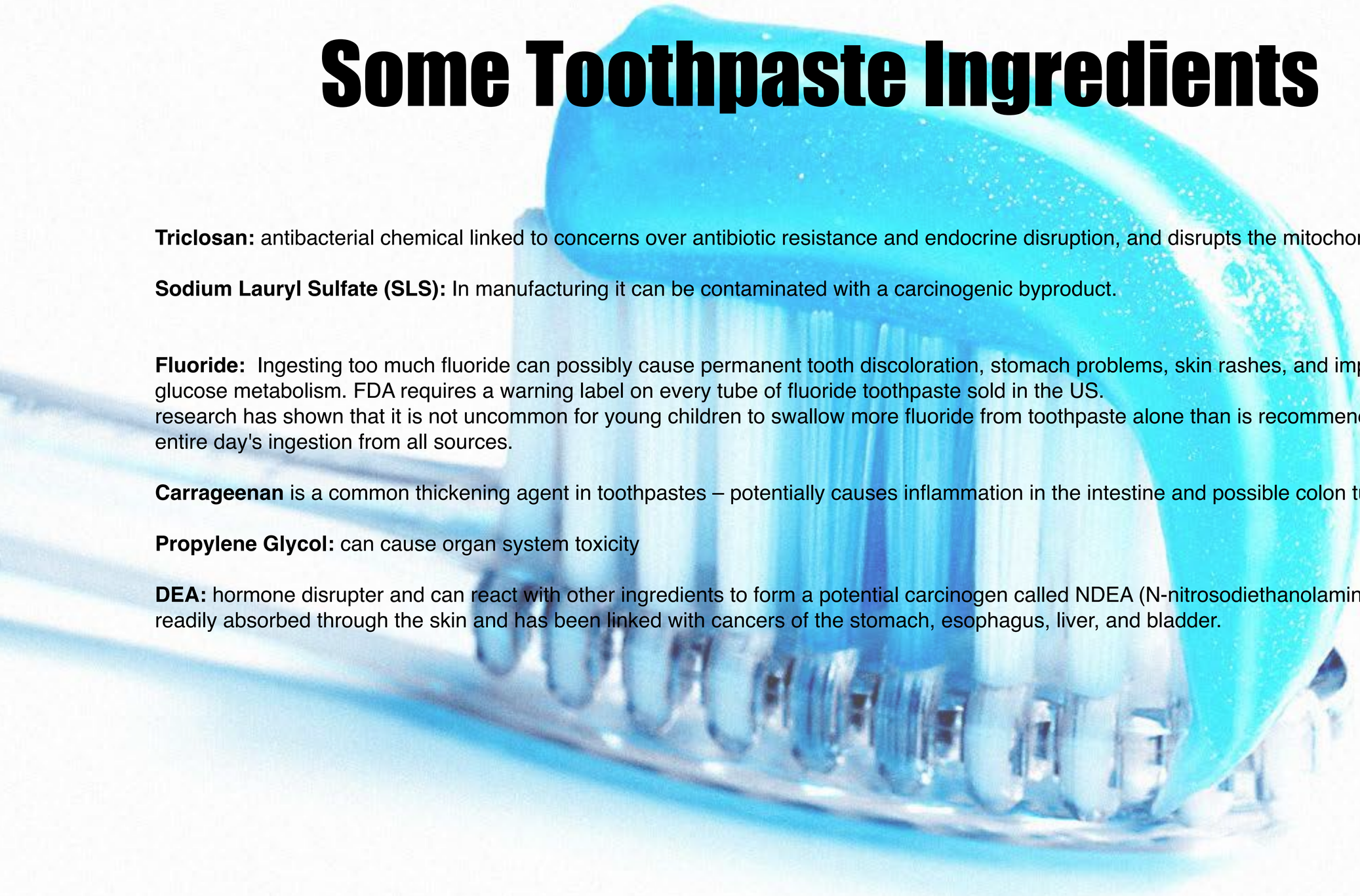
Mucosa diseases

Oral cancer


Obesity
HIV infection
Liver cirrhosis
Inflammatory bowel disease
Polycystic ovary syndrome
Adverse pregnancy outcomes
Alzheimer's disease
Atherosclerosis
Diabates
Pancreatic cancer
Rheumatoid arthritis

DR. Cindy!

Some Toothpaste Ingredients



Triclosan: antibacterial chemical linked to concerns over antibiotic resistance and endocrine disruption, and disrupts the mitochondria

Sodium Lauryl Sulfate (SLS): In manufacturing it can be contaminated with a carcinogenic byproduct.

Fluoride: Ingesting too much fluoride can possibly cause permanent tooth discoloration, stomach problems, skin rashes, and impairment in glucose metabolism. FDA requires a warning label on every tube of fluoride toothpaste sold in the US.
research has shown that it is not uncommon for young children to swallow more fluoride from toothpaste alone than is recommended as an entire day's ingestion from all sources.

Carrageenan is a common thickening agent in toothpastes – potentially causes inflammation in the intestine and possible colon tumors.

Propylene Glycol: can cause organ system toxicity

DEA: hormone disrupter and can react with other ingredients to form a potential carcinogen called NDEA (N-nitrosodiethanolamine), which is readily absorbed through the skin and has been linked with cancers of the stomach, esophagus, liver, and bladder.

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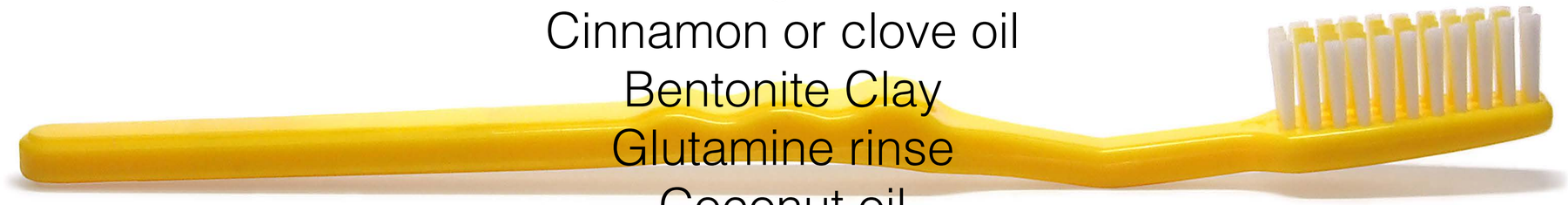
What can we put on our toothbrush?

Bilberry extract
Noni
Milk Thistle
Echinacea
Goldenseal
Shiitake
White willow
Garlic
Grapeseed Extract
Black Walnut
Gentian
Oregano Oil
Baking Soda
Cinnamon or clove oil
Bentonite Clay
Glutamine rinse
Coconut oil
Chewable or liquid CoQ10



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Certain circumstances in sport may increase the susceptibility of athletes to infectious diseases.

INTRODUCTION

Athletes are prone to different medical problems which are
activities and may result in training abstinence. Injuries are one of the most common of these problems. Another medical problem which could be a reason for athletes' training abstinence is infection. Certain circumstances in sport may increase the susceptibility of athletes to infectious diseases. On the other hand, athletes with infectious diseases may be at risk for severe complications if they continue their physical activity. Finally they may have transmittable diseases, so other people including sports competitors, trainers and even audiences could be at risk. Transmission of infectious diseases in sports usually occurs via direct contacts, fecal oral routs, common source exposure and airborne or droplets [1, 2].

Despite high prevalence of infectious diseases in athletes and the important role of this medical problem in training and competition abstinence, there is little evidence and guidelines for physician in the literature [2–4].

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Immune function in sport and exercise

Michael Gleeson

1 AUG 2007 // https://doi.org/10.1152/japplphysiol.00008.2007

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Abstract

Regular moderate e
with a completely s
temporary depressi
burst, lymphocyte p
fter exercise, depending on the intensity and duration of the exercise bout. Postexercise
mmune function dysfunction is most pronounced when the exercise is continuous,
prolonged (>1.5 h), of moderate to high intensity (55–75% maximum O_2 uptake), and
d without food intake. Periods of intensified training (overreaching) lasting 1 wk or
t in longer lasting immune dysfunction. Although elite athletes are not
deficient, it is possible that the combined effects of small changes in
ameters may compromise resistance to common minor illnesses, such as
act infection. However, this may be a small price to pay as the anti-
ts of exercise mediated through cytokines and/or downregulation of toll-
tor exp
ercise.

Prolonged bouts of strenuous exercise cause a temporary depression of various aspects of immune function.

Periods of intensified training lasting 1 week or more may result in longer lasting immune dysfunction compromising resistance to common minor illnesses

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Exercise and Immune System

Exercise and changes in immunity have a proven relationship [5]. Very heavy sports may increase risk of infections, such
training [6]. In a
infection (URI)
system. Some ex
infection. Accor
decreases rate of
inhibitory effect
to the second the
exercise, this time is an "open window" and the risk of infections may be increased in this period[5].
However, despite these attracti
different parts of immune syste

Exercise may influence quality
(NK) cells, neutrophils and lym
elow pre-exercise level after 30 min which probably is not clinically important. Heavy exercise does
company a decreased cytotoxic effect of NK cells while moderate, regular activity increases number of
K cel

Neutro
Lymph
them [4].
air neutrophil function.
e but moderate exercise increases

Salivary IgA decreases with heavy and prolonged activities but IgG level has a small decrease. While low-intensity short term exercise incr
<1.5 following exercise. In acut
α, interleukin (IL) 1 and IL6 and
increased [9]. Physical activity m
to
F)
and change of breathing from nose to mouth breathing induces progressive cooling and drying of the
spiratory tract mucous. Decreasing movement of ciliated cells and increasing mucosal viscosity, finally
pairing filtering of microorganisms from the upper respiratory tract system [10].

In summary athletes have brief immunosupression after acute, heavy exercise in the open window period, when there is reduced ciliary activity, lymphocyte count, CD4 to CD8 ratio and mucosal IgA level. Indeed, intensive activity is associated with increased risk of infection. According to J curve, regular moderate exercise can reduce respiratory tract infections.

In a marathon, 33.3% of athletes who ended the marathon got an upper respiratory tract infection.

Heavy exercise does accompany a decreased cytotoxic effect of NK cells

High intensity exercise impairs neutrophil production

Salivary IgA decrease with heavy and prolonged activities.

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Fungal Skin Infections

Fungal skin infections termed also as dermatophytosis or ringworms are one of the most common infections in athletes especially those with contact sports. Twenty to 77% of wrestlers are infected with s, 34.2% of the wrestlers with skin lesions had fungal ton tonsurance (30%), and epidermophyton floccosum isms [21]. Skin to skin contact is the main way of uipment (including mats) in transmission is not clear and prevalence of contamination in the environment is variable in different studies. Dermatophytosis presents as circular, scaly, red, itchy skin lesions, usually with an active border, in different parts of the body including head and neck, trunk and extremities [3, 25]

20-77% of wrestlers are infected with dermatophytes and up to 34% had fungal infections

Fungal infections are one of the most common in contact sports

Athlete's foot or tinea pedis is common in athletes because it grows in dark, moist and warm environments. Sweaty feet, tight shoes, not drying one's feet after swimming or bathing are predisposing factors [26]. Organisms causing tinea pedis include Trichophyton rubrum and Trichophyton mentagrophytes[27]. The lesions are peels, crack toes. It may cause itching and burning of the feet.

Athlete's foot is also common

Fungal infections can affect the nails. These lesions can cause scaling, crumbing, thickening and partial loss of the nails. The diagnosis is made with scraping the scaly area [26]. Tinea corporis is common among wrestlers and athletes who have close contact with each other, so it's called tinea corporis gladiatorum. The most common organisms are trichophyton tonsurans, trichophyton rubrum and microsporum canis. The lesions are annular scaling, erythematous plaques with swolen margins. Topical corticosteroids may change the clinical picture of tinea corporis and lesions present without raised margins [28]. Because wrestlers with dermatophytosis are prohibited from participating in contact sports, surveillance and rapid initiation of therapy can reduce the absence rate of athletes from competition [29].

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Most common: Respiratory tract
Other common: digestive system, skin and underlying tissues, genitourinary system

Infection is a huge reason athletes miss training session

Other factors such as lack of sleep and inadequate nutrition (particularly deficiencies of protein and essential micronutrients can also depress immunity)
Gleeson 2016

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Illness among athletes at major competitions

Incidence proportion (%)

In Major international games lasting 9-18 days,
6-17% of registered athletes are likely to suffer an illness episode.

Games / Competition	Season	Duration (days)	Athletes (n)	Males (n)	Females (n)	All athletes (%)	Males (%)	Females (%)	Respiratory (% total)
Paralympics 2014	Winter	12	547	418	129	**17.4**	17.0	18.6	30
Olympics 2014	Winter	18	2780	1659	1121	**8.9**	7.3	10.9	64
Paralympics 2012	Summer	14	3565	2347	1218	**14.2**	17.6	20.1	34
Olympics 2012	Summer	17	10568	5892	4676	**7.2**	5.3	8.6	41
Youth Olympics 2012	Winter	10	1021	562	459	**8.4**	6.0	11.0	61
IAAF 2011	Summer	9	1851	971	880	**6.8**	7.1	7.7	39
Olympics 2010	Winter	17	2567	1522	1045	**7.2**	5.2	8.7	63
IAAF 2009	Summer	9	1979	1082	897	**6.8**	5.6	8.4	36
FINA 2009	Summer	18	2318	1306	1012	**6.6**	5.1	7.9	50

IAAF: International Athletics Federation; FINA: Federation Internationale de Natation; n = number of registered athletes.

by Professor Mike Gleeson

Illness appears to be consistently more common in female athletes
Also higher in winter compared to summer Olympic games

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Potential causes of illness in athletes

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www.mysportscience.com

Increased exposure to pathogens

- Lung ventilation
- Skin abrasions
- Foreign travel
- Crowds
- Poor hygiene

Too much stress for the athlete

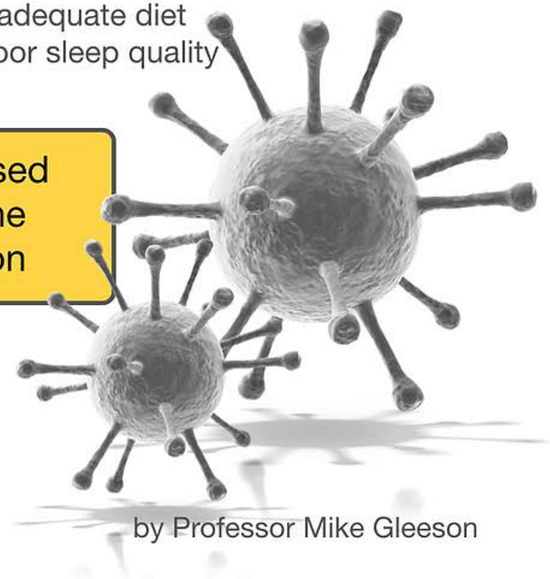
- Physiological stress
- Psychological stress
- Environmental stress
- Inadequate diet
- Poor sleep quality

Increased susceptibility to develop symptoms of infection

Depressed immune function

- Breathing cold air
- Breathing dry air
- Breathing polluted air

Allergy / inflammation



by Professor Mike Gleeson

DR. Cindy!

Daniel

History/Physical

19YOM
WT: 237 HT 6’1” P: 85, R 18, T 99.0, BP seated left 141/70, Right 142/66
EENT WNL, Chest, Lungs, Abdomen WNL

Concussed on the field
Evaluated by athletic trainer
Returned to play after headaches vanished and Baseline levels of Scat 5 were obtained
Presents with elevated blood pressure and sleep disruption
Stress Level ranges from 4-7/10
Takes over an hour to fall asleep
Waking at 3 am
T, TH up at 5 am for training, other days at 8
Alcohol consumption
Headaches
Chest pain
Heart races
Anxiety
EKG WNL
Light headed when skipping meals
Afternoon fatigue
Difficulty gaining weight

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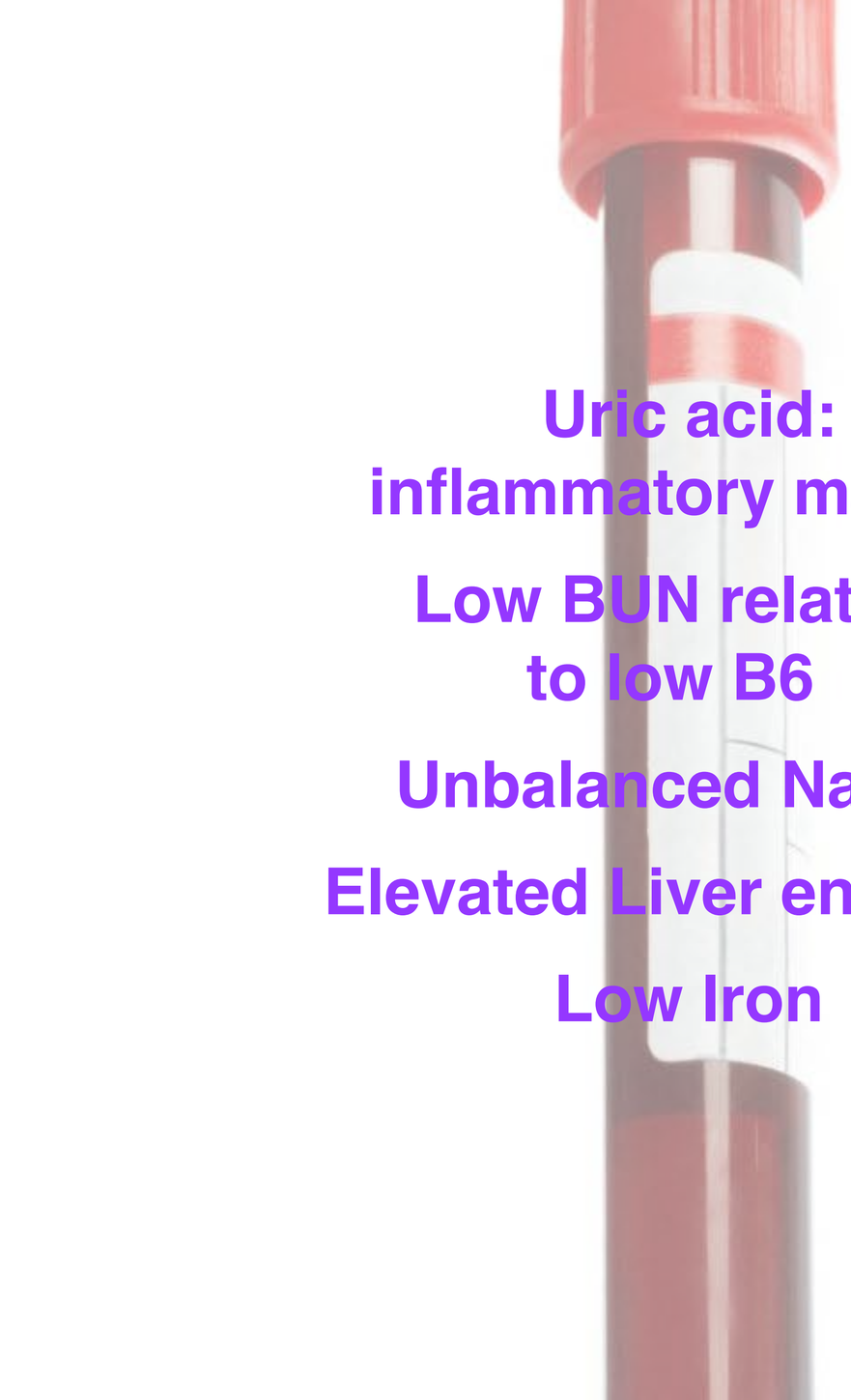

Uric acid: inflammatory marker

Low BUN relates to low B6

Unbalanced Na, K

Elevated Liver enzymes

Low Iron

Item Name	Client's #	Results	Functional Ranges	Laboratory Ranges
Glucose	89.00	NORMAL	85.00 - 99.00	65.00 - 99.00
HbA1C	5.10	NORMAL	4.80 - 5.60	4.80 - 5.60
Uric Acid	7.90	HIGH	3.70 - 6.00	3.70 - 8.60
BUN	11.00	LOW	13.00 - 18.00	8.00 - 27.00
Creatinine	1.08	NORMAL	0.85 - 1.10	0.76 - 1.27
BUN/Creat Ratio	10.00	NORMAL	10.00 - 20.00	10.00 - 22.00
Sodium	143.00	HIGH	135.00 - 140.00	134.00 - 144.00
Potassium	4.70	HIGH	4.00 - 4.50	3.50 - 5.20
Chloride	101.00	NORMAL	100.00 - 106.00	97.00 - 108.00
Calcium	9.70	NORMAL	9.20 - 10.10	8.70 - 10.20
Magnesium	2.20	NORMAL	2.00 - 2.50	1.60 - 2.60
CO2	23.00	NORMAL	22.00 - 27.00	19.00 - 28.00
Protein	7.30	NORMAL	6.90 - 7.40	6.00 - 8.50
Albumin	4.60	NORMAL	4.00 - 5.00	3.50 - 5.50
Globulin	2.70	NORMAL	2.40 - 2.80	1.50 - 4.50
A/G Ratio	1.70	NORMAL	1.50 - 2.00	1.10 - 2.50
Alk Phos	104.00	LAB HIGH	44.00 - 90.00	44.00 - 102.00
LDH	281.00	LAB HIGH	140.00 - 180.00	0.00 - 225.00
AST/SGOT	33.00	HIGH	10.00 - 26.00	0.00 - 40.00
ALT/SGPT	42.00	HIGH	10.00 - 26.00	0.00 - 44.00
Bilirubin	0.80	NORMAL	0.10 - 1.20	0.00 - 1.20
Serum Iron	75.00	LOW	85.00 - 135.00	40.00 - 155.00
TIBC	359.00	HIGH	250.00 - 350.00	250.00 - 390.00
Ferritin	144.00	NORMAL	33.00 - 236.00	30.00 - 400.00
RBC	5.38	HIGH	4.40 - 4.90	4.14 - 5.80
HGB	16.00	HIGH	14.00 - 15.00	12.60 - 17.70
HCT	46.40	NORMAL	39.00 - 50.00	37.50 - 51.00
MCV	86.00	NORMAL	85.00 - 92.00	79.00 - 97.00
MCH	29.70	NORMAL	27.70 - 32.00	26.60 - 33.00
MCHC	34.50	NORMAL	32.00 - 35.00	31.50 - 35.70
RDW	12.80	NORMAL	12.30 - 15.00	12.30 - 15.40
WBC	4.10	LOW	5.00 - 8.00	3.40 - 10.80
Neutrophils	49.00	NORMAL	40.00 - 60.00	> 0.00

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CRP Significantly elevated inflammatory marker

Monocytes elevated indicate infection

Item Name	Client's #	Results	Functional Ranges	Laboratory Ranges
Lymphocytes	27.00	NORMAL	25.00 - 40.00	> 0.00
Monocytes	22.00	HIGH	4.00 - 7.00	> 0.00
Eosinophils	1.00	NORMAL	0.00 - 3.00	> 0.00
Basophils	1.00	NORMAL	0.00 - 3.00	> 0.00
Platelets	169.00	NORMAL	155.00 - 379.00	155.00 - 379.00
ESR	4.00	NORMAL	0.00 - 15.00	0.00 - 15.00
CRP	10.90	LAB HIGH	0.00 - 4.90	0.00 - 4.90
Triglycerides	127.00	HIGH	75.00 - 100.00	0.00 - 149.00
Cholesterol	158.00	NORMAL	150.00 - 199.00	100.00 - 199.00
LDL	98.00	NORMAL	0.00 - 99.00	0.00 - 99.00
HDL	35.00	LAB LOW	55.00 - 100.00	> 39.00
TSH	1.97	NORMAL	1.80 - 3.00	0.45 - 4.50
T4	7.90	NORMAL	6.00 - 12.00	4.50 - 12.00
T3	124.00	NORMAL	100.00 - 180.00	71.00 - 180.00
T3 U	32.00	NORMAL	28.00 - 38.00	24.00 - 39.00
fT4	1.45	NORMAL	1.00 - 1.50	0.82 - 1.77
fT3	2.50	LOW	3.00 - 4.00	2.00 - 4.40
TPO - Ab	Negative	NORMAL	0.00 - 34.00	0.00 - 34.00
Insulin	11.30	NORMAL	2.60 - 24.90	2.60 - 24.90
VLDL	25.00	NORMAL	5.00 - 40.00	5.00 - 40.00
Iron Saturation	21.00	NORMAL	15.00 - 55.00	15.00 - 55.00
25 (OH) Vitamin D	40.70	NORMAL	35.00 - 100.00	30.00 - 100.00
Thyroglobulin, Antibody	0.90	NORMAL	0.00 - 0.90	0.00 - 0.90
UIBC	284.00	NORMAL	150.00 - 375.00	150.00 - 357.00

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Comprehensive Stool Analysis / Parasitology x3

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group	4+ Alpha hemolytic strep	3+ Citrobacter freundii complex
4+ Bifidobacterium spp.		
2+ Escherichia coli		
1+ Lactobacillus spp.		
NG Enterococcus spp.		
3+ Clostridium spp.		

NG = No Growth

BACTERIA INFORMATION

Expected /Beneficial bacteria make up a significant portion of the total microflora in a healthy & balanced GI tract. These beneficial bacteria have many health-protecting effects in the GI tract including manufacturing vitamins, fermenting fibers, digesting proteins and carbohydrates, and propagating anti-tumor and anti-inflammatory factors.
Clostridia are prevalent flora in a healthy intestine. Clostridium spp. should be considered in the context of balance with other expected/beneficial flora. Absence of clostridia or over abundance relative to other expected/beneficial flora indicates bacterial imbalance. If *C. difficile* associated disease is suspected, a Comprehensive Clostridium culture or toxigenic *C. difficile* DNA test is recommended.
Commensal (Imbalanced) bacteria are usually neither pathogenic nor beneficial to the host GI tract. Imbalances can occur when there are insufficient levels of beneficial bacteria and increased levels of commensal bacteria. Certain commensal bacteria are reported as dysbiotic at higher levels.
Dysbiotic bacteria consist of known pathogenic bacteria and those that have the potential to cause disease in the GI tract. They can be present due to a number of factors including: consumption of contaminated water or food, exposure to chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

YEAST CULTURE

Normal flora	Dysbiotic flora
No yeast isolated	

MICROSCOPIC YEAST

Result:	Expected:
Few	None - Rare

YEAST INFORMATION

Yeast normally can be found in small quantities in the skin, mouth, intestine and mucocutaneous junctions. Overgrowth of yeast can infect virtually every organ system, leading to an extensive array of clinical manifestations. Fungal diarrhea is associated with broad-spectrum antibiotics or alterations of the patient's immune status. Symptoms may include abdominal pain, cramping and

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Been taking Melatonin 1-3 mg every night

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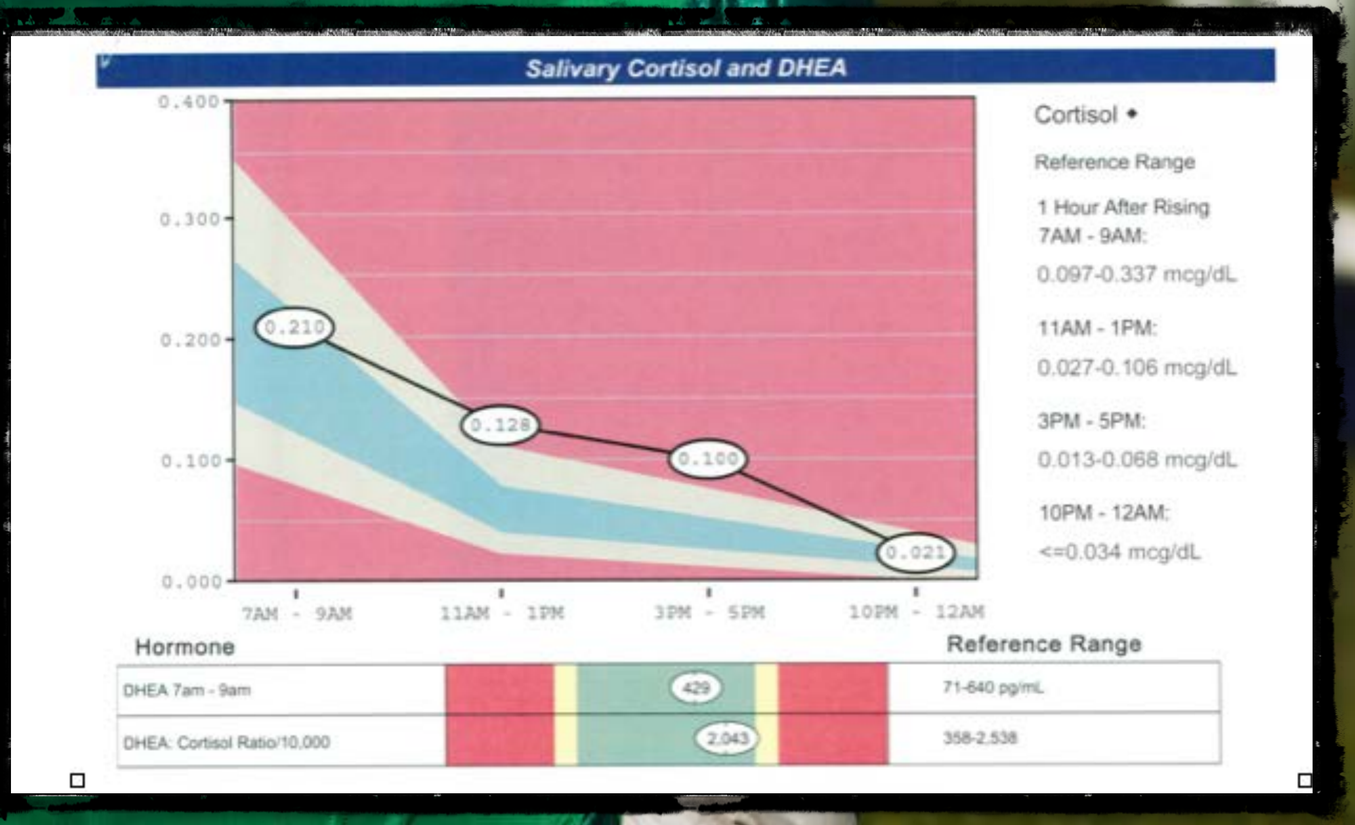
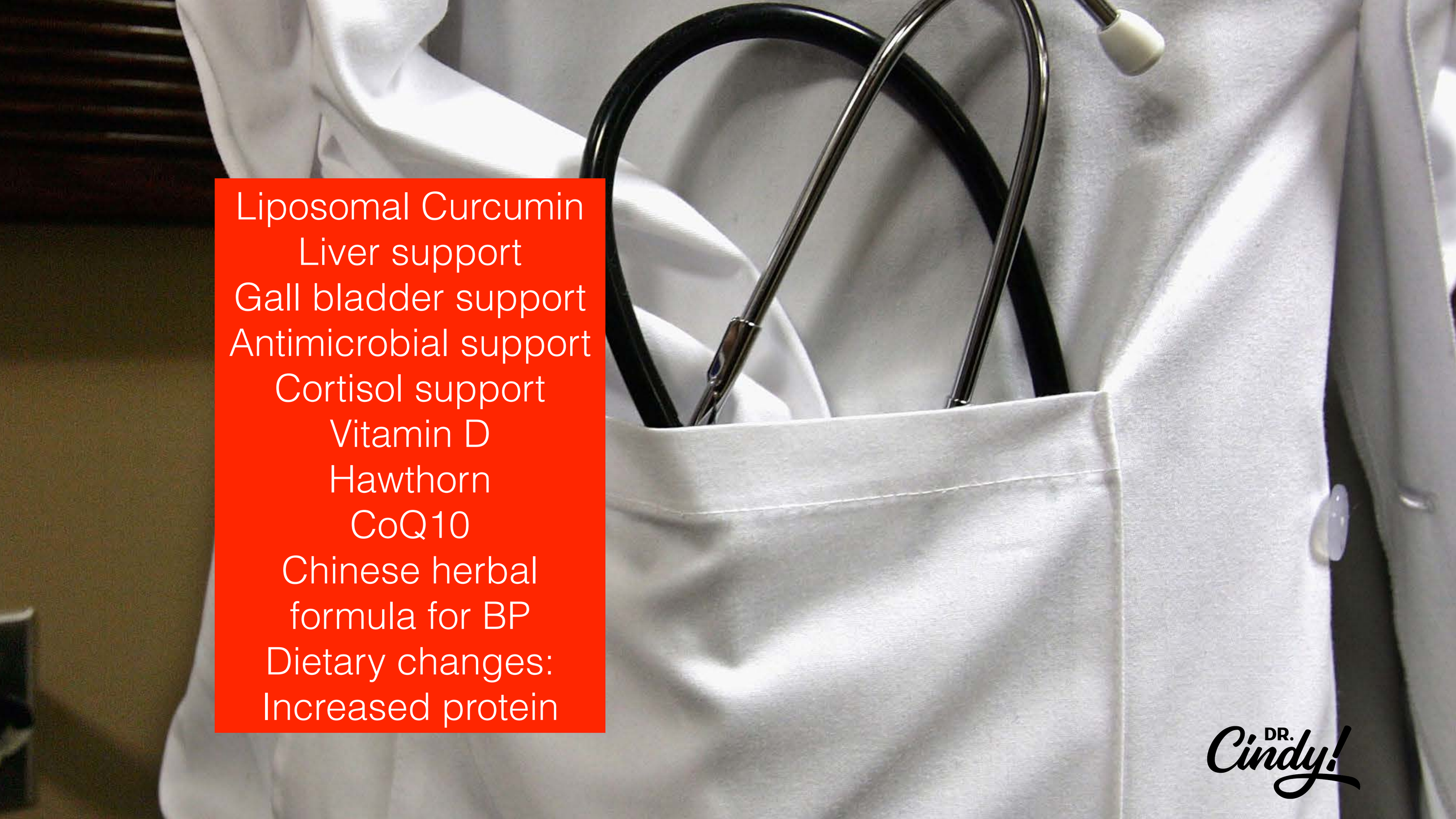
Mid day Cortisol typical for stress management, excessive exercise and alcohol consumption




Liposomal Curcumin
Liver support
Gall bladder support
Antimicrobial support
Cortisol support
Vitamin D
Hawthorn
CoQ10
Chinese herbal formula for BP
Dietary changes:
Increased protein
DR. Cindy!

Follow up blood test
6 weeks later

Improved Uric acid
Improved LDH
Improved Liver Enzymes
Lower Ferritin
Lower Monocytes
Lower CRP
Higher Vitamin D
Higher iron

Item Name	Client's #	Results	Functional Ranges	Laboratory Ranges
Glucose	88.00	NORMAL	85.00 - 99.00	65.00 - 99.00
Uric Acid	6.00	HIGH	3.20 - 5.50	2.50 - 7.10
BUN	12.00	LOW	13.00 - 18.00	6.00 - 20.00
Creatinine	1.08	LAB HIGH	0.65 - 0.90	0.57 - 1.00
BUN/Creat Ratio	10.00	NORMAL	10.00 - 20.00	8.00 - 20.00
Sodium	141.00	HIGH	135.00 - 140.00	134.00 - 144.00
Potassium	4.30	NORMAL	4.00 - 4.50	3.50 - 5.20
Chloride	101.00	NORMAL	100.00 - 106.00	97.00 - 108.00
Calcium	9.60	NORMAL	9.20 - 10.10	8.70 - 10.20
Magnesium	2.20	NORMAL	2.00 - 2.50	1.60 - 2.60
CO2	24.00	NORMAL	22.00 - 27.00	19.00 - 28.00
Protein	7.30	NORMAL	6.90 - 7.40	6.00 - 8.50
Albumin	4.60	NORMAL	4.00 - 5.00	3.50 - 5.50
Globulin	2.70	NORMAL	2.40 - 2.80	1.50 - 4.50
A/G Ratio	1.70	NORMAL	1.50 - 2.00	1.10 - 2.50
Alk Phos	104.00	HIGH	44.00 - 90.00	42.00 - 107.00
LDH	260.00	LAB HIGH	140.00 - 180.00	0.00 - 214.00
AST/SGOT	30.00	HIGH	10.00 - 26.00	0.00 - 40.00
ALT/SGPT	30.00	HIGH	10.00 - 26.00	0.00 - 32.00
Bilirubin	0.80	NORMAL	0.10 - 1.20	0.00 - 1.20
Serum Iron	80.00	LOW	85.00 - 135.00	35.00 - 155.00
TIBC	342.00	NORMAL	250.00 - 350.00	250.00 - 450.00
Ferritin	144.00	HIGH	13.00 - 122.00	13.00 - 150.00
RBC	5.38	LAB HIGH	3.90 - 4.50	3.77 - 5.28
HGB	15.00	HIGH	13.50 - 14.50	11.10 - 15.90
HCT	46.00	HIGH	37.00 - 44.00	34.00 - 46.60
MCV	86.00	NORMAL	85.00 - 92.00	79.00 - 97.00
MCH	28.00	NORMAL	27.70 - 32.00	26.60 - 33.00
MCHC	34.00	NORMAL	32.00 - 35.00	31.50 - 35.70
RDW	12.80	NORMAL	12.30 - 15.00	12.30 - 15.40
WBC	5.00	NORMAL	5.00 - 8.00	3.40 - 10.80
Neutrophils	51.00	NORMAL	40.00 - 60.00	> 0.00
Lymphocytes	25.00	NORMAL	25.00 - 40.00	> 0.00

Item Name	Client's #	Results	Functional Ranges	Laboratory Ranges
Monocytes	14.00	HIGH	4.00 - 7.00	> 0.00
Eosinophils	1.00	NORMAL	0.00 - 3.00	> 0.00
Basophils	1.00	NORMAL	0.00 - 3.00	> 0.00
Platelets	158.00	NORMAL	155.00 - 379.00	155.00 - 379.00
ESR	3.00	NORMAL	0.00 - 32.00	0.00 - 32.00
CRP	8.00	LAB HIGH	0.00 - 4.90	0.00 - 4.90
Triglycerides	135.00	HIGH	75.00 - 100.00	0.00 - 149.00
Cholesterol	160.00	NORMAL	150.00 - 199.00	100.00 - 199.00
LDL	98.00	NORMAL	0.00 - 99.00	0.00 - 99.00
HDL	36.00	LAB LOW	55.00 - 100.00	> 39.00
25 (OH) Vitamin D	68.00	NORMAL	35.00 - 100.00	30.00 - 100.00

DR. Cindy!

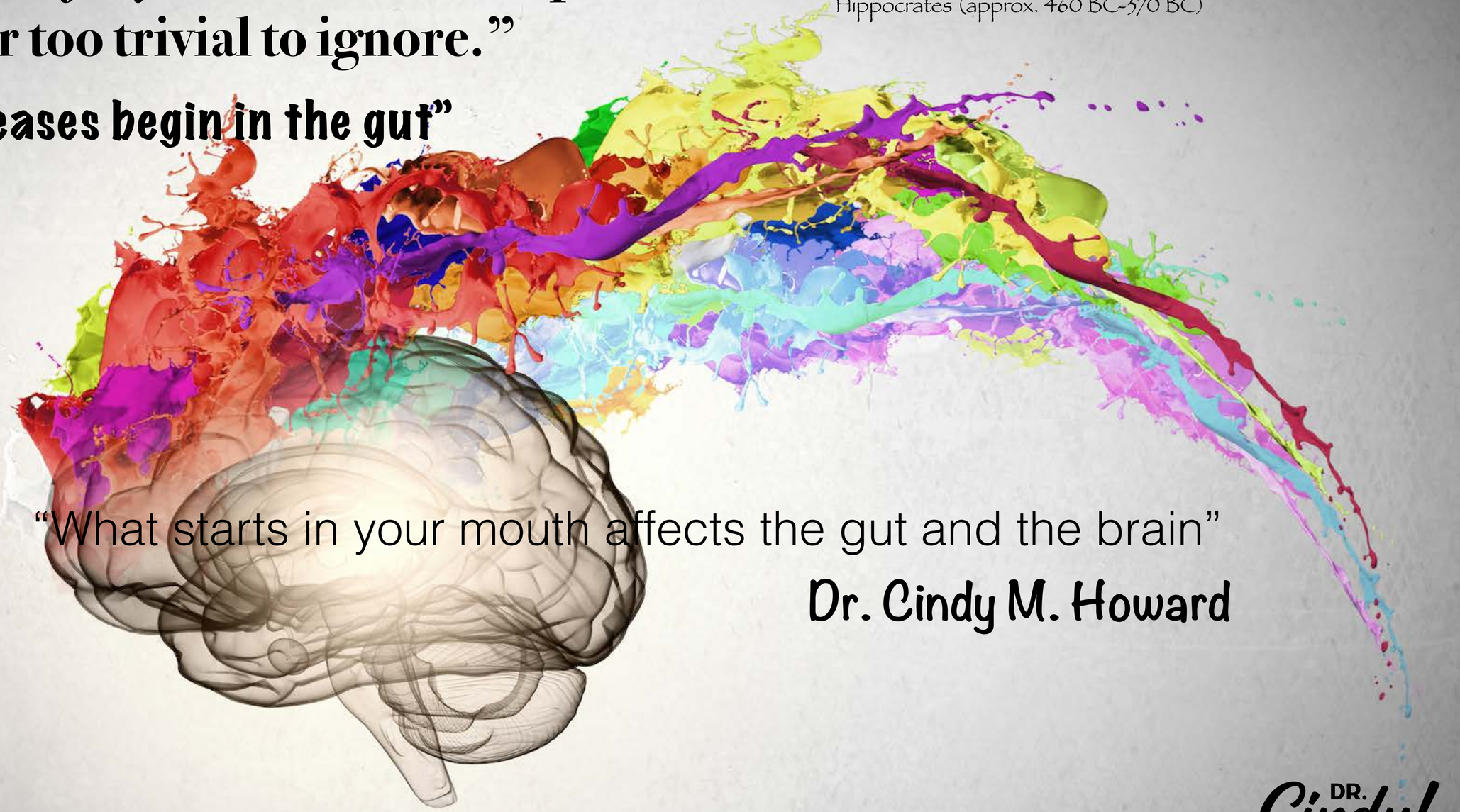
6 week follow up

Sleeping through the night
Anxiety is significantly reduced
Monocyte and CRP reduction
Elevation of Vitamin D and iron
BP 126/86

“No head injury is too severe to repair,
nor too trivial to ignore.”

Hippocrates (approx. 460 BC-370 BC)

“all diseases begin in the gut”



“What starts in your mouth affects the gut and the brain”

Dr. Cindy M. Howard

DR. Cindy!

My thanks to you: I have 3 gifts to share!

1. **A handy bonus guide!**
2. **My latest list of recommended books** on leadership, health, and humor.
3. **A warm welcome to my Daily Dose newsletter** to brighten your brain and inbox.

PLUS, by simply opening the survey, **I'll enter your name in a drawing** to win one of my favorite books!

Don't forget to find me at my Positively Altered Podcast on Apple and Spotify!

Text THANKS to 66866 to receive these gifts!

DR. Cindy!

DR. Cindy!

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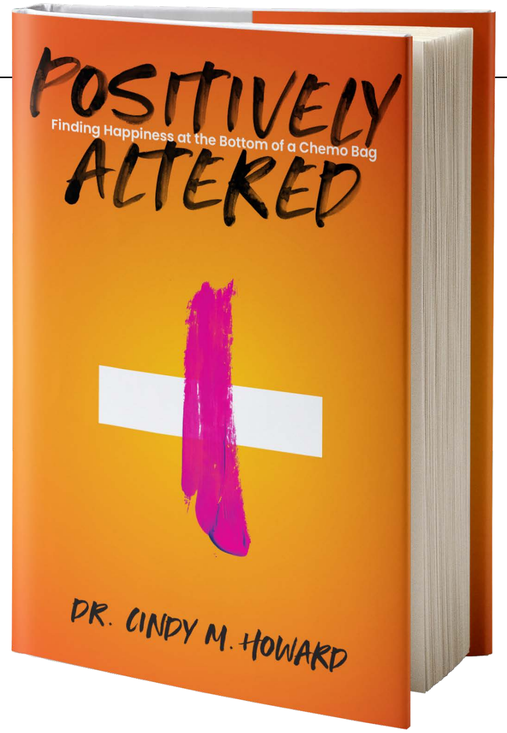

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