Head Injury and Nutrition

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NutriDyn

Northwestern Health
Human Performance Center

Disclosure

I am an employee of Northwestern Health Sciences University and a sports nutrition fellow that is being funded by Nutri Dyn.

Any discussion involving clinical nutrition practice involving supplementation may be perceived as a conflict of interest.

Objectives

- What is a head injury
- Neuroinflammation and what can go wrong
- Nutritional Treatment Options

Definitions

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.” - AANS

TBI - “a nondegenerative, noncongenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness.” - Medscape

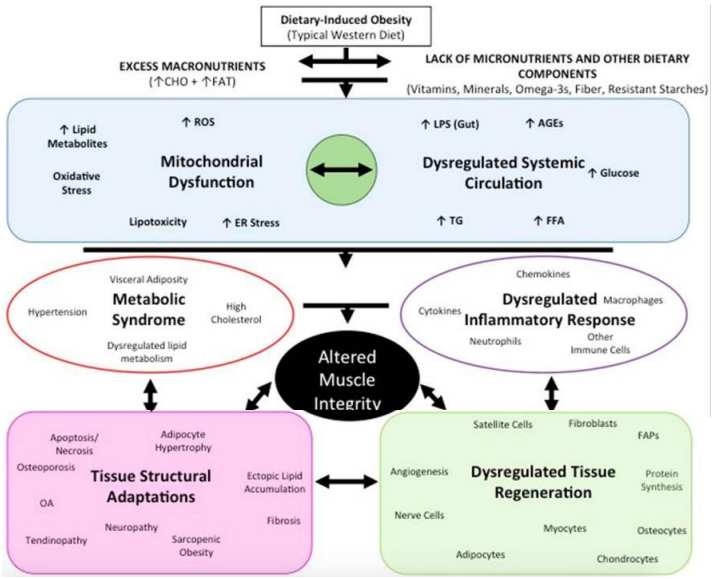
CTE - “a progressive degenerative disease of the brain found in people with a history of repetitive brain trauma (often athletes), including symptomatic concussions as well as asymptomatic subconcussive hits to the head that do not cause symptoms.” - BU

What is the injury?

TBI - Primary and Secondary Injury

"...our study shows that following TBI, secondary injury chiefly involves inflammatory processes and chemokine signaling, which comprise putative targets for pharmaceutical neuroprotection."

Israelsson, C., Bengtsson, H., Kylberg, A., Kullander, K., Lewén, A., Hillered, L., & Ebendal, T. (2008). Distinct cellular patterns of upregulated chemokine expression supporting a prominent inflammatory role in traumatic brain injury. *Journal of neurotrauma*, *25*(8), 959-974.



Collins, K. H. M. C., Herzog, W., MacDonald, G. Z., Reimer, R. A., Rios, J. L., Smith, I. C., ... & Hart, D. A. (2018). Obesity, Metabolic Syndrome, and Musculoskeletal Disease: Common Inflammatory Pathways Suggest a Central Role for Loss of Muscle Integrity. Frontiers in physiology, 9, 112.

*rat study

Hyperglycemia exacerbates Brain Swelling

TUNEL Assay used to identify and quantify apoptotic cells via detecting fragmented DNA segments.

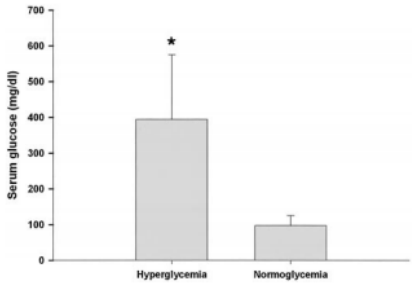


Figure 1. Mean serum glucose level after ICH induction. Values are mean±SD. *P<0.01 compared with normoglycemic group.

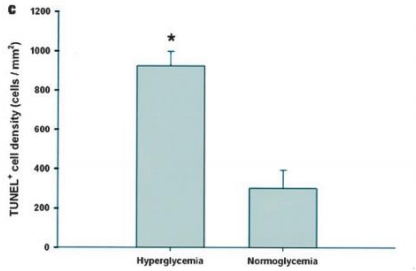


Figure 4. Results of TUNEL staining. a, b, Representative section in the perihematomal zone of the hemorrhagic lesion shows the presence of TUNEL-positive stained cells (a, hyperglycemic group; b, normoglycemic group). Counterstaining with toluidine blue indicates that polymorphonuclear leukocytes do not show TUNEL-positive staining. Note the more abundant TUNEL-positive cells in the hyperglycemic group (a). c, Density of TUNEL-positive stained cells per square millimeter. Values are mean±SD; n=6 per group. *P<0.01 compared with normoglycemic group.

Song, E. C., Chu, K., Jeong, S. W., Jung, K. H., Kim, S. H., Kim, M., & Yoon, B. W. (2003). Hyperglycemia exacerbates brain edema and perihematomal cell death after intracerebral hemorrhage. *Stroke*, *34*(9), 2215-2220.

“There’s nothing we can do but wait.”

Treatment

Food to Eat, Foods to Avoid

Eat		Avoid
Grass Fed Meat, Eggs		Bread, Pasta, Rice
Vegetables		Processed Sugar
Fruits		Milk
Nuts		Legumes (inc. peanuts)
Dark Chocolate (80% cocoa)		Processed Oils (Sunflower, Palm, Canola)
Herbs and Spices		Margarine
Whole Fat Cheese		Soy
Olive or Coconut Oil		
Full Fat Grass Fed Butter		
Almond, Coconut Milk		
Yogurt		
Red Wine		

Concussion and Supplements

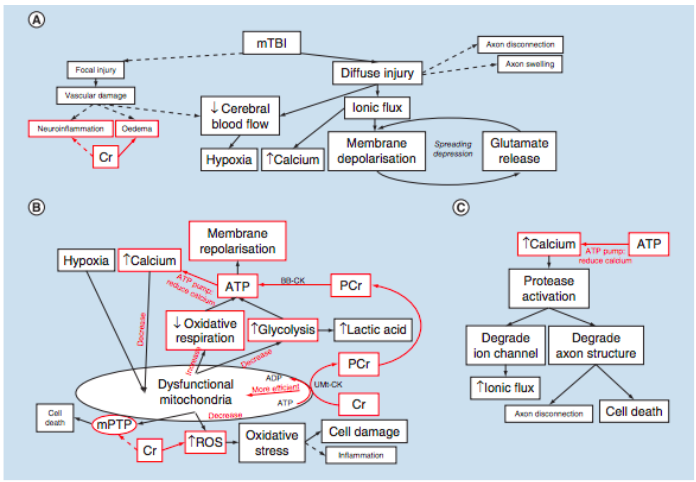
- Omega 3s, Curcumin, Resveratrol, Melatonin, Creatine, Vitamins C, D, E
- Review article of mostly rat studies
- Cost vs Benefit

Ashbaugh, A., & McGrew, C. (2016). The role of nutritional supplements in sports concussion treatment. *Current sports medicine reports*, *15*(1), 16-19.

Creatine and mTBI

Rat studies

- Neuroprotective qualities
- Anti-Inflammatory
- Creatine Kinase

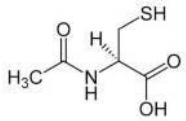


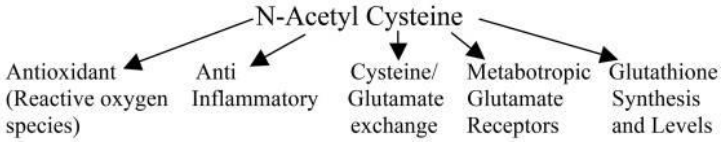
Ainsley Dean, P. J., Arikan, G., Opitz, B., & Sterr, A. (2017). Potential for use of creatine supplementation following mild traumatic brain injury. *Concussion*, *2*(2), CNC34.

*rat study

N- Acetyl Cysteine

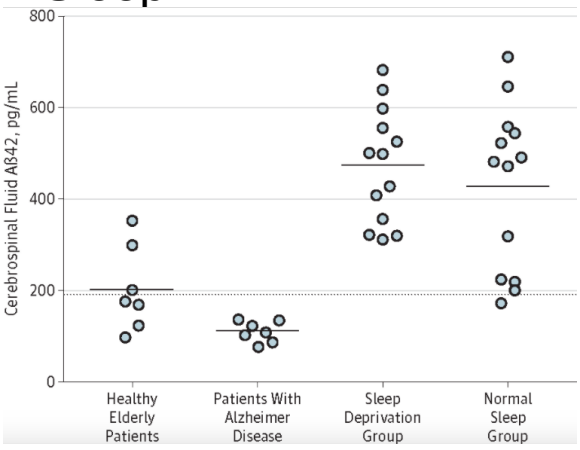
Mice administered NAC within 24 hours of head injury

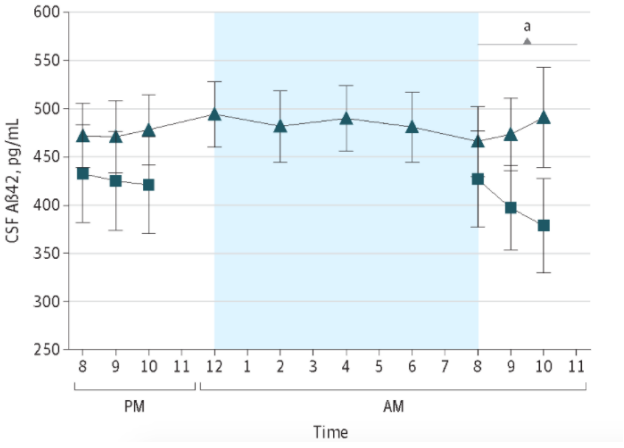




Eakin, K., Baratz-Goldstein, R., Pick, C. G., Zindel, O., Balaban, C. D., Hoffer, M. E., ... & Hoffer, B. J. (2014). Efficacy of N-acetyl cysteine in traumatic brain injury. *PLoS One*, *9*(4), e90617.

Sleep





Ooms, S., Overeem, S., Besse, K., Rikkert, M. O., Verbeek, M., & Claassen, J. A. (2014). Effect of 1 night of total sleep deprivation on cerebrospinal fluid β-amyloid 42 in healthy middle-aged men: a randomized clinical trial. *JAMA neurology*, *71*(8), 971-977.

Emotional Stress

Stress response

- Glial cell activation
- Increased WBC recruitment

Table 3 **Intracellular ROS accumulation in cerebral and peripheral cells of anxious and non-anxious mice**

	Mean fluorescent intensity (arbitrary units)	
Type of cells	**Anxious mice**	**Non-anxious mice**
Neurons of cerebral cortex	1223 ± 232**	177 ± 76
Neurons of cerebellum	546 ± 128*	227 ± 41
Neurons of hippocampus	1749 ± 566*	227 ± 51
Glial cells of cerebral cortex	429 ± 48	310 ± 72
Glial cells of cerebellum	343 ± 36*	195 ± 31
Glial cells of hippocampus	468 ± 43*	319 ± 29
Lymphocytes	251 ± 46*	152 ± 11
Monocytes	56 ± 9*	35 ± 2.5
Granulocytes	1979 ± 405**	448 ± 77

The mean fluorescent intensity corresponds to fluorescence resulting from intracellular DCFH-DA oxidation by intracellular ROS. Data are expressed as mean ± SEM. ($n = 10$). *$p < 0.05$; **$p < 0.01$. (data published by Rammal et al.[42] in Brain Behav Immun).

Rammal, H., Bouayed, J., Younos, C., & Soulimani, R. (2008). The impact of high anxiety level on the oxidative status of mouse peripheral blood lymphocytes, granulocytes and monocytes. *European journal of pharmacology*, *589*(1-3), 173-175.

Recommendations

Low Glycemic Index, Reduce Meat

Minimize Processed Foods

Multi Vitamin

Fish Oil - 1-3g

Vitamin D3 - 3,000-5,000 IU

Creatine - 5-10 g

Adequate Sleep

Stress Management

Hydration

No Alcohol

*patients are individuals

Ketogenic Diet for Head Injury?

Ketogenic Diet?

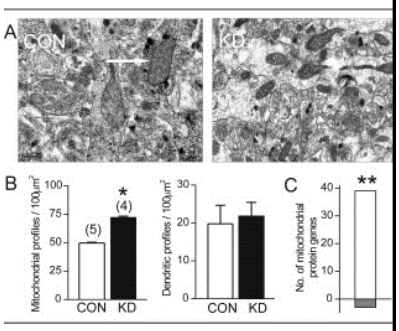
"It has long been recognized that the ketogenic diet is associated with increased circulating levels of ketone bodies, which represent a more efficient fuel in the brain, and there may also be increased numbers of brain mitochondria. It is plausible that the enhanced energy production capacity resulting from these effects would confer neurons with greater ability to resist metabolic challenges."

Maalouf, M., Rho, J. M., & Mattson, M. P. (2009). The neuroprotective properties of calorie restriction, the ketogenic diet, and ketone bodies. *Brain research reviews*, *59*(2), 293-315.

*rat study

Mitochondria on Ketogenic Diet

- 3 weeks KD
- 46% increase in the density of mitochondria
- Suggestive of mitochondrial biogenesis increasing ATP production capacity, with excess high-energy phosphates stored as phosphocreatine



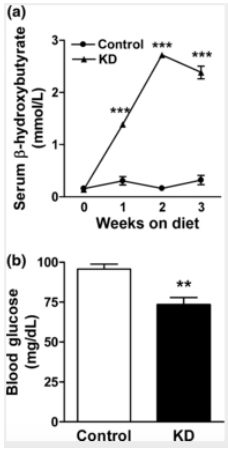
Bough, K. J., Wetherington, J., Hassel, B., Pare, J. F., Gawryluk, J. W., Greene, J. G., ... & Dingledine, R. J. (2006). Mitochondrial biogenesis in the anticonvulsant mechanism of the ketogenic diet. Annals of neurology, 60(2), 223-235.

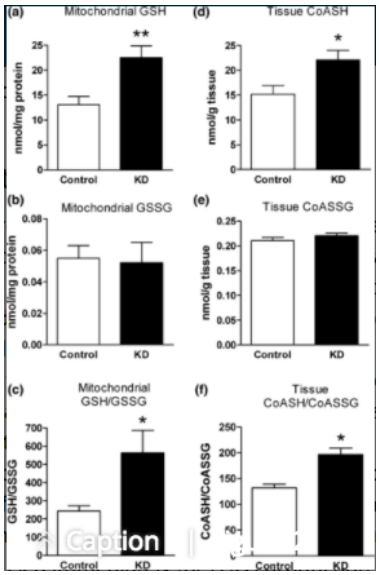
Ketogenic Diet?

- Acetoacetate also decreased the formation of early cellular markers of glutamate-induced apoptosis and necrosis
- KD has increased Glutamate Peroxidase to reduce formation of ROS in rat studies
- Reduction in NF-kB, IL-1, IL-6

Gasior, M., Rogawski, M. A., & Hartman, A. L. (2006). Neuroprotective and disease-modifying effects of the ketogenic diet. *Behavioural pharmacology*, *17*(5-6), 431.

KD and Glutathione Peroxidase





Jarrett, S. G., Milder, J. B., Liang, L. P., & Patel, M. (2008). The ketogenic diet increases mitochondrial glutathione levels. *Journal of neurochemistry*, *106*(3), 1044-1051.

*rat study

Ketogenic Diet

- Between 36-58% contusion reduction at 7 days following controlled cortical trauma
- Age dependant response

Prins, M. L., Fujima, L. S., & Hovda, D. A. (2005). Age-dependent reduction of cortical contusion volume by ketones after traumatic brain injury. *Journal of neuroscience research*, *82*(3), 413-420.

KD Research

Clinical trials on ketogenic diet hampered by:

- Poor compliance by humans
- Potential (mild) side effects
- Keto-Adaptation vs. Short Ketogenic Diet

Are they a KD candidate?

Summary

1. Include questions on diet and lifestyle to address inflammatory process
2. Take pro-inflammatory foods out, add anti-inflammatory foods in
3. Prescribe supplements when necessary
4. Determine if a ketogenic diet may be right for them

Questions/Comments?

a apple a day keeps the doctor away