

PERFORMANCE BRAIN HEALTH CENTERS

COLORADO SPRINGS, COLORADO

TREATMENT OF CONCUSSIONS, BRAIN INJURIES, AND MENTAL ILLNESS

INTRODUCTION

Brain function remains a great mystery to all people, despite the intense focus on it over the last several decades. Research on the brain, how it functions, and treating its disorders has exploded over the past several decades, and yet problems and disorders of the brain are still not well understood. For example, an academic database search (EBSCOhost) reveals that there have been 157,508 scholarly and research articles on mental illness, with 97,370 occurring since 2009 and 138,555 research articles specifically focused on concussions, with 107,241 occurring since 2009. To put the exploding interest in concussions (and traumatic brain injuries) and mental illness in perspective, 62% of all research articles published on mental illness and 77.4% of all research articles on concussion have been published since 2009. Despite this research focus on mental health and brain injuries both are often misdiagnosed and even more frequently inadequately treated.

BRAIN FUNCTION OVERVIEW

In order to effectively treat concussions, brain injuries, and mental illness (CBIMI) there must be an accurate understanding of the brain and how it functions. There are thousands of research articles on CBIMI, many of which are devoted to animal models of brain injury or mental illness, neurotransmitter function and its relationship to CBIMI, oxygen to the brain, blood flow to and within the brain, genetics and its role in CBIMI, trauma and how it affects CBIMI, relational factors contributing to CBIMI, etc. Interestingly, despite the hundreds of thousands of scholarly, research articles on the topic there has been no definitive answer or scientific agreement on the cause of depression, the reason why mood-stabilizing medications reduce manic symptoms in Bipolar Disorder, or curative treatment for concussions, traumatic brain injuries, or post-concussion syndrome.

The basis for effectively treating CBIMI is a fundamental and correct understanding of brain function. The brain must be adequately fueled if it is to function appropriately (Glenn, T.C., Martin, N.A., Horning, M.A., Hovda, D, 2015; Cunnane, S.C., Corchesne-Loyer, A., Vandenberghe, C., 2016) . There has been renewed interest in ketones as a source of brain fuel, per the articles cited in the previous sentence as well as other research, but ketones and the ketogenic system are a secondary fuel source at best and are activated in the case of crisis and starvation (Fann, S. 2019). As will be explained later in this paper, glucose is the primary fuel source for the brain.

All areas of brain function that were previously identified as the subjects of extensive research (neurotransmitter function, oxygen supply to the brain, blood supply to the brain, and others) are important and necessary for proper brain function but cannot function properly without adequate fueling. Consider the example of how a car functions. The transmission and drive shaft may work fine, but if the fuel filter is clogged the engine will not run smoothly and the car will not move as it should.

This does not mean that there are mechanical problems with those parts but rather the lack of consistent fuel makes them not operate as intended. The brain works in the same manner. When it is fully fueled all the “mechanical” parts and systems function appropriately; when it is not receiving adequate fuel there are “mechanical” problems with the neurotransmitters, brain structures, and basic human functions (memory, attention, emotional regulation, behavioral control, etc.). Unfortunately, researchers are incorrectly assuming that there is a “mechanical” problem in the brain and so are looking for and finding ways in which the “mechanics” (neurotransmitters and brain structures, among other things) are not functioning as they should and inaccurately identifying those things as the brain’s problem and source of functional deficits (Patterson & Holahan, 2012). An accurate understanding of brain function leads to identifying the source of the problem, which is inadequate or inconsistent fueling.

CAUSE OF BRAIN_DYSFUNCTION

Trauma is the factor that is responsible for inadequate or incomplete fueling of the brain and corresponding brain dysfunction. Trauma for the purposes of this paper and brain function is defined as any event or experience that activates the sympathetic nervous system, the fight/flight/freeze response, and corresponding brain fuel limitation. Trauma occurs primarily in the form of concussive injuries, traumatic brain injuries, experiences of fear, and psychological injuries in the form of neglect, emotional abuse, physical abuse and sexual abuse.

When the sympathetic nervous system is activated by concussion, traumatic brain injury, fear experiences, or psychological injuries extra energy or fuel is required by the brain to ensure the person survives (Flak, J.N., Arble, D., Pan, W., et al., 2017). The brain creates a chemical cascade when it is activated by fear, danger, or injury, which results in it receiving extra fuel from the body. The infusion of extra fuel contributes to the development of hyperglycolysis, which is not a normal brain process and can be harmful and dangerous to the brain if it persists (Giza et al., 2001; Jalon et al., 2015; Hovda et al., 2011). As a result, the brain begins limiting the amount of fuel it requires from the body and does so permanently with each physical or psychological injury, experience of fear or danger, and activation of the sympathetic nervous system (Glenn, T.C., Martin, N.A., Horning, M.A., Hovda, D, 2015). This fuel limitation is “permanent” (until the fuel dysfunction is eliminated through treatment, which will be explained in detail below), because the brain actively works to eliminate the risk of a re-occurrence of hyperglycolysis (Zetterberg, Smith, Blennow, 2015).

The fuel limitation caused by activation of the sympathetic nervous system due to the injuries noted above is not only “permanent,” but is cumulative (Daneshvar, D.H., Riley, D.O., Nowinski, C.J., McKee, A.C., et al, 2011; Guerriero, R.M., Giza, C.C., & Rotenberg, A., 2015). Each sympathetic nervous system activation and injury results in hyperglycolysis, which the brain interprets as not only dangerous but as representing increased risk of additional episodes of hyperglycolysis, resulting in the need for additional limitations on fuel (Zetterberg, Smith, Blennow, 2015). When each subsequent trauma occurs, the brain responds with additional limitations on the amount of fuel it receives in an effort to reduce future episodes of hyperglycolysis and prevent serious damage and even death.

Fuel limitation results in reduction of efficiency or effectiveness of all brain functions. This reduction in efficiency and effectiveness is experienced as symptoms of brain disorders and mental illness. The abilities that are most commonly thought of as the primary domains of brain function such as memory, attention and concentration, language skills, reading, math, and information processing are reduced or limited due to inadequate fuel as described above. The abilities and functions that are thought of as

being the primary domain of psychology such as emotional regulation, interpersonal relatedness, and behavioral control are also reduced or limited due to inadequate fuel. The symptoms of mental illness, such as anxiety, depression, lack of anger control, excessive inhibition in social situations, aggressive or violent behavior, and perhaps even mania and psychotic symptoms are due to the brain's inability to regulate these behaviors and experiences appropriately because there is not enough fuel to do so.

Current research is also pointing towards the role of inadequate brain fuel in what have been considered primarily or exclusively physical symptoms, such as the muscle weakness in multiple sclerosis, the seizures of epileptic disorders, fainting, and even the symptoms of auto-immune disorders. This research is quite recent and should be considered tentative.

BRAIN FUEL

The brain is fueled by a number of chemicals and substances, but its primary fuel is glucose, which is also a primary fuel for the rest of the body. Cellular respiration (or the process of creating energy for the body) cannot occur without glucose. It's importance in the body is further illustrated by the fact that there are two specific substances in the body whose entire function is to regulate the availability of glucose to the body and the brain, glycogen and glucagon (Chobot, A., Otto-Buczkowski, E. 2011).

The process of brain fueling begins with the body converting a portion of everything that is eaten to glucose. Glucose is used to fuel the body and the brain and any "extra" that is not used for those two purposes is converted to glycogen and stored in the liver and elsewhere throughout the body. The liver releases glucose as needed via glucagon, which converts glycogen back to glucose and then can be used by the body for fuel and energy (Chobot, A., Otto-Buczkowski, E. 2011) .

The brain consumes approximately 20% to 40% of all glucose created by the body via the above mechanism (Mergentheler, P., Lindauer, U., Dienel, G., et al., 2013; Harvard Mahoney Neuroscience Institute, 2019). It consumes more glucose than any other organ or process in the body. The brain allocates glucose first to the brain stem, which is responsible for the heart pumping, respiration, and other survival-necessary functions. Following fueling of the brain stem, then glucose is allocated geographically and functionally through the sensory and motor functions and organs up to the outer-most layer of the brain, the cerebral cortex. The cerebral cortex is primarily responsible for higher-level brain functions such as thinking, memory, language, vision, and other domains of human expertise.

As was described above trauma results in the process of hyperglycolysis, which results in the brain limiting the amount of glucose it allows to come in to it from the body. The limitation of glucose is the limitation of brain fuel, resulting in the symptoms of CBIMI (Burns, C.M., 2015). In most cases, the symptoms following a first trauma (brain or psychological) are minimal and transient, so much so that first traumas (unless they are severe enough to result in loss of consciousness) are undetected. The explanation for this is that each traumatic event results in a relatively minor reduction in glucose provision to the brain, and the brain can adapt and adjust to the minor limitation in glucose availability. Due to the cumulative nature of these injuries, and the successive decreases in glucose availability to the brain following each injury, symptoms increase in frequency and severity as the number of experienced traumatic events multiply.

At the time of first trauma there may be brief, mild headaches, tiredness, transient blurry vision, or other similarly mild symptoms. These symptoms usually continue for a period of hours and then seem

to completely disappear. In reality, the symptoms continue but are misinterpreted by the brain as “normal” or “sinus” headaches, expected tiredness or fatigue due to stress, “laziness,” “normal teenage moodiness”, or a variety of other “normal” conditions. The brain utilizes a variety of methods (including the symptoms noted above) to signal a reduction or deficit in fuel, but until now those methods have been misinterpreted and not associated with glucose (or fuel) deficits at all.

Typically, as the traumatic injuries accumulate, symptoms associated with the functions of the cerebral cortex (memory, language or word-finding problems, attention/concentration, etc.) and symptoms related to the pain and pleasure centers of the brain (headaches, painful sensory experiences, etc.) are most common and become more “permanent” (Maruta, J., Spielman, L.A., Yarusi, B.B., et al., 2016). Sensory changes, which are frequently connected to the organs and systems in the geographically mid-level of the brain, occur more frequently in the senses of vision (light sensitivity) and hearing (sound sensitivity). In more severe injuries, or after experiencing a higher number of injuries, the senses of taste and smell begin to be affected. The last symptoms to appear are impairments in motor function, systemic deterioration, and finally in cases of repeated or severe injury, death. The symptoms of trauma and the corresponding glucose limitation are “permanent,” until treated appropriately (Zetterberg, Smith, Blennow, 2015).

TREATMENT OF CBIMI

The first and most important treatment for CBIMI is taking over-the-counter glucose tablets, gummies, or dextrose powder mixed in liquid. Glucose taken in these forms by-passes the brain’s glucose-limiting mechanism, which occurs following trauma as described above. In this way, the brain is fueled directly and responds very quickly to receiving fuel.

The brain must be fully fueled with over-the-counter glucose throughout the day. This typically requires 3-4 doses of glucose spread evenly over the course of the day. The principle is as simple as the need to re-charge the battery on your cell phone. When the battery level drops below 5 or 10%, in most phones, the screen is darkened and the phone will not function as designed. When the glucose level in the brain drops below a certain level due to using it up through activity or stress brain function diminishes, symptoms like headache, dizziness, and brain fog emerge, and there are often sensory difficulties such as tinnitus, light sensitivity, and sound sensitivity.

During the several months of treatment, as the brain remains adequately or fully fueled, the brain begins to decrease the limiting factor and allows more of the glucose that is naturally produced by the body from food to enter it. This results in the ability to reduce the amount of glucose taken with each dose, because now the brain is receiving a higher percentage of the glucose created by the body.

Glucose treatment of CBIMI typically requires 3-6 months of taking the glucose 3-4 times per day. After this amount of time the brain chemistry re-sets, and the limiting factor is no longer occurring. As such, glucose treatment can be discontinued in approximately 3-6 months, with no return of symptoms following discontinuation. All cognitive symptoms (thinking difficulty, memory difficulty, word-finding difficulty, language difficulty, information processing difficulty, attention/concentration difficulty, etc.), psychological symptoms (anxiety, depression, anger management difficulty, mania, psychosis, emotional numbness, anhedonia, low motivation, poor self-esteem and inaccurate self-perception, feelings of worthlessness, etc.), behavioral symptoms (“laziness,” aggression, violence, isolation/withdrawal, etc.), and many physical symptoms (headache, migraine headache, tinnitus, light sensitivity, sound sensitivity,

balance difficulty, many pains, limited mobility, tremor, seizure, etc.), (Wallace et al., 2016) are eliminated following the 3-6 months of glucose treatment.

In addition to the glucose treatment the person with CBIMI must participate in a modified form of cognitive-behavioral therapy (CBT). The brain misinterprets what has happened with the experience of trauma and there are resulting thought patterns that need to be modified. It is common for the brain to cycle through a variety of misinterpretations as it is recovering, each of which must be addressed and changed to accurate thought patterns. When CBT is attempted without adequate fueling it can be successful, but it takes much more time, is much more difficult, and can lead to subtle maladaptations in other areas of brain function (such as worsened memory, development or exacerbation of headaches, etc.).

Finally, after the brain has been adequately fueled with glucose for a period of a few weeks to a few months, training of peripheral physical systems, such as in visual information processing and balance, frequently must be provided (Bigelow, R.T., Semenov, Y.R., duLac, S. 2016). Many clinicians attempt to provide vision training immediately after the trauma, which leads to compensations that are not helpful and can increase or exacerbate headaches. Vision training in most cases consists of several sessions of learning the exercises and then completing the exercises at home for a few minutes each day.

SUMMARY

A vast body of research is accumulating on concussions, brain injuries, and mental illness (CBIMI). Unfortunately, this research is focusing more on the “mechanics” of brain function than on the foundation of brain function, which is the fueling (or lack of fueling) of the brain.

Traumatic experiences (either through head injuries or psychological trauma) are defined as anything that results in the activation of the sympathetic nervous system and subsequent limiting of glucose (or fuel) to the brain. This glucose limitation is “permanent” and increases following every traumatic experience that is sufficient to activate the sympathetic nervous system. Every traumatic incident is cumulative in its effect on limiting brain fueling or glucose delivery to the brain.

Treatment with a combination of over-the-counter glucose that is taken orally, in combination with a modification of cognitive-behavioral therapy and brief peripheral physical system training is effective in eliminating the effects of CBIMI.

SEE APPENDIX BELOW FOR CASE HISTORIES OF PATIENTS TREATED WITH THE CBIMI TREATMENT.

APPENDIX

CASE HISTORIES

Case History 1

Diagnosis: Post-Concussion Syndrome; Chronic Traumatic Encephalopathy

68 year-old male. History of 20+ concussions and/or traumatic brain injuries. Neuropsychological test scores all at or below the 1st percentile at time of first appointment. Headache, head pressure, balance, dizziness, anger, anxiety, depression all at 90th percentile or higher at time of first appointment. Began with 20 grams of glucose on initial appointment. This dose reduced all symptoms to below the 50th percentile during initial appointment. Recommended increasing dose that day to 50 grams, 3 times per day. That dose reduced symptoms to 20th percentile after 1 week. Increased dose to 100 grams 4 times per day. That dose eliminated all headaches, head pressure, with return of ability to “multi-task, no longer needing to hold on to the walls in the shower, and no more symptoms.” Neuropsychological test scores all at or above the 70th percentile at conclusion of treatment. Headache, head pressure, balance, dizziness, anger, anxiety, and depression all resolved at conclusion of treatment. Glucose discontinued.

Case History 2

Diagnosis: Post-Concussion Syndrome

17 year-old female. History of 3 identified concussions; most recent concussion 12 months prior to first appointment. Cumulative high school GPA: 4.3. Currently failing classes. Neuropsychological test scores ranged from 27th percentile to 84th percentile. Headache, tinnitus, light sensitivity, anxiety, depression ranged from 50th to 90th percentile at time of first appointment. Began with 12 grams of glucose on initial appointment and all symptoms reduced to below the 20th percentile by the end of the initial appointment. Did not take glucose/dextrose for the 3 days until next appointment. At that appointment anxiety in the 70th percentile and depression in the 90th percentile; headache, tinnitus, light sensitivity all above the 50th percentile. Increased glucose/dextrose dose to 28 grams at time of appointment and all symptoms disappeared. Continued taking glucose/dextrose for 3 months at 28 grams 3 times per day. All symptoms continued to be resolved with no depression, anxiety, headache, tinnitus, or light sensitivity. Neuropsychological test scores ranged from 75th percentile to 93rd percentile at conclusion of treatment. Glucose discontinued at conclusion of treatment with no return of symptoms.

Case History 3

Diagnosis: Concussion without Loss of Consciousness

17 year-old male. Treatment after concussion with a history of 1 prior concussion. Neuropsychological test scores ranged from 4th percentile to the 41st percentile. Symptoms included headache, light sensitivity, tinnitus, difficulty paying attention at 80th percentile at time of initial appointment. Began with 16 grams of glucose at initial appointment that eliminated all symptoms by the end of the initial appointment. Recommended to take 16 grams 3 times per day until next appointment. At time of second appointment headache, head pressure, tinnitus, remained at 20th to 30th percentile. Glucose dose recommended to increase to amount needed to eliminate all symptoms. Achieved elimination of symptoms at 80 grams of glucose per dose, 4 times per day. Concluded treatment after 3 months and glucose discontinued. All symptoms resolved. Neuropsychological test scores ranged from the 55th to the 77th percentile.

Case History 4

Diagnosis: Major Depressive Disorder, PTSD

64 year-old male. History of depressive episodes throughout his lifetime. Depression rated at 60th percentile at time of initial appointment. History of rocket and gun attacks in Afghanistan during State Department missions. Anxiety, sleep disruption at 50th percentile at time of initial appointment. Began with 20 grams of glucose at initial appointment. Depression and anxiety reduced to 10th percentile by the end of appointment. Recommended 40 grams of glucose 4 times per day. Continued at that dose with no further symptoms of depression or anxiety for 4 months. Depression, anxiety, sleep problems eliminated at conclusion of treatment. Glucose discontinued at that time.

Case History 5

Diagnosis: Post-Concussion Syndrome, Suspected Chronic Traumatic Encephalopathy

41 year-old male. History of “dozens of concussions.” Neuropsychological test scores ranged from the 1st percentile to the 25th percentile. Depression, anxiety, tinnitus, head pressure, memory problems, sleep difficulty, and anger all rated at the 80th percentile or higher. Began with 40 grams of glucose at initial appointment, which reduced all symptoms down to the 20th percentile or lower by the conclusion of the initial appointment. Recommended dose of 100 grams of glucose 4 times per day by the third appointment. This dose maintained symptoms at or below the 20th percentile. Dose increased to 160 grams 4 times per day by the fourth appointment, which eliminated all symptoms. Treatment

concluded after 5 months with no return of symptoms. Glucose discontinued apart from occasional 40 to 50 gram one-time administration during times of high stress. Neuropsychological test scores ranged from 50th to 78th percentile at time of discharge from treatment.

Case History 6

Diagnosis: Concussion with LOC, Suspected Chronic Traumatic Encephalopathy

63 year-old female. Immediate reason for referral was motor vehicle accident with concussion with loss of consciousness. Neuropsychological test scores at the time of initial appointment were artificially elevated due to her familiarity with neuropsychological testing as a result of her career as a rehabilitation/occupational therapy specialist and having given neuropsychological screening instruments to many of her patients. Her neuropsychological test scores ranged from the 16th percentile (despite specific familiarity with this task) to the 92nd percentile. Depression, anxiety, headache, head pressure, mental “fogginess”, memory problems, “word-finding” difficulty, anger, balance problems all rated at the 80th percentile or higher at the time of initial appointment. Began with 60 grams of glucose at initial appointment, which reduced all symptoms to 30th percentile or lower during initial appointment. Recommended dose of 100 grams 4 times per day at conclusion of initial appointment. At second appointment she reported that this dose reduced symptoms to the 30th or 40th percentile or lower. Recommended dose increase to 160 grams 4 times per day. At third appointment she reported this dose reduced symptoms consistently to 20th percentile or lower, with some intermittent higher intensity of symptoms. By 5th appointment she was taking between 200 and 240 grams 4 times per day, which eliminated all symptoms. She continued on this dose for 5 months, at which time all symptoms had been eliminated for a month and she discontinued use of glucose apart from occasional doses of 20 to 40 grams during times of high stress. Neuropsychological test scores at the time of completion of treatment ranged from the 84th to the 97th percentile.

Case History 7

Diagnosis: Post-Concussion Syndrome; Chronic Traumatic Encephalopathy

39 year-old male. History of “more than 50 fights with light flashes or dark spots in vision;” motor vehicle accident at 100-120 mph, which resulted in 30-day medically induced coma; incarceration in Department of Corrections for 3 years with multiple more fights resulting in concussion. Neuropsychological test scores at initial appointment ranged from .1 of the 1st percentile to the 27th percentile. Headache, head pressure, tinnitus, mental “fogginess,” anger, anxiety, depression, “word

finding difficulty," memory, dizziness, balance problems all rated at the 90th percentile or higher at the initial appointment. Began with 160 grams of glucose, which eliminated all symptoms by the conclusion of the initial appointment. Has been taking 160-240 grams of glucose 4 times per day, with no return of symptoms, for three weeks. Treatment is continuing.

BIBLIOGRAPHY

Barkhoudarian G, Hovda D, Giza C. The Molecular Pathophysiology of Concussive Brain Injury. 2011.

Bergersen L. Lactate transport and signaling in the brain: potential therapeutic targets and roles in body-brain interaction. *Journal of Cerebral Blood Flow and Metabolism*. 2015; 35, 176-185.

Bigelow, R.T., Semenov, Y.R., du Lac, S., et al. Vestibular vertigo and comorbid cognitive and psychiatric impairment: the 2008 National Health Interview Survey. *Journal of Neurology, Neurosurgery, and Psychiatry*. 2016; 87(4), p. 367-372.

Burns CM. The relationship between fasting serum glucose, brain metabolism and neuropsychological functioning in older and younger adults. *Dissertation Abstracts International: Section B: The Sciences and Engineering*. 2015;75(10-B(E)).

Chobot A, Otto-Buczkowska E. Glucose homeostasis from foetus through childhood. *Experimental & Clinical Diabetology / Diabetologia Doswiadczalna i Kliniczna*. 2011;11(1):29-38.

<http://search.ebscohost.com.proxy->

Collins-Praino, L.E., Corrigan, F. Does neuroinflammation drive the relationship between tau hyperphosphorylation and dementia following traumatic brain injury? *Brain, Behavior, and Immunity*. 2017; 60, p. 369-382.

Cunnane SC, Courchesne-Loyer A, Vandenberghe C, et al. Can Ketones Help Rescue Brain Fuel Supply in Later Life? Implications for Cognitive Health during Aging and the Treatment of Alzheimer's Disease. *Frontiers In Molecular Neuroscience*. 2016;9:53.

Fann, S. The fat fueled brain: Unnatural or advantageous. *Scientific American*. 2019

Flak JN, Arble D, Pan W, et al. A leptin-regulated circuit controls glucose mobilization during noxious stimuli. *The Journal Of Clinical Investigation*. 2017;127(8):3103-3113. doi:10.1172/JCI90147.

Giza Christopher, Hovda David. The Neurometabolic Cascade of Concussion. *Journal of Athletic Training* 2001; 36(3) 228-235.

Giza, C.C. Lasting effects of traumatic brain injury. *Indian Journal of Neurotrauma*. 2006; 3(1). P. 19-26.

Glenn TC, Martin NA, Horning MA, et al. Lactate: Brain fuel in human traumatic brain injury: A comparison with normal healthy control subjects. *Journal of Neurotrauma*. 2015;32(11):820-832. doi:10.1089/neu.2014.3483.

Guerriero, R.M., Giza, C.C., & Rotenberg, A. Glutamate and GABA imbalance following traumatic brain injury. *Current Neurology & Neuroscience*. 2015. 15 (5), p. 27.

Harmon, K. Brain injury rate seven times greater among U.S. prisoners. *Scientific American*. 2012.

Henry, L.C., Tremblay, S., De Beaumont, L. Long-term effects of sports concussions: bridging the neurocognitive repercussions of the injury with the newest neuroimaging data. *The Neuroscientist*. 2017; 23(5), p. 567-578.

Jalloh, I., Carpenter, K.L., Helmy, A., et al. Glucose metabolism following human traumatic brain injury: Methods of assessment and pathophysiological findings. *Metabolic Brain Disease*. 2014 30, p. 615-632.

Jalloh I, Helmy A, Shannon R, Gallagher C, Menon D, Carpenter K, Hutchinson P. Lactate Uptake by the Injured Human Brain: Evidence from an Arteriovenous Gradient and Cerebral Microdialysis Study. *Journal of Neurotrauma*. 2013; 30:2031-2037.

Harvard Mahoney Neuroscience Institute, 2019

Maruta, J., Spielman, L.A., Yarusi, B.B., et al. Chronic post-concussion neurocognitive deficits: Relationship with persistent symptoms. *Frontiers in Human Neuroscience*. 2016; 45(10), p. 45.

Mergenthaler P, Lindauer U, Dienel GA, Meisel A. Sugar for the brain: the role of glucose in physiological and pathological brain function. *Trends Neurosci*. 2013;36(10):587–597. doi:10.1016/j.tins.2013.07.001

Patterson, Z.R. & Holahan, M.R. Understanding the neuroinflammatory response following concussion to develop treatment strategies. *Frontiers in Cell Neuroscience*. 2012; 58 (6), p. 58.

Zavaglia, M. Causal functional contributions and interactions in the attention network of the brain: an objective multi-perturbation analysis. *Brain Structure and Function*. 2016; 221(5), p. 2553-2568.

Zetterberg H, Smith D, Blennow K. (2013). Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood. *Nat Rev Neurol*. 2013 April; 9(4): 201-210.