ALBION MINERALS
Human Nutrition

RESEARCH NOTES

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Minerals Critical for Cognitive Function

The interest in formulating products to enhance cognitive function has been increasing in recent years, and the statistics indicate that this interest will continue to grow for a variety of reasons. One of the largest reasons rests on the fact that the age group of people in the US over the age of 55 is growing, and with the lifespan extension rising, this segment of society will get larger.

Decline in cognitive function has been seen to be associated with the elderly, especially in the area of short-term memory. More emphasis has been placed on the need for proper nutrition during pregnancy, in order to deliver infants with well-developed brain function. The importance of proper nutrition intake during infancy and early life, in order to continue to develop optimum brain function, has been clearly indicated – this is especially true for iron intake in the young. In the elderly, the decline in cognitive function has been tied in many cases, to a poor intake of minerals that are key to cognitive function. However, poor dietary intake of these minerals can impact cognitive function at any age. What is cognitive function?

Cognitive Function

An intellectual process by which one becomes aware of, perceives, or comprehends ideas. It involves all aspects of perception, thinking, reasoning, and remembering. (Mosby's Medical Dictionary, 9th edition.© Elsevier)

Figure 1. Depiction of Brain activity, taken from : www.spring.org.uk/psyblog

Minerals for Cognitive Function

The maintenance of good nutritional status is important for normal cognition. Studies have shown that certain micronutrients are directly or indirectly involved in many aspects of cognitive function. These micronutrients include certain vitamins and minerals. In this newsletter, we will concentrate on the minerals that play roles in cognitive function. These minerals include: calcium, iodine, iron, magnesium, selenium, and zinc.

Roles for the Minerals in Cognitive Function

As mentioned, several minerals have important biochemical roles needed for proper cognitive function. Here is an overview of the roles of the minerals involved.

Calcium: Intracellular Ca2+ signals play wide ranging functions as a second messenger in many cellular processes, including synaptic transmission and neural plasticity. Neural plasticity is the brain's ability to reorganize itself throughout life. Neural plasticity allows neurons in the brain to compensate for injury and disease, and to adjust their activities in response to new situations or changes in the environment (MedicineNet.com,1/29/2016). These processes are critical to hippocampal spatial memory formation

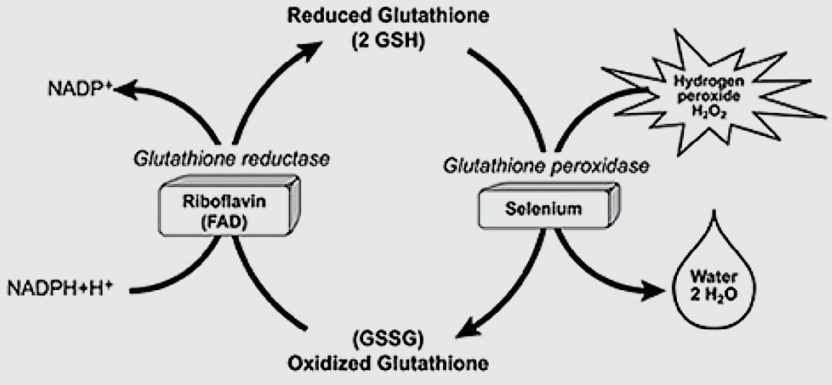
and storage. Endoplasmic reticulum Ca2+ release channels, the ryanodine receptors (a class of intracellular calcium channels), and the inositol 1,4,5-triphosphate receptors contribute to these processes, as well. Calcium ions are important intracellular signals that regulate neuronal gene expression and neuronal secretion of neurotranmitters. Altered Ca 2+ release channel function, presumably contribute to the abnormal memory processes that occur during aging and the neuronal death seen in neurodegenerative diseases (Antioxid Redox Signal 2014 Aug20;21(6) 892-914).

Iron: It is essential for normal neurological function. It is an important component of hundreds of proteins and enzymes involved in cellular metabolism. Iron deficiency in the very young can lead to permanent learning and memory deficits – impaired cognitive function – as well as emotional problems. Iron is needed for the development oligodendrocytes, which are the brain cells that produce myelin (white matter). The myelin layer or sheath is a protective coating that surrounds fibers called axons (R. Williams, The Scientist, Jan.,2012). Myelin contains a high iron content, and myelin increases the speed at which impulses propagate along the neurons. Iron is an important component of several enzymes that synthesize neurotransmitters. Iron transport proteins are important in particular to the hippocampal neurons, which play a large role in learning and memory (Adv. Nutr. Vol 2:112-121, 2011).

Zinc: Has been shown to be essential for neurogenesis, neuronal migration, synaptogenesis, and its deficiency could interfere with neurotransmission and subsequent neuropsychological behavior. Zinc is present in the brain at high levels. Most of the brain zinc is tightly protein bound, but the Zn+2 is present in synaptic vesicles, where it plays a role in neurotransmission mediated by glutamate and GABA, which play roles in cortical excitability (this balance of cortical excitability plays a role in every aspect of human behavior, from abstract thinking to emotional responses). Deficiency of zinc during critical periods of cognitive development can lead to congenital malformation, deficits in attention, learning, memory, and neuropsychological behavior (OSU, Micronutrient Information Center, Drake, V., Linus Pauling Inst., Spring/Summer, 2011). New studies have indicated that zinc is critical to communication between neurons in the hippocampus, the brain's learning and memory center.

Magnesium: Magnesium is necessary for the function of many metabolic enzymes involved in brain function. It is a key regulator of calcium channels involved in neurotransmission (eg. NMDA receptors, important to the maintenance of learning and memory). Extracellular Mg+2 is an important regulator of synaptic density and plasticity in the hippocampus and enhances learning abilities, working memory, and both short and long term memory. Magnesium promotes proper electrical and neurotransmitter functions in the brain (J Neursci, Oct. 19, 2011). Magnesium induces the production of brain-derived neurotrophic factor (BDNF), a compound used by the brain to rejuvenate

Figure 2. Glutathione Oxidation Reduction (Redox) Cycle



One molecule of hydrogen peroxide is reduced to 2 molecules of water while 2 molecules of glutathione (GSH) are oxidized in a reaction catalyzed by the selenoenzyme, glutathione peroxidase. Oxidized glutathione (GSSG) may be reduced by the flavin adenine dinucleotide (FAD) -dependent enzyme, glutathione reductase.

(Figure from OSU, Linus Pauling Institute Micronutrient Center, on-line)

cellular function. Magnesium is required to dissipate the effects of traumatic stress that can occur from intense episodes of fear or anxiety. Magnesium is essential to maintain high brain energy.

Selenium: Selenium in the form of the selenoprotein, selenocysteine, is present at the catalytic site of glutathione peroxidase. Selenium availability regulates glutathione peroxidase enzyme activity, one of the body's master antioxidants. Glutathione peroxidase (GSH-Px) is present throughout the body, but it plays it greatest roles in cognitive function and synaptic plasticity processes. The maintenance of GSH-Px is necessary to fight against reactive oxygen species' potential to cause oxidative damage to neuronal components, which underlies the molecular basis of neurodegeneration and brain aging (See Figure2). The maintenance of normal glutathione levels is important for acquisition, but not consolidation, of spatial memory. Lack of glutathione induces failures in hippocampal synaptic plasticity mechanisms, possibly related to a spatial memory deficit.

Iodine: This mineral is more important to cognitive function than commonly believed. Iodine is a critical component for thyroid hormone synthesis. The thyroid hormones are involved in the myelination of the central nervous system. A major impact of hypothyroidism due to iodine deficiency is impaired neurodevelopment, particularly at critical periods of fetal development and early in life. In extreme cases, the cognitive effect of developmental iodine deficiency is irreversible mental retardation. Milder cognitive effects include a variety of neurodevelopmental deficits, including intellectual impairment. Correction of mild to moderate iodine deficiency in primary school aged children improves cognition and motor function [M.B Zimmerman, Seminars in Cell & Developmental Biology 22(2011)645-652].

Clinical Study Demonstrating Albion's Zinc Bisglycinate's Impact on Cognition

Objective: The aim of this study was to explore the relationship of zinc nutrition to the severity of attention-deficit/hyperactivity disorder (ADHD) symptoms in a middle-class American sample with well-diagnosed ADHD. Previous reports of zinc in ADHD, including two positive clinical trials of supplementation, have come mainly from countries and cultures with different diets and/or socioeconomic realities.

Method: Children 5–10 years of age with DISC- and clinician-diagnosed ADHD had serum zinc determinations and parent and teacher ratings of ADHD symptoms. Zinc levels were correlated (Pearson's and multiple regression) with ADHD symptom ratings.

Results: Forty-eight children (37 boys, 11 girls; 33 combined type, 15 inattentive) had serum zinc levels with a median/mode at the lowest 30% of the laboratory reference range; 44 children also had parent/teacher ratings. Serum magnesium levels were normal. Nutritional in- take by a parent-answered food frequency questionnaire was unremarkable. Serum zinc correlated at r = -0.45 (p = 0.004) with parent-teacher-rated inattention, even after controlling for gender, age, income, and diagnostic subtype, but only at r = -0.20 (p = 0.22) with CPT omission errors. In contrast, correlation with parent-teacher-rated hyperactivity-impulsivity was nonsignificant in the opposite direction.

Conclusion: These findings add to accumulating evidence for a possible role of zinc in ADHD, even for middle-class Americans, and, for the first time, suggest a special relation- ship to inattentive symptoms. They do not establish either that zinc deficiency causes ADHD nor that ADHD should be treated with zinc. Hypothesis-testing clinical trials are needed. (L.E. Arnold, et al, J. of Child and Adolescent Psycho. Vol.15,No 4., 2005, pp. 628-636)

Summation

This study demonstrates that Albion's bisglycinate chelates can cross the blood brain barrier, and therefore impact cognitive function. Therefore, the listed minerals (calcium, iron, magnesium, and zinc) as bisglycinate chelates can cross the blood brain barrier and have the impact on cognitive function in the ways that were discussed for each mineral in this newsletter. Albion's Selenium Glycinate Complex, is not a chelate, and the selenium is separated from the glycine in GI tract, and absorbed as free selenium, which can cross the blood brain barrier. A study by DiSilvestro, et al,(FASEB J. 21:227.2), demonstrated that Albion's Selenium Glycinate Complex significantly raised Serum Glutathione Peroxidase activity, which plays a critical role in protecting the central nervous system, and asserting a positive impact on cognitive function.

There have been many published clinical studies on these Albion bisglycinate chelates that have demonstrated the high bioavailability, lack of negative side effects, and freedom from interactions in the gut that have negative impact on mineral absorption.

So, if you are formulating cognitive function supplements, you can look to Albion's minerals to assist in helping improve brain function.

(Note, Albion does not produce any iodine ingredients)

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