Using caloric restriction to improve brainpower*!*

Any chess player would agree that maximum utilization of cognitive capabilities is vital for winning chess; but as far as I know, no well-known master has yet adopted the best nutritional strategy for achieving this. I predict that will change. Over the next year my wife Meredith, chairman of the board of the Calorie Restriction Society, and I will introduce a new dietary/lifestyle system for improving thinking capability and achieving the best of health. Our system combines ketone utilization with calorie restriction, the only scientifically proven way to slow the aging process and protect against disease.

By adopting this lifestyle, your brain will likely undergo healthy physiological changes that may include:

Increased secretion of trophic factors – vital for formation of new neural pathways[1]

Better utilization of glucose through increased glucose transporters on cell surfaces[2]

Formation of more IGF-I (Insulin-like Growth Factor-I) receptors[3]

Activation of neuroprotective genes such as SIRT1[4]

All of this contributes to better memory, increased concentration, preservation of neurons, enhanced creativity and planning, as well as greater endurance – essential attributes of developing chess skills to the fullest potential. While leading chess players spend a lot of time studying and some follow exercise regimens, even better results could be achieved by adopting this advanced nutritional lifestyle of calorie restriction and glucose control.

Take a look at the press release, embedded below, about the Calorie Restriction and Glucose Control Workshop.

Here we will include the methodology of how to improve brainpower through glucose control. We will introduce a radically different concept of the ketogenic diet that shows how to produce ketones through a healthy intake of complex carbohydrate.

Calorie restriction has been proven by thousands of scientific experiments as the gold standard for improving function and extending life. My prediction is that once this new regimen is adopted by a few leading masters, their results will be so superior that others will be forced to follow, just to keep up.

If you care about optimal mental function – you owe it to yourself to find out how to use this new system to preserve and improve your cognitive capabilities.

If you would like to discuss it, please call toll free at 1-866-894-1812.

Notes

[1] **Evidence that brain-derived neurotrophic factor is required for basal neurogenesis and mediates, in part, the enhancement of neurogenesis by dietary restriction in the hippocampus of adult mice**
Authors: Lee, Jaewon; Duan, Wenzhen1; Mattson, Mark P.
Source: *Journal of Neurochemistry*, Volume 82, Number 6, September 2002, pp. 1367-1375(9)
Abstract:

To determine the role of brain-derived neurotrophic factor (BDNF) in the enhancement of hippocampal neurogenesis resulting from dietary restriction (DR), heterozygous BDNF knockout (BDNF +/-) mice and wild-type mice were maintained for 3 months on DR or ad libitum (AL) diets. Mice were then injected with bromodeoxyuridine (BrdU) and killed either 1 day or 4 weeks later. Levels of BDNF protein in neurons throughout the hippocampus were decreased in BDNF +/- mice, but were increased by DR in wild-type mice and to a lesser amount in BDNF +/- mice. One day after BrdU injection the number of BrdU-labeled cells in the dentate gyrus of the hippocampus was significantly decreased in BDNF +/- mice maintained on the AL diet, suggesting that BDNF signaling is important for proliferation of neural stem cells. DR had no effect on the proliferation of neural stem cells in wild-type or BDNF +/- mice. Four weeks after BrdU injection, numbers of surviving labeled cells were decreased in BDNF +/- mice maintained on either AL or DR diets. DR significantly improved survival of newly generated cells in wild-type mice, and also improved their survival in BDNF +/- mice, albeit to a lesser extent. The majority of BrdU-labeled cells in the dentate gyrus exhibited a neuronal phenotype at the 4-week time point. The reduced neurogenesis in BDNF +/- mice was associated with a significant reduction in the volume of the dentate gyrus. These findings suggest that BDNF plays an important role in the regulation of the basal level of neurogenesis in dentate gyrus of adult mice, and that by promoting the survival of newly generated neurons BDNF contributes to the enhancement of neurogenesis induced by DR.
PMID: 12354284

Glossary**: Neurogenesi**s: creation of new neurons; common in young brains, it has only recently been found to occur in adult brains, **D**R: dietary restriction, another term for CR calorie restriction
Knockout: deactivation of specific genes; used in laboratory organisms to study gene function.
Dentate gyrus: a substructure of the brain's hippocampus, highly active during encoding (learning) of face-name pairs.

[2, 3] **A ketogenic diet increases brain insulin-like growth factor receptor and glucose transporter gene expression.**
Authors: Cheng CM, Kelley B, Wang J, Strauss D, Eagles DA, Bondy CA.
Developmental Endocrinology Branch, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland 20892, USA.
Source Endocrinology. 2003 Jun;144(6):2676-82:

A ketogenic diet suppresses epileptic seizure activity in children and in juvenile rats. To investigate whether alteration in brain IGF activity could be involved in the beneficial effects of the ketogenic diet, we examined the effects of this diet on IGF system gene expression in the rat brain. Juvenile rats were fed one of three different diets for 7 d: ad libitum standard rat chow (AL-Std), calorie-restricted standard chow (CR-Std), or a calorie-restricted ketogenic diet (CR-Ket). The calorie-restricted diets contained 90% of the rats' calculated energy requirements. The AL-Std diet group increased in weight, whereas the two CR groups merely maintained their weight during the 7-d diet. Glucose levels were significantly reduced in both CR groups compared with the AL-Std group, but only the CR-Ket group developed ketonemia. IGF1 mRNA levels were

reduced by 30-50% in most brain regions in both CR groups. IGF1 receptor (IGF1R) mRNA levels were decreased in the CR-Std group **but were increased in the CR-Ket diet group.** Brain IGF binding protein (IGFBP)-2 and -5 mRNA levels were not altered by diet, but **IGFBP-3 mRNA levels were markedly increased by the ketogenic diet** while not altered by calorie restriction alone.

Brain glucose transporter expression was also investigated in this study. Glucose transporter (GLUT) 4 mRNA levels were quite low and not appreciably altered by the different diets. Parenchymal GLUT1 mRNA levels were increased by the CR-Ket diet, but endothelial GLUT1 mRNA levels were not affected. Neuronal GLUT3 expression was decreased with the CR-Std diet and increased with the CR-Ket diet, in parallel with the IGF1R pattern. These observations reveal divergent effects of dietary caloric content and macronutrient composition on brain IGF system and GLUT expression. In addition, the data may be consistent with a role for enhanced IGF1R and GLUT expression in ketogenic diet-induced seizure suppression.
PMID: 12746332

Glossary **Ketogenic diet:** A diet that generates ketone bodies, which are byproducts of fat metabolism -- It is an alternative therapy to treat epilepsy. It was developed in the early 1900s as a high-fat, low-carbohydrate, low-protein diet for the purpose of reducing or eliminating seizures. **mRNA (**Messenger RNA): Template for protein synthesis, it carries the "message" of the DNA to the cytoplasm of cells where protein is made in amino acid sequences specified by the mRNA. **Parenchymal:** Describing the essential or functional elements of an organ

4. 1: Genes Dev. 2003 Feb 1;17(3):313-21.

How does calorie restriction work?

Koubova J, Guarente L.

Department of Biology, MIT, Cambridge, Massachusetts 02139, USA.

PMID: 12569120

Expression of **genes** for growth and neurotrophic factors increased during CR. This includes **genes** encoding neuroplasticity factors such as neuroserpin. This finding may help explain the improved psychomotor performance observed in CR animals (Ingram et al. 1987).