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IN THE NEWS

Calcium inhibits the production of body fat



Epidemiological studies show that people who consume large amounts of calcium tend to show significantly less body fat than people with low calcium consumption. A preliminary study found that when two cups of yogurt a day were added to the diet, raising the daily consumption from 400 mg to 1000 mg, the subjects lost close to 11 lbs. over 12 months. Another study found that subjects put on a milk-only diet for six weeks lost an average of nearly 20 lbs., while subjects consuming the same amount of calories in the form of standard diet lost less than 4 lbs.

Studies using transgenic mice expressing the human obesity gene showed that calcium deficiency results in increased levels of parathyroid hormone and a metabolite of vitamin D called dihydroxycholecalciferol, in turn leading to greater influx of calcium ions into the fat cells. This means greater activity of the enzyme fatty acid synthase (FAS), resulting in greater production of body fat and lower breakdown of fat for fuel. The mice on a fattening but high-calcium diet showed only half the activity and expression of FAS and produced up to 36% less body fat, compared to the group on the low-calcium diet. The low-calcium diet caused a 67% suppression of the utilization of body fat for fuel. The high-calcium mice had higher core body temperature, indicating that some excess calories were used for heat production (thermogenesis). The high-dairy diet was somewhat more effective at reducing obesity than supplementation with calcium carbonate. This cheap form of calcium does not absorb well, which could explain why it did not promote fat loss. Calcium citrate and calcium bis-glycinate absorb much better into the blood stream.

—Ivy Greenwell

Zemel MB et al. Regulation of adiposity by dietary calcium. *FASEB J* 2000; 14:1132-8.

Cracking the Secrets of Aging

An example of how close we may be to developing therapies to stop aging is the research being performed by Sierra Sciences, Inc. of Reno, Nevada. This company is headed by Dr. William Andrews (formerly of Geron), who was involved in the discovery of a “longevity gene” that produces telomerase.



In an exclusive conversation with a Life Extension contributor, Dr. Andrews indicated that he is very close to introducing a pill to be taken once a year that activates the telomerase gene to produce a limited amount of the telomerase enzyme. This in turn can produce one telomere unit every two years, thus replacing the telomeres that are normally lost to aging.

If this pill becomes a reality, it may be considered a perpetuation of youth because of its ability to restore telomere chains in aged cells. If two pills are taken every two years, the length of the telomere string can be increased, potentially reversing this aspect of aging.

How telomeres function

At the end of each chromosome, there is a string of telomeres that determines the number of times a cell can divide. Humans are composed of groups of many cells that continually die off (about every two years). These cells must be replaced by the division of younger cells. Adding telomeres to the chromosome string is one way of maintaining youthful cells and theoretically prolonging life.

Cells with long strings of telomeres act young, whereas cells with short lengths of telomere are usually impaired. So, if we could add telomeres to the chromosome string every two years, we could potentially stop aging. Adding more telomeres than are lost could result in the reversal of aging, since cells act young with a long string of telomeres and behave older as the string shortens.

Research At Sierra Sciences, Inc.

Here is how Sierra Sciences describes its research:

Sierra Sciences, Inc. is an early stage biotechnology company whose long-term goal is to develop drugs and therapies that will reverse the effects of human aging—enabling people to live longer lives, with health and vigor, free of many of the debilitating effects of old age.

The company’s short-term goal is to develop a drug that will temporarily activate telomerase expression and re-lengthen telomeres of aging human cells. The company believes this drug will help:

- Prevent or treat diseases such as atherosclerosis, osteoporosis, immune senescence and skin aging—where cell division leads to telomere loss.
- Enable stem cell research to advance using “adult” (non-embryonic) stem cells—ultimately making life-saving tissue and organ transplants more readily available.
- Provide anti-aging health benefits, and lower health care costs by enabling our bodies to remain more “youthful” and better able to fight disease as we age.

As human cells grow and divide, the ends of the chromosomes, called telomeres, become shorter with each cell division. Oxidative damage from free radicals also affects telomeres and accelerates this process. Telomere shortening leads to changes in gene expression, and ultimately to the cessation of growth and division—called cellular senescence. Telomerase is an enzyme that can re-lengthen telomeres.

Much evidence suggests that control of telomerase expression in normal cells will have broad therapeutic benefits. For example, a 1998 paper in the journal *Science* showed that expression of telomerase in human cells, grown in culture, enabled them to continue growing and dividing without reaching cellular senescence. A 2000 paper reported altered gene expression, subepidermal effects and tissue fragility in aged human skin (in a reconstituted skin model) that were reversed when telomerase was introduced. A 2001 paper reported chromosomal instability and premature aging phenotypes in telomerase knockout mice that were reversed by re-introduction of the telomerase gene.

After an intensive effort, Sierra Sciences has discovered and is currently preparing scientific papers for publication reporting on key

elements of the transcriptional regulation of telomerase. The company believes that these results provide a basis for developing a drug to temporarily activate (“de-repress”) the telomerase gene in normal human cells, at sufficient levels to re-lengthen telomeres in these cells. Sierra Sciences is now pursuing direct molecular approaches to temporarily activate telomerase expression, and is preparing for external high-throughput screening and drug discovery efforts based on this work.

For more information about Sierra Sciences, log on to its website: www.sierrasci.com.

Magnesium deficiency causes arrhythmias in women

The March 2002 issue of the American Journal of Clinical Nutrition published the results of a study of 22 women, which showed that magnesium deficient diets increased supraventricular ectopy, a type of heart arrhythmia. The women, aged 47 to 78, received diets containing less than half the U.S. recommended dietary allowance for magnesium. Subjects were maintained in a metabolic unit and were supervised for six months. For the initial 81 days of the study, participants were randomized to receive either a placebo or a magnesium gluconate supplement that would raise their magnesium level to greater than the RDA of 320 milligrams per day. Following this period, subjects were switched to the opposite regimen. One or three milligrams of copper was supplemented throughout the course of the study to compensate for the copper deficiency that accompanies magnesium deficient diets.

The low magnesium regimen was determined to provide 130 milligrams magnesium per day, while the high magnesium diet provided 411 milligrams. Magnesium deficient diets predictably lowered serum, urinary and erythrocyte magnesium. Ionized plasma magnesium levels were not affected. Heart rhythms were monitored via Holter electrocardiograms. Supraventricular beats as well as supraventricular plus ventricular beats were significantly higher when the subjects were not consuming adequate magnesium.

Because magnesium is central to many cellular mechanisms that control muscle and nerve cell activity, a deficiency may result in heartbeat abnormalities. Less than half of the RDA consumed for an 81-day period changed heart rhythm in this study. A

magnesium deficient diet could be detrimental to individuals with valvular disease or cardiac hypertrophy, or consumers of alcohol or caffeine who are predisposed to cardiac arrhythmia, and these individuals may require more magnesium than an average diet provides.



—Dayna Dye

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