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Subject: schluckt ihr noch Vitamintabletten?

Posted by [Eisenhauer](#) on Wed, 05 Apr 2006 23:13:22 GMT

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ich bin umgestiegen auf 5 mal obst+gemüse am tag

dazu Prof.Dr.Bernhard Watzl und Dr.Dr.Gerhard Rechkemmer

"Es ist nicht die Einzelsubstanz aus einem Nahrungsmittel, die vor Krankheit schützt", sagt der Münchner Experte Gerhard Rechkemmer, "sondern das Zusammenspiel und die Dosierung einer ganzen Reihe verschiedener Substanzen, die ein Mensch beim Essen von Obst und Gemüse zu sich nimmt." Und sein Kollege Bernhard Watzl von der Bundesforschungsanstalt für Ernährung in Karlsruhe ergänzt: "Nur bei direktem Verzehr der gesamten Frucht wird wirklich das ganze Spektrum an essenziellen Nährstoffen und Sekundären Pflanzenstoffen aufgenommen." Zahlreiche Konsumenten glauben aber ungebrochen an die vorbeugende Kraft von Vitamin- oder Mineralstoffkonzentraten: Das Geschäft mit Pillen, Kapseln und Ampullen wächst, die "hochkonzentriert" und "hochwirksam" gegen überreizte Nerven, den drohenden Herzinfarkt oder Krebs helfen und einen ungesunden Lebenswandel ausgleichen sollen. Jeder fünfte Erwachsene konsumiert hierzulande nach Angaben des pharmakritischen Newsletters "arznei-telegramm" mindestens einmal pro Woche ein Mineralstoff- oder Vitaminpräparat, obwohl die Einnahme nur in speziellen Lebenslagen anzuraten ist.

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [glockenspiel](#) on Wed, 05 Apr 2006 23:16:04 GMT

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eisenhauer, wie oft denn noch...das wurde schon zehntausendmal duchgenommen hier, lies es nochmal nach, die leute, die vitamin-tabs nehmen, kennen diese aussagen, und kennen auch die andere (wahrheit)

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [Eisenhauer](#) on Wed, 05 Apr 2006 23:24:34 GMT

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...ja, das ist Tinos Meinung, aber ich glaube Pilos tristan oder fred nehmen nciht solche Mengen davon und die haben auch Ahnung.

Pilos zB nimmt die doppelten DGE empfehlungen

ein kumpel aus meiner Fußballmannschaft studiert biochemie und meint, dass zuviel von einem vitamin oxidativ wirkt und nur das zusammenspiel wirklich hilft

so long

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [glockenspiel](#) on Wed, 05 Apr 2006 23:28:52 GMT

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Eisenhauer schrieb am Don, 06 April 2006 01:24....ja, das ist Tinos Meinung, aber ich glaube Pilos tristan oder fred nehmen nicht solche Mengen davon und die haben auch Ahnung.

Pilos zB nimmt die doppelten DGE empfehlungen

ein kumpel aus meiner Fußballmannschaft studiert biochemie und meint, dass zuviel von einem vitamin oxidativ wirkt und nur das zusammenspiel wirklich hilft

so long

jetzt mal ernsthaft: tino hat nie gesagt, dass man nicht auf das zusammenspiel achten muss, du bist doch schon eine weile hier...

ausserdem war ich NIE der ansicht, diese mengen wären gut: fred nimmt ausserdem auch eine gewisse menge an antioxidanten, aber darum gehts nicht, ich verteidige die mengen nicht, ich schon gar nicht, aber dieses thema haben wir 100 mal durchgekaut...iss einfach 5x obst, und gut

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [tristan](#) on Thu, 06 Apr 2006 00:48:56 GMT

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Ich habe das auch schon öfters gesagt dass es jeder für sich wissen muss und seinen eigenen Bedarf kennen sollte. Ganz einfach geht das wenn man mal ein paar Wochen die Ernährung mit einer speziellen Software überwacht die die zugeführten Lebensmittel analysiert und die Nährstoffe zusammenfasst. Das geht glaube ich auch online, es gibt verschiedene Datenbanken die überprüft werden. Ich habe das mal gemacht und kann sagen dass ich bei den 2500-3000 kcal die ich am Tag esse bei weitem nicht auf alle benötigten Nährstoffe komme. Dann muss man noch bedenken dass dieses Ergebnis nur eine Richtlinie sein kann, denn diese Datenbanken beachten nicht die heutigen Umweltbedingungen und deren Einfluss auf Lebensmittel. Das heißt, selbst wenn ich es schaffen würde mit 2500 kcal die benötigten Nährstoffe über die Zusammenstellung theoretisch zu mir zu nehmen sähe die Wirklichkeit immernoch anders aus und wird sich als viel schwieriger rausstellen zumal sich bestimmte Stoffe auch gegenseitig behindern. Es sollte ein Mittelweg gefunden werden zwischen den Aussagen der Hersteller (so und so viel braucht man...) und den Aussagen der Ernährungsgesellschaften (ausgewogene Ernährung reicht...). Erstere beachten meist nicht dass jeder einen anderen Bedarf hat je nach Lebensstil, letztere wirken jedoch fast schon lächerlich durch Ignoranz. Wirtschaftlich gesehen sind beide Positionen einleuchtend. Ausreichend versorgt bin ich jedoch nicht ohne NEM. Und wer's genau wissen will kann ja nachmessen lassen. Mit dem doppelten Tagesbedarf werden die meisten nichts falsch machen denke ich, aber Mängel kann man damit meist nicht beheben. Ich sage nicht dass jeder so und

so viel einnehmen, sondern sich über den eigenen Bedarf bewusst sein sollte. Das ist jetzt alles nur bezogen auch Grundversorgung wohl gemerkt.

Bei Antioxidantien kann man halt auch Sachen falsch machen, aber trotzdem mindert das nicht das große Potential das sie besitzen und die tägliche Notwendigkeit. Wenn du mal die Forschung anschaust, dann wirst du sehen dass sie in vielen Bereichen absolut zu empfehlen sind. Da wird es in Zukunft denke ich auch Änderungen geben bzgl. der Empfehlungen, dann werden es auch die Ärzte begreifen dass 90mg Vitamin C eben in vielen Fällen zu wenig sind tgl. Aber momentan ist das die selbe Ignoranz die man auch in anderen Bereichen findet wenn man sogenannte Ernährungsexperten oder gesellschaften befragt. Zum Beispiel die Empfehlungen für die Zufuhr von Kohlenhydraten. Oder dass Brot gesund ist. Oder auch Beispiel Milch. Ohne Milch Osteoporose etc. Der größte Quatsch überhaupt. Selbst Obst ist überbewertet. Gemüse ist z.b viel wichtiger. Von dem Programm 5 Stück am Tag halte ich nicht wirklich viel. Äpfel esse ich kaum noch, da sie heute entweder so kleben dass man mit den Zähnen abrutscht oder sie nach Spülmittel und Chemie schmecken (lol) und jetzt auch noch dieser nette Stoff zugelassen wurde, wie hieß er? Smartfresh?... Paprika sind das am meisten mit Pestiziden und Pflanzmitteln belastete Gemüse überhaupt, die würde ich nichtmal gegen Bezahlung essen. Und Bananen sind eh ungesund. Nicht was den Nährwert angeht, der ist gut, sondern wegen des hohen glykämischen Index. Bekomme zudem Pickel davon.

Long story short:

- Grundversorgung heutzutage schwer alleine über die Ernährung
- Bei höheren Dosen bestimmter Stoffe immer über eigene Situation bewusst sein und ggf. vorher messen lassen.
- Dosierungen von Antioxidantien die über die Empfehlungen der DGE hinausgehen halte ich für sinnvoll (was jedoch nicht Hammerdosen bedeutet).

Eisenhauer, ausgewogene Ernährung ist natürlich wichtig, aber meiner Meinung nach eher in Bezug auf Energiebereitstellung und weniger hinsichtlich der Vitalstoffe. Denn die alle dadurch zu bekommen ist definitiv mehr als schwierig heutzutage.

P.S.: Empfehlen kann ich übrigens Brokkoli und Blaubeeren. Das könntest du für die Paprika und Bananen tauschen.

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [Wüstenmungo](#) on Thu, 06 Apr 2006 08:15:25 GMT

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schlucke noch keine vitamintabletten. werd aber demnächst damit anfangen, denke so gegen anfang/mitte mai. will vitamintabletten, acc + aminosäurekonzentrat, in etwa der dosierung, wie tino sie empfiehlt, über einen zeitraum von mindestens 3 monaten nehmen und meine eigenen schlüsse ziehen!

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Subject: Re: schluckt ihr noch Vitamintabletten?  
Posted by [Eisenhauer](#) on Thu, 06 Apr 2006 12:55:38 GMT  
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jau, danke tristan für die aufklärende Antwort

was mich noch interessieren würde, ist, ob es wirklich stimmt, dass man bei einem kaloriendefizit auch weniger oxidativen stress ausgesetzt ist.

z.B.einfach 500 kcal weg von dem sonst normalen umsatz und gleich hat man eine antioxidative wirkung.(hat irgendwas mit den mitochondrien zu tun, aber dafür fehlt mir das hintergrundwissen)

MfG!

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Subject: Re: schluckt ihr noch Vitamintabletten?  
Posted by [tristan](#) on Thu, 06 Apr 2006 14:16:08 GMT  
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Hey,

Ich habe mich bisher noch nicht soviel damit beschäftigt, aber ich kopiere mal nen Text zu calorie restriction rein den ich auf der platte habe...  
ich denke dass das sinnvoll ist solange man nicht hungert, denn das regt die Nebenniere an und ist stress für den Körper..

one method is acknowledged by most biogerontologists to extend maximum lifespan: Caloric Restriction with Adequate Nutrition (CRAN). The life extending properties of CRAN have been known to science since the beginning of this century, and were studied in detail on rats by Clive McCay at Cornell University in the 1930s. But the foremost name in CRAN studies by far is that of Roy L. Walford, M.D.

Dr. Walford has devoted much of his life to the theory&practice of CRAN. His book THE RETARDATION OF AGING AND DISEASE BY DIETARY RESTRICTION (1988, co-authored with Richard Weindruch, Ph.D.) presents the scientific understanding of CRAN based on his experimental work at the University of California in Los Angeles (UCLA), analysis of experiments and review of the published works of other scientists. Walford's book THE 120 YEAR DIET (1986) popularizes CRAN and gives guidelines on how to practice it. His book THE ANTI-AGING PLAN (1994) describes the experience of Walford and 7 others in the 2-year BioSphere II experiment.

The scientific basis of CRAN explained in this essay is primarily a summary of the material in Dr. Walford's books, although a few citations of other references will be given. Scientific information on food physiology comes primarily from MODERN NUTRITION IN HEALTH AND DISEASE by M.Shils, J.Olsen & M.Shike, Editors (8th Edition, 1994) and REVIEW OF MEDICAL

PHYSIOLOGY by William Ganong (1993). I will also describe my own experiences with CRAN.  
PRELIMINARY REMARKS ON TEMPERATURE, ENERGY AND MATTER

In 1960, SI units (Système Internationale d'Unités) were adopted by the General Conference on Weights and Measures as the ultimate standard for all scientific work. The SI temperature scale is the Kelvin, defined by absolute zero (0°K) and by the temperature at which air-free water freezes at the pressure of its own vapor (the triple point of water, assigned the value 273.15°K). The Celsius scale is defined relative to the Kelvin scale by °C = °K-273.15. The centigrade scale is defined by the melting temperature of ice in air at a pressure of one atmosphere (0°) and the boiling point of water at one atmosphere (100°). Celsius, not centigrade, is an official SI unit, but Celsius and centigrade temperatures are very close for most practical purposes.

The SI unit of energy is the Joule, which is equal to one Newton-meter. The Joule is used to measure heat and work, both of which are forms of energy. A Newton-meter is the work performed when an object is moved a distance of one meter by a force of one Newton. BTUs, Liter-atmospheres, Ergs, Electron-volts, Foot-pounds, Calories and other units of energy measure are now all defined in terms of Joules. Originally, one calorie was defined as the amount of heat required to raise the temperature of one gram of water from 14.5 to 15.5°C. Now the calorie is defined as 4.1840 Joules. The word "Calorie" as used by nutritionists is actually equivalent to the scientific kilocalorie (1,000 calories). Supposedly, biologists are to use a capital "C" to distinguish "Calories" (kilocalories) from "calories", but I have rarely observed nutritionists doing this in practice. I will use "Calorie" to mean kilocalorie in accordance with the recommendations.

Fats, carbohydrates and proteins are the energy-supplying macronutrients found in foods. If these macronutrients are combusted in a laboratory they yield 9.3 Calories, 4.1 Calories and 5.3 Calories, respectively. The figures for fats and carbohydrates correspond to energy-yields of metabolism because these macronutrients are completely oxidized in the body to carbon dioxide and water. But since protein is not completely oxidized (urea is formed), the energy value for protein metabolism is about 4.1 Calories. Even these figures do not represent usable energy, however. From 100 Calories of fat, carbohydrate and protein the body must expend 4, 6 and 30 Calories (respectively) for assimilation -- resulting in 96, 94 and 70 Calories.

In human terms, a pound of body fat is equivalent to about 3,500 Calories. Women normally have more fat. Fat deposits in nonobese individuals compose 11-15% of body weight in men and 18-21% in women. The typical American diet contains about 3,000 Calories per day for men and 2,000 Calories per day for women. Energy consumption is a function of activity and body weight. The following table gives the approximate Calorie use for 10 minutes of the listed activities: (Calories Burned in 10 Minutes of Activity)

BODY WEIGHT (POUNDS)	125	175	250
Sleeping	10	14	20
Watching TV	10	14	18
Standing	12	16	24
Walking at 2 mph	29	40	58
Swimming (crawl)	40	56	80
Walking at 4 mph	52	72	102

Walking at 5.5 mph 90 125 178  
Walking at 7 mph 118 164 232  
Walking upstairs 146 202 288

Typically, 60-70% of daily Calories are used to maintain resting metabolic rate (for breathing, heartbeat, body temperature, etc.). 10% is used to digest and process food. At least 15-30% of Calories are used in everyday activities, even for people who are not "physically active". As shown above, the most sedentary activities (sleeping, watching TV), result in about 1,400; 2,000 and 2,900 Calories per day consumed by people weighing 125, 175 and 250 pounds, respectively. However, if CRAN results in higher metabolic efficiency for people of given weights, these figures may have to be adjusted accordingly. In any case, there is individual variation in Caloric use for a given weight. Losing weight is ultimately a matter of more Calories used than Calories consumed on a regular basis.

#### THE METABOLIC UTILIZATION OF MACRONUTRIENT ENERGY

Carbohydrates are simple sugars (the monosaccharides glucose, fructose, mannose and galactose), disaccharides (sucrose, lactose and maltose) or starches (polysaccharides, ie, chains of monosaccharides). Sucrose (cane sugar) is composed of glucose+fructose, lactose (found only in milk) is composed of glucose+galactose, and maltose is two glucoses. These disaccharides are hydrolyzed (broken) into monosaccharides by enzymes in the small intestine. Digestion results in only monosaccharides being absorbed into the bloodstream.

Starches are chains (polymers) of glucose connected by alpha-1-4 & alpha-1-6 linkages, which can be broken by digestive enzymes. Cellulose is also chains of glucose, but connections are by beta-1-4 linkages, which human digestive enzymes cannot break. Hence, cellulose is an insoluble fiber for humans. Because starch most frequently is found within the cells of plant foods, cooking (especially with moist heat) is useful to burst the cell walls and make the food more digestible. There is also enzyme-resistant starch (20% of the carbohydrate from baked beans and 7-10% of the carbohydrate from wheat, oats & potatoes) which passes through the digestive tract to the colon where it is fermented by bacteria to fatty acid, carbon dioxide and methane gas.

Glucose is the primary carbohydrate fuel, which is combined with oxygen to yield water, carbon dioxide and energy. Glycolysis is the first step in glucose metabolism, wherein a single 6-carbon glucose molecule is converted to two 3-carbon pyruvate molecules with a yield of two high-energy ATP molecules. No oxygen is required for this conversion. Fructose, mannose and galactose are converted into a glucose form in the liver before formation of pyruvate. If oxygen is present, pyruvate is completely metabolized to carbon dioxide and water by the citric acid cycle (aerobic glycolysis). Pyruvate formation occurs outside the mitochondria, but the citric acid cycle which oxidizes pyruvate to produce high energy phosphate (ATP) only occurs in the mitochondria (a metabolic process called oxidative phosphorylation). Just prior to entering the citric acid cycle, the 3-carbon pyruvate molecule loses a carbon atom in an energy-yielding reaction that produces carbon dioxide. The remaining 2-carbon acetyl group combines with Coenzyme A to form acetyl-CoA. It is acetyl-CoA that enters the citric acid cycle where it is oxidized to water and carbon dioxide.

If oxygen is not present, pyruvate is converted to lactate (anaerobic glycolysis). Aerobic glycolysis results in a net yield of 19 times as many high-energy ATP molecules as anaerobic glycolysis. But anaerobic glycolysis is useful for short bursts of muscular activity where energy demands do not



allow adequate time for oxygen exchange. For example, a 10-second, 100-metre dash would derive 85% of the energy anaerobically, whereas 95% of the energy would be aerobic in a 60-minute long-distance race. Carbohydrate is stored in the body as a glucose polymer known as glycogen. A 70-kilogram (154-pound) man would have about 100 grams of glycogen stored in the liver and 400 grams stored in muscles -- a total of about 2,000 Calories. By contrast, a 70-kg man would have over 50 times more Calories stored as fat. Normally, about half of ingested glucose is oxidized to carbon dioxide and water, 30-40% is converted to fat and about 5% is stored as glycogen. Dietary carbohydrate reduces the level of serum HDL cholesterol, and sucrose reduces HDL to a greater extent than glucose.

Dietary fat consists mainly of triglycerides, although some phospholipids (mostly lecithin and sphingolipids) and a small amount of sterol (most notably cholesterol) is also ingested. Triglycerides consist of a molecule of glycerol bound to 3 fatty acid molecules. Most fatty acids of foods contain chain lengths of 16 or 18 carbon atoms, notable exceptions being milk fat, coconut oil and palm oil, which have a high proportion of shorter-chain fatty acids. Fatty acids with chain lengths less than 14 carbons are bound to albumin and transported directly to the liver by the portal vein and are not used for fat storage. Phospholipids, cholesterol and long-chain triglycerides are transported from the intestinal mucosa in chylomicrons. Of the 100-140 grams of fat consumed daily in the average North American diet, about 2% will be phospholipid. The primary food sources of phospholipid are egg yolk and legumes (beans, peas and lentils). The fat soluble (pro) vitamins beta-carotene, A, D, E and K are absorbed along with other lipids in the intestine. Linolenic, linoleic and arachidonic acids are also essential nutrients (the essential fatty acids) which accompany fat absorption.

Naturally-occurring fatty acids contain an even number of carbon atoms. Long-chain fatty acids outside the mitochondria must be linked to carnitine (a derivative of the amino acid lysine) to cross the mitochondrial membrane. Then 2-carbon fragments are serially split-off to combine with Coenzyme A to form acetyl-CoA, which enters the citric acid cycle. Most synthesis of fatty acids occurs outside the mitochondria, in the microsomes, beginning with acetyl-CoA building-blocks. These fatty acids are combined with glycerol to form triglycerides for fat storage. Fatty acid synthesis occurs primarily in the liver, although some synthesis also occurs in adipose (fat) tissue. Most fat stored originates from dietary fat. 23% of the energy from carbohydrate is lost in the process of converting it to storage fat, whereas only 2% of the energy from dietary fat is used to store the fat.

Proteins are chains (polymers) of amino acids. The average daily protein intake by a 70-kilogram (154-pound) man in Western countries is about 100 grams. Proteins are hydrolyzed by digestive enzymes and pass into the blood stream as free amino acids. Amino acids entering the blood stream go first to the liver, where most of them are broken-down. Meat protein, for example, is composed of 20% branch-chain amino acids (isoleucine, leucine and valine), but 70% of amino acids leaving the liver after a meat meal are branch-chain. The primary importance of amino acids in human nutrition is as building blocks for structural proteins and enzymes. This is especially true for infants & children, who also need a much higher proportion of the essential amino acids (those the body cannot synthesize). The essential amino acids for human adults are isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan & valine. Human infants also need histidine, and the young of many other species require arginine. Purines and pyrimidines, which form essential components of DNA & RNA, are synthesized from amino acids (ultimately glutamic acid), primarily in the liver.

The amino acids alanine, glycine, cysteine, serine and threonine can be converted to pyruvate and then to glucose. The process of synthesizing new glucose from non-carbohydrates is called gluconeogenesis. The enzymes that allow for reversing the direction of glycolysis (to form glucose from pyruvate) are found in the liver&kidney, and it is in these tissues that gluconeogenesis occurs. Other amino acids can yield energy by entering the citric acid cycle at various points or by being metabolized to glutamic acid, which can enter the citric acid cycle. Although glucose can be converted to fat (via acetyl-CoA), fats are not converted to glucose (unless one allows for the quantitatively insignificant conversion from glycerol resulting from triglyceride hydrolysis).

The brain requires water-soluble nutrient and does not produce glucose by gluconeogenesis. Therefore, glucose is normally the energy source for the brain. Other organs, particularly the heart, oxidize free fatty acids as another source of energy. Skeletal muscles have their own glycogen (glucose) storage and additionally store energy as phosphocreatine. (Creatine is synthesized in the liver from the amino acids methionine, glycine and arginine.) Only when glycogen is low do skeletal muscles use considerable amounts of branch-chain amino acids (especially leucine) for energy. But when skeletal muscles use free fatty acids for energy, branch-chain amino acids use is high. The role of branch-chain amino acids in skeletal muscle metabolism remains to be explained.

#### HORMONES AND MACRONUTRIENT METABOLISM

The influence of hormones on macronutrient metabolism is so complex that only a cartoon can be hoped-for in a brief summary. Chemically insulin is a 21-amino-acid polypeptide chain connected to a 30-amino-acid polypeptide by two disulfide bridges. The half-life of insulin in humans is about 5 minutes. Insulin is secreted by the pancreas, primarily in response to high blood glucose, although amino acids (particularly arginine and leucine -- potentiated by cholecystokinin), keto acids, glucagon, intestinal hormones, acetylcholine and even vagus nerve stimulation can increase insulin release. Conversely, high blood insulin, norepinephrine, epinephrine and pancreas stimulation by sympathetic nerves can inhibit insulin release.

Insulin promotes transport of glucose into the cells of most tissues. Notable exceptions are the brain, the liver and the pancreas itself -- glucose transport is insulin-independent in these organs. Normally, insulin increases skeletal muscle glucose uptake over 3-fold, but skeletal muscle glucose uptake is increased without insulin during the anaerobic conditions of exercise. In addition to facilitating glucose uptake, insulin stimulates the synthesis of proteins from amino acids (particularly in muscles), it stimulates fat synthesis & fat storage, and it increases glycogen synthesis.

The sleepiness that follows a high-carbohydrate meal is an indication of the complexity of insulin's actions. The glucose from the carbohydrate stimulates insulin release, which in turn causes an increased uptake of branch-chain amino acids by muscle tissue. The increased proportion of blood tryptophan experiences less competition for transport into the brain. More tryptophan in the brain leads to increased serotonin synthesis, which promotes sleepiness.

Glucagon is a 29-amino-acid polypeptide with a bloodstream half-life of 5-10 minutes. It is secreted by the pancreas when blood glucose is low or by direct stimulation of the pancreas by sympathetic nerves. Glucagon is also secreted in response to a high protein meal, particularly a meal high in the amino acids (alanine, serine, glycine, cysteine and threonine) that are converted



to glucose in the liver under the influence of glucagon. Glucagon not only promotes gluconeogenesis, it promotes glycogen breakdown, and the release of free fatty acids & glycerol from adipose (fat) tissue. Approximately 60 to 70% of the increased glucose production seen in exercise is mediated by increased glucagon secretion coupled with inhibited insulin release -- and another 30-40% is due to epinephrine.

Epinephrine, thyroid hormones, prostaglandins, sex hormones, intestinal hormones and other hormones have significant effects on the metabolism of macronutrients. Growth hormone mobilizes free fatty acids from adipose tissue and promotes amino acid uptake by muscles for protein synthesis. During the first 2 hours after administration, growth hormone facilitates insulin action by promoting insulin release, but its longer-term actions are more "anti-insulin" since it decreases glucose uptake in some tissues and (more significantly) inhibits the glucose phosphorylation which is the first step in glycolysis (glucose breakdown). The glucocorticoids -- steroids from the adrenal cortex -- have widespread effects on protein & carbohydrate metabolism. Cortisol is the most potent glucocorticoid, followed by cortisone, corticosterone and aldosterone (the latter is the most potent mineralocorticoid -- affecting sodium/potassium excretion). Glucocorticoids increase protein breakdown in skeletal muscle, increase amino acid uptake by the liver, release more glucose from the liver into the bloodstream, increase plasma free fatty acids, decrease fat synthesis in the liver and increase body fat.

#### THE PHYSIOLOGICAL EFFECTS OF FASTING AND A LOW-CALORIE DIET

A study of 32 non-obese young men placed on a diet containing 1600 Calories (two-thirds their normal requirement) showed a loss of 70% of body fat and 23% of body weight. Although protein intake was adequate, 24% of lean tissue weight was lost as well -- and it was observed that loss of body weight proved to be a good index to the amount of lean tissue loss. Basal metabolic rate decreased by 40%, largely due to the loss of lean tissue. Most protein loss comes from liver, spleen and muscles -- and relatively little comes from the heart and brain. Conversion of thyroxine (T4) to triiodothyronine (T3) is reduced, which apparently results in less tissue wasting. Some of the symptoms of hypothyroidism are displayed by "starving" subjects, they display hypothermia and show a weak thermic response to environmental cold.

During the first days of fasting, insulin level drops while glucagon increases slightly. Glycogen stores are soon depleted and glucagon levels peak on the third day when gluconeogenesis is maximal. Muscle tissue releases large amounts of amino acid (primarily alanine and glutamine) -- equivalent to about 50 grams/day for a 70-kilogram man. Much of the glutamine is transformed to alanine by the intestine. Alanine is particularly easily converted to glucose by the liver. During fasting, most of the free fatty acids in the blood are from triglyceride breakdown in adipose tissue rather than from the hydrolysis of chylomicron and VLDL triglyceride seen in the fed condition. Normally, fatty acids are broken down into acetyl-CoA, which enters the citric acid cycle, but when carbohydrate intake is less than 150-180 grams per day, the entry of acetyl-CoA into the citric acid cycle is depressed. Acetoacetyl-CoA accumulates and is converted to the ketone body acetoacetate by the liver. With acid, this can form beta-hydroxybutyrate and (in some tissues outside the liver) acetone. While the muscles are supplying glucose to the brain by protein loss, the ketone bodies become the major energy source for muscle.

As the fast progresses beyond 3 days, the brain adapts to utilizing ketone bodies rather than glucose as an energy source. The efficiency of the kidney in conserving ketone bodies increases. Muscles come to rely increasingly on free fatty acids (rather than ketones) as an energy source,

and muscle protein loss declines considerably (as does gluconeogenesis in the liver).

## THE SCIENTIFIC BASIS OF CALORIE CONSUMPTION DRIVES

There are evidently two primary mechanisms for the control of food consumption in mammals. Short term consumption (immediate hunger) controls the body's food intake on a daily basis. On a long-term basis, a set-point has been proposed which is the weight at which an organism stabilizes.

In normal people, blood glucose seems most directly connected to the craving for food (rather than stomach-filling). But diabetics can have high blood sugar and still be unsatisfied because insulin-deficiency is preventing sugar utilization. Therefore, the difference between glucose in the arteries and glucose in the veins is the best index of hunger. There is also direct signalling from the duodenum (between the stomach & small intestine) to the hypothalamus: the lateral hypothalamus (the so-called "hunger center") and the ventromedial hypothalamus (the so-called "satiety center") show suppressed & increased activity (respectively) in response to increased nutrient in the duodenum.

The "set-point" supposedly governs long-term weight maintenance. If an individual overeats one day, there is a tendency to undereat the following day -- and vice versa. Below a person's "set-point" there is a chronic increase in hunger, and metabolism is reduced (and becomes more efficient) to make further weight loss more difficult. It has been postulated that many obese people have a high genetically-determined set-point which makes it inordinately difficult to lose weight. Fat stores are believed to control set-point, and when circulating levels of free fatty acids are high due to the breakdown of stored fat, food consumption is increased.

Other mechanisms also influence food consumption. The pancreatic hormone glucagon, calcitonin and the small-intestine hormone cholecystokinin can decrease appetite. The number of adipose cells can increase when a person gains weight (especially, but not exclusively, in childhood), but the number can never decrease. Psychological factors can be very important. A 1991 survey in West Germany rated "a fine meal at home" as one of the greatest pleasures of life, exceeded only by holidays, sex/love and family activities (East Germans differed only by excluding sex/love) [APPETITE, 20:246,1993].

## THE EFFECTIVENESS OF C.R.A.N. IN MANY SPECIES

Protozoans of the species *Tokophyra infusionum* were divided into 3 groups of 60 individuals each: (1) abundant food (2) abundant food every second day and (3) limited food every second or third day. The average lifespan for the 3 groups was 7, 10 and 13 days, respectively. From each group, after 9 days there were 11, 32 and 46 survivors. Only members of the third group survived more than 17 days.

Spiders of the family Linyphiidae fed 8, 5 or 1-3 flies per week had maximum lifespans of 30 days, 99 days, and 139 days, respectively. *Caenorhabditis elegans* nematodes have a mean lifespan of 16 days in culture of  $10 \times 10^9$  *Escherichia coli* bacteria cells per millimeter and 26 days for  $10 \times 10^8$  cells per millimeter.

LIFESPAN PROLONGATION by the Ukrainian biogerontologists V. Frolkis and K. Muradian describes studies on 200 different mammalian species. The greatest life-prolonging effect was seen in the range of 40% to 60% caloric restriction, and 85% of the studies concentrated on this

range. The effect on males of the species studied tended to be 3 times as great as on females, and males were studied 3 times more often. Mean lifespan was extended by 22% on average, and maximum lifespan was extended by 23%, on average. For male mice, rats and hamsters, caloric restriction of 25%, 55% and 65% begun at 1 month (just before maturity) increased mean lifespan by 19%, 54% & 65% -- and maximum lifespan by 13%, 44% & 51%. The effectiveness of CRAN on rats tended to be 50% greater than on mice.

#### DR. WALFORD'S RODENT STUDIES

A review of 100 rat studies reported in the JOURNAL OF NUTRITION in 1969 noted that only 24 of the studies had used diets with adequate vitamins and minerals [ANNUAL REVIEWS OF BIOCHEMISTRY, 40:549,1971]. High standards of nutrition and uniformity of diet were established in Dr. Walford's laboratories, so his data deserves to be taken most seriously. He even ensured that coprophagy was prevented. The animals were fed on Mondays, Wednesdays and Fridays, which has been shown to be slightly more effective than reduced rations of food given on a daily basis. Restriction of fat, protein or carbohydrate without a reduction in total Calories did not increase maximum lifespan.

Dr. Walford decided that many previous attempts to apply CRAN to adult animals often did not succeed because the restriction of Calories had been applied too rapidly. CRAN produces the greatest extension of lifespan when applied just before puberty, but the result is a smaller animal. Applying CRAN just after maturity, Walford was able to achieve roughly 90% of the extension of both average and maximum lifespan seen in the rodents restricted before puberty -- without the stunted growth. Walford observed extension of mean and maximum lifespan even when CRAN was begun (gradually) in middle-aged and older rodents. According to Walford, adoption of CRAN "halfway through life will yield half the extension one could obtain by starting in childhood".

#### THE EFFECTS OF C.R.A.N. -- CLUES TO WHY IT WORKS

From an evolutionary or survivalist point of view, many of the observed effects of CRAN make a great deal of sense. When food is abundant an animal grows large, matures fast and reproduces. When food is scarce, less energy is devoted to growth, basal metabolism or reproductive capacity. Energy is maintained for muscular action, which is most important for survival. Adult male rats with 50% Caloric Restriction show a 42% drop in serum testosterone and a 29% drop in luteinizing hormone (LH). Female adult rats also show a drop in LH, but follicle-stimulating hormone (FSH) rises. There are, nonetheless, irregularities in estrus cycles (which could be eliminated by re-feeding). Puberty is delayed in both pre-pubescent males and females, and fecundity is reduced after puberty.

CRAN reduces the weight of most organs and tissues. This is especially true of fat, liver, kidney and lymphoid tissue, but less so for muscle and bone. Brain weight is not reduced. Brain, kidney and liver all show a reduction of total DNA, RNA and protein, but the DNA, RNA and protein content per cell increases in all cases except for kidney RNA (which declines). Ribosomal activity increases in heart muscle and decreases in skeletal muscle. Protein synthesis and turnover normally declines with age, but this occurs much more slowly with CRAN animals. In fact, most organs (except skeletal muscles & lungs) show some increase in protein synthesis with CRAN.

In normal aging animals, an increase was seen in DNA chromatin compactness in non-histone chromatin proteins due to an increased number of disulfide bonds. This would presumably inhibit transcription. But CRAN animals (age-matched to controls) showed a reduction in the disulfide

bonds which inhibit transcription. Higher levels of DNA repair were observed for middle-aged and older CRAN animals when compared to age-matched control animals. Much higher levels of gene expression were also seen (as indicated by synthesis, mRNA levels and transcription in liver preparations).

Laboratory rodents normally spend 5% of energy on physical activity, 35% on basal metabolism and 60% on heat production to maintain body temperature. Genetically obese rodents typically are colder and spend less energy on heat production due to defective thermoregulatory systems. On a CRAN diet mice show a lower internal body temperature, but rats do not. Therefore, lowering of body temperature does not appear to be the means by which CRAN lengthens lifespan. Although total basal metabolism is lowered for CRAN animals, this lowering is entirely in proportion to loss of body mass -- basal metabolism per unit of body mass is little different from that seen in controls. There is evidence that CRAN rodents become more metabolically efficient in their use of food energy -- and this metabolic efficiency is retained when the animals are returned to normal feeding.

The principal hormones secreted by the thyroid gland are thyroxine (T4) and triiodothyronine (T3). Both hormones are iodine-containing amino acids which are chemically different only insofar as T4 contains 4 iodines and T3 contains 3 iodines. One third of T4 in the bloodstream is normally converted to T3 by iodine removal. T4 and T3 both increase oxygen consumption in almost all metabolically active tissues, with the notable exception of the brain, testes, uterus, lymph nodes, spleen and anterior pituitary. T3 acts more rapidly and is 3-5 times more potent than T4. CRAN animals show a drop in serum T3 to levels less than half that of control animals, but total body T3 (including that bound to proteins) is the same for both animal-types.

F344 rats on a CRAN diet show a 15% reduction in blood glucose, which could mean less protein cross-linking insofar as glucose is known to assist non-enzymatic glycosylation (one hypothetical mechanism of aging). And, in fact, reduction of blood levels of glycated hemoglobin has been observed in both human diabetics corrected for hyperglycemia (high blood glucose) and for animals on a CRAN diet. CRAN animals show less collagen aging (cross-linking) as evidenced by greater fibril contractility & relaxation, as well as by reduced breaking time of collagen fibrils. Glycation of collagen in the kidney could be a factor in diabetic kidney failure, and glycation of capillary membranes could contribute to age-related insulin resistance.

15% of people over 60 experience maturity-onset diabetes, with excessive Caloric intake probably being the most frequent cause. The likelihood of developing diabetes doubles with each decade of life, and also doubles with every 20% gain in body weight above average. Diabetics show many features of accelerating aging and are more susceptible to arteriosclerosis, cataracts and nerve damage. Diabetic neuropathy may involve glycation of myelin, with widespread effects on brain function and brain control of hormonal regulation through the hypothalamus. Experimental diabetes in rats induces abnormalities in hypothalamic neurons, whereas rats on a CRAN diet show reduction in age-related peripheral neuropathy.

Accumulation of the age pigment lipofuscin, thought to be the final product of free-radical lipid peroxidation, is one of the most universal changes seen in post-mitotic cells. CRAN diets have been shown to reduce lipofuscin accumulation in all species tested. 20-50% reduction in brain lipofuscin is seen in mice on low protein diets. 25% reduction in heart lipofuscin is seen in middle age mice on CRAN diets. SuperOxide Dismutase (SOD) antioxidant activity increase is seen with

low protein, but not with CRAN diets. In fact, brain homogenates of CRAN mice show lower activities of SOD and lysosomal enzymes. CRAN diets have been shown to increase the antioxidant glutathione somewhat, and to very dramatically increase the activity of the antioxidant catalase. Catalase activity is normally high in the liver & kidney, but typically falls with age. Catalase may also be important in preventing cataracts in the lenses of the eye.

The thymus gland of CRAN mice never reaches the maximum size seen in the controls, but decline in mass is much slower so that its size eventually exceeds that of controls. When 45% of control thymus cortex is lymphoid, 60% of CRAN thymus cortex is lymphoid. Both leukocytes and lymphocytes are less in CRAN animals than in controls, but the CRAN rodents show a robust response to stimulation of immune response. Antibody levels are low in the serum of CRAN mice, but cell mediated T-cell killing of tumor cells is increased. It does not appear that the vitality of the CRAN rodents' immune system is the basis of extended lifespan, however. Rejuvenation of the immune system of an older animal by thymus transplants from a younger animal does not extend maximum lifespan. The fact that invertebrates (including protozoa) that lack an immune system can still show the benefits of CRAN suggests that cellular metabolic alterations are probably more fundamental than the immune system improvement.

CRAN animals show less age-related decline in dopamine receptors of the brain striatum, less age-related decline in corticosteroid receptors in the hippocampus, less age-associated increase in serum calcitonin & parathyroid hormone, and elevated hemoglobin-content & erythrocyte count. Many of the alterations seen in CRAN animals match those seen with the removal of the hypothalamus. But hypophysectomy may simply induce Caloric Restriction by removing or damaging hunger centres -- such animals voluntarily eat considerably less.

Dr. Walford believes that CRAN works primarily because of an evolutionarily selected adaptive response at the level of "proliferative systems". In his own words: "This adaptation involves a selective upregulation in repair and protective processes, an increased metabolic efficiency, a decreased production of damaging agents, and is accompanied by signals to the neuroendocrine network, particularly the hypothalamus." He does not believe that intrinsic cellular events in non-proliferative systems (ie, non-dividing cells, such as neurons and heart muscles) are the distinguishing features that differentiate maximum lifespan in CRAN animals from maximum lifespan in controls -- even though these events may be the major cause of death for CRAN animals.

CRAN has benefits for mean lifespan as well as for maximum lifespan, and these benefits should not be discounted. CRAN animals show a marked decrease in susceptibility to most diseases. Diet is known to be an even greater causative factor for cancer than tobacco. Kidney disease & cataracts are reduced and the immune system is healthier with CRAN. Dr. Walford estimates that susceptibility to cancer, heart disease and diabetes is half as great for people practicing CRAN. He also suspects a reduction in osteoporosis. Complications due to bone fractures can be fatal subsequent to accidents in the elderly. (George Bernard Shaw died after fracturing a hip when trying to climb a tree at age 93.) Calcitonin (which inhibits bone resorption and lowers plasma calcium) rises with age to a lesser degree for CRAN rodents than for controls. Nonetheless, unlike rodents, humans show a decline in calcitonin with age. The effect of CRAN on human osteoporosis is still unknown. It is known, however, that the rate of osteoporosis in human vegetarians is lower, despite a lower calcium intake.



My own view is that CRAN works primarily by 2 mechanisms: (1) reduced glycation (protein cross-linking by sugars like glucose, fructose and galactose) and (2) reduced generation of free-radicals. Reduced calorie intake ultimately results in reduced body mass, which requires less energy to sustain in steady state. Less energy requirements means (1) reduced blood sugar required and (2) reduced ATP-generation by mitochondria required. The drop in blood glucose and reduced free-radicals mean less cross-linking and free-radical damage. Ordinarily, reducing blood glucose would result in hypoglycemia, but if glucose demands are reduced, this is not a problem. Reducing the production of free-radicals is a far more efficient way to reduce free-radical damage than by attempting to quench the free radicals with anti-oxidants once they are formed. (Also, there are homeostatic mechanisms that reduce natural anti-oxidant enzymes when supplemental anti-oxidants are introduced.)

#### THE COMBINATION OF PROTEIN RESTRICTION WITH C.R.A.N

Early studies showing lifespan increase with protein restriction created controversy concerning whether these benefits were simply another example of CRAN. This issue was apparently resolved by a paper that appeared in the JOURNAL OF NUTRITION Vol.116, p.641-654 (1986), authored by Richard Weindruch, Roy Walford, Suzanne Fligiel and Donald Guthrie. Since Weindruch (who got his Ph.D. under Walford) is listed first, he may deserve the most responsibility for this study. These data illustrate a study of the effect of combining protein restriction with CRAN.

Mean and maximum lifespan for 6 cohorts of mice under 6 dietary protocols can be summarized by the following table (RES = Restriction):

	MEAN (months)	MAXIMUM (months)
(1) ad lib diet	27	35
(2) 25% RES after weaning (day 21)	33	40
(3) 55% RES after weaning (day 21)	42-43	51
(4) 55% RES before weaning (day 7)	42-43	51
(5) 55% RES after weaning (day 21) with gradual protein RES	40	48-49
(6) 65% RES after weaning (day 21)	45	53

Maximum lifespan was taken as the mean life span of the longest-lived 10% in each group.

Mice in cohorts (3), (4), (5) and (6) all had 35% casein (milk protein) in their diets. But the mice on gradual protein restriction received 35% casein from weaning until 4 months, 25% casein at 4-12 months, 20% casein at 12-24 months and 15% casein from 24 months until death. Calories lost from protein restriction were compensated for by carbohydrate in an equal mixture of sucrose and corn starch. The authors make the statement that "Mice restricted in both calorie and protein

intake exhibited shorter mean and maximum lifespans (~5%) than did mice fed the same number of calories of a high protein diet."

This appears to be convincing evidence that protein restriction does not increase lifespan in CRAN animals. The probable main reason that vegetarians tend to live longer is that by eliminating meat they are (A) eliminating saturated fat [with all its associated cardiovascular problems] and (B) reducing their caloric intake by eating foods that are not so high in either fat or protein. The group (5) and (6) mice had the least increase in kidney weight with age, which is consistent with claims that high protein diets create stress on kidney function. A high protein diet also pre-disposes one to gout. Nonetheless, these hazards of protein on specific systems are evidently in the opposite direction to the general benefits of higher protein elsewhere in the body -- leading to higher mean and maximum lifespans. The paper does not prove, however, that excessively high protein diets do not shorten lifespan nor does it prove that protein restriction would not be of benefit for those not practicing CRAN.

This paper made the interesting observation that there was a tendency for heavier mice in the CRAN cohorts to live longer than lighter mice in the same cohort. The authors make the statement: "The increased longevity of the heavier restricted mice might suggest that metabolically efficient individuals on low calorie regimens are longer lived than individuals less capable of storing ingested calories as body mass."

ARE ANIMAL C.R.A.N. STUDIES APPLICABLE TO HUMANS?

Although humans and other primates do not synthesize Vitamin C, rats&mice do. Therefore, experiments concerning Vitamin C done on rats&mice may not be applicable to humans. Nonetheless, these rodents have hearts, brains, livers, kidneys, cellular functions, hormones and immune systems very similar to that of humans. Pharmaceutical companies would not spend vast amounts of money on experiments with rats&mice if these did not usually lead to results relevant to human beings. Humans have such long lifespans that it is not feasible to perform the necessary lifespan experiments and get useful results in reasonable time. The benefits of CRAN have been seen in such a wide variety of species that it is difficult to believe that they are not applicable to humans.

Claims have been made that actuarial tables show that it is healthier to be slightly above average in weight. But the Framingham study has shown that 80% of smokers are in the underweights group whereas 55% are in the overweight group. Dr. Walford has said that controlling the data for subclinical disease which can lower body weight, for the biological effects of obesity (hypertension, hyperglycemia), and for smoking shows that human mortality is optimal for weights at least 10% below average. Moreover, body weight is not necessarily an indication of Caloric Restriction. Genetically obese mice show the benefits of CRAN even though their body weights are the same as the body weights of controls. Additionally, among the control rodents, no relationship was found between body weight and lifespan.

The objection has been raised that if CRAN works for humans, it would have been naturally discovered. But although Caloric Restriction has been common in human history, Adequate Nutrition has not. Doing a computer search of random combinations of high quality foods, one of Dr. Walford's co-workers found that only rarely would a 1,500 kcal/day diet contain adequate nutrition. Nutrients in low supply (in order of scarcity) were: zinc, Vitamin E, copper, magnesium, iron, niacin, Vitamin B12, pantothenic acid, calcium, riboflavin, folacin, Vitamin A, Vitamin B6,

thiamine and Vitamin C. Since only one person in 50-100 thousand people lives over 105 years of age, it seems unlikely that enough people would have adopted CRAN accidentally to exceed the maximum lifespan of the population at large.

Dr. Walford does believe, however, that there is a population of people living on something approximating CRAN -- namely the people of Okinawa. Although vitamins and minerals are adequate, the energy intake of Okinawan children is only 62% of the "recommended intake" for Japan. The average Okinawan adult consumes 20% less Calories than the average Japanese. Death rates in the 60-64 age group have been 60% lower than for Japan. Despite the larger proportion of people over 65 in Okinawa, the number of Okinawan centenarians per 100,000 people over 65 is many times greater than for the Japanese.

The typical American daily diet contains about 3,000 Calories for men and 2,000 Calories for women -- 42% from fat (a 2:1 ration of saturated:unsaturated fat). The 8 people in BioSphere averaged 1,800 Calories/day during the first 6 months, increasing to 2,200 Calories/day by the end of 2 years. In the first 6 months, body weights dropped an average of 15%, blood sugar dropped 20%, blood cholesterol dropped 38%, blood pressure dropped 30%/27%, and white blood cell count dropped 24%.

Life expectation (remaining years of life expected, on average) is the same as mean lifespan from birth. A North American male at birth has a mean lifespan & life expectation of about 72 years. At age 65, the life expectation is about 15 years, which means that the average age of death for males still alive at age 65 is 80 years. One could also speak of maximum life expectation, which does not normally change at any age. But if rodent studies on CRAN are applicable to humans, a 60% Caloric Restriction should produce a roughly 50% increase in mean&maximum life expectation from whatever age the program is begun. A 10% Caloric Restriction could produce a roughly 25% increase in life expectation. Dr. Walford says that even a person between the ages of 50 and 60 could experience a 10 to 15-year life extension with CRAN. In this sense, it can be compared to cessation of cigarette smoking -- a benefit at whatever age it is begun, although the sooner the better.

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [Haar-in-der-Suppe](#) on Thu, 06 Apr 2006 14:26:11 GMT

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@ tristan : könntest du mal einen link zu einem solchen programm posten? also so eins wo man die nährstoffe etc sieht...

am besten nicht via internet databank, ein programm wäre schon besser

das mit den mitrochondrien und dem oxidativem stress hab ich mir schon immer irgendwie gedacht... aber das hat dann nix mit kaloriendefizit zu tun sondern mit dem umsatz

---

Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [tristan](#) on Thu, 06 Apr 2006 14:33:47 GMT

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auch interessant...

<http://www.geo.de/GEO/kultur/gesellschaft/4767.html?p=1>

"Kannenweise Tee. Wenig Salz, kein Brot und keine Milchprodukte. Und Gewürze und Kräuter, löffelvoll: Chili, Ingwer, Kurkuma."

"Das bunte Essen ist also das Geheimnis der langlebigen Menschen von Okinawa? Nicht nur; noch wichtiger als Vielfalt sei der Verzicht, belehrt Ushi ihre 77-jährige Tochter. Wenn du auch so alt werden willst wie ich, musst du weniger essen. Stopf dich niemals voll. Hör auf mich, "hara hachi bu". Es klingt wie eine Zauberformel, ein Mantra der Selbstkontrolle. "Hara hachi bu" heißt so viel wie: "den Magen nur zu 80 Prozent füllen". Die Tochter, eine hübsche Frau, die kaum älter als 50 wirkt, gießt dampfenden Tee in Porzellanschalen und verdreht dezent die Augen. Wie oft mag sie das wohl schon gehört haben? Wo ihr doch anzusehen ist, dass sie diese Regel personifiziert. Eine Regel, die mittlerweile unzählige Experimente in aller Welt bestätigt haben: Kalorienreduktion führt zu einer gesunden Verlängerung des Lebens."

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [tristan](#) on Thu, 06 Apr 2006 14:44:21 GMT

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Hi,

Ich habe das nicht mehr installiert, aber ich hatte dieses Programm:

<http://nutrition-software-review.toptenreviews.com/kathleens-diet-planner-software.html>

Als Database gibt es die USDA Nutrient database, die kann man leicht über dieses Tool abrufen wenn man einzelne Sachen wissen möchte..

<http://www.dietsoftware.org/navigator.shtml>

Ig

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Subject: Re: schluckt ihr noch Vitamintabletten?

Posted by [Eisenhauer](#) on Thu, 06 Apr 2006 15:19:39 GMT

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"das mit den mitochondrien und dem oxidativem stress hab ich mir schon immer irgendwie

gedacht... aber das hat dann nix mit kaloriendefizit zu tun sondern mit dem umsatz"

hmmm , verstehe ich noch nicht so ganz...

also wenn mein "gesamtumsatz" 2400 betrat und ich nur 2000 zu mir nehme, ist das korrekt so ?

irgendwie ist ein kaloriendefizit(vom gesamtumsatz namlich) doch der grundumsatz+gesamtumsatz minus die reduktion der kalorien

so long

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Subject: Re: schluckt ihr noch Vitamintabletten?  
Posted by [Gast](#) on Sun, 09 Apr 2006 15:13:02 GMT  
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Ich habe fruher ohne NEM gut gelebt und kann es auch heute. Ich war jetzt einige Zeit im Suden, ohne auch nur ein Mal an NEM zu denken, habe Gutes gegessen, mehr (und vor allem besseres) Obst Gemuse, war viel in der Luft, unter der Sonne, hatte erholsamen Schlaf, kurz ich habe ein angemessenes Leben gefuhrt. Der HA in dieser Zeit war gegen null, die Haut in perfektem Zustand. Und das habe ich durch kein NEM je erreicht, schon gar nicht in der kurzen Zeit. Hatte davor auch schon seit einiger Zeit nur Vit C u. Zink beides nur sporadisch genommen. Nun bereits seit Wochen gar nichts mehr.

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Subject: Re: schluckt ihr noch Vitamintabletten?  
Posted by [glockenspiel](#) on Mon, 10 Apr 2006 07:50:27 GMT  
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sancho pansa schrieb am Son, 09 April 2006 17:13Ich habe fruher ohne NEM gut gelebt und kann es auch heute. Ich war jetzt einige Zeit im Suden, ohne auch nur ein Mal an NEM zu denken, habe Gutes gegessen, mehr (und vor allem besseres) Obst Gemuse, war viel in der Luft, unter der Sonne, hatte erholsamen Schlaf, kurz ich habe ein angemessenes Leben gefuhrt. Der HA in dieser Zeit war gegen null, die Haut in perfektem Zustand. Und das habe ich durch kein NEM je erreicht, schon gar nicht in der kurzen Zeit. Hatte davor auch schon seit einiger Zeit nur Vit C u. Zink beides nur sporadisch genommen. Nun bereits seit Wochen gar nichts mehr.

perfekt !

wurde ich auch sofort, nur ich komm hier nicht raus