Volume 3 Issue 1

In This Issue

Protein Overload

Muscle, vitality, strength, power, energy, vigor, aggressiveness, and liveliness are words that come to mind when people think of the benefits of protein in their diet. The truth is quite the opposite. ***Bone loss, osteoporisis, kidney damage, kidney stones, immune dysfunction, arthritis, cancer promotion, low-energy, and overall poor health*** are the real consequences from overemphasizing protein. Protein serves as raw material to build tissues. Without sufficient protein from your diet, your body would be in trouble – but, aside from starvation, this never happens. Yes, a little protein is good, but more is not better. Protein consumed beyond our needs is a health hazard as devastating as excess dietary fat and cholesterol. Unfortunately, almost everyone on the typical Western diet is overburdened with protein to the point of physical collapse. The public has almost no awareness of problems of protein overload, but scientists have known about the damaging effects of excess protein for more than a century. See page 2

Could Mad Cow Disease Already be Killing Thousands of Americans Every Year?

by Michael Greger, M.D.
January 7, 2004

October 2001, 34-year-old Washington State native Peter Putnam started losing his mind. One month he was delivering a keynote business address, the next he couldn't form a complete sentence. Once athletic, soon he couldn't walk. Then he couldn't eat. After a brain biopsy showed it was Creutzfeldt-Jakob disease, his doctor could no longer offer any hope. "Just take him home and love him," the doctor counseled his family.[1,2,3] Peter's tragic death, October 2002, may
have been caused by Mad Cow disease.
Seven years earlier and 5000 miles away, Stephen Churchill was the first in England to die. His first symptoms of depression and dizziness gave way to a living nightmare of terrifying hallucinations; he was dead in 12 months at age 19.[4] Next was Peter Hall, 20, who showed the first signs of depression around Christmas, 1994. See page 13

In his book, *Physiology Economy in Nutrition*, Russell Henry Chittenden, former President of the American Physiological Society (APS) and Professor of Physiological Chemistry at Yale, wrote in 1905, "Proteid (protein) decomposition products are a constant menace to the well-being of the body; any quantity of proteid or albuminous food beyond the real requirements of the body may prove distinctly injurious…Further, it requires no imagination to understand the constant strain upon the liver and kidneys, to say nothing of the possible influence upon the central and peripheral parts of the nervous system, by these nitrogenous waste products which the body ordinarily gets rid of as speedily as possible."[1]

A Cluttered House

Say you are building a house that requires one hundred sheets of plywood, but the lumberyard delivers two hundred sheets. Initially, you might think this is a good thing – a real windfall. But what do you really have? – A big pile of expensive lumber cluttering your yard. The mess is an eyesore, a fire hazard, a potential source of physical injury, and it detracts from your property value. Disposal of the excess lumber will be costly and could result in physical injury. To make matters worse, the lumber yard delivered "Fire Retardant Treated" (FRT) plywood. The fire-retardants used are very acidic, and under humid conditions the acids weaken the lumber, leading to a potential structural failure of your house – a catastrophic threat to life and limb – not to mention your pocketbook.[2]

Lesson Learned: When building a house, take delivery of only the amount of lumber you actually need – and use only the safest construction materials.

What are Your Construction (Protein) Needs?

Protein from your diet is required to build new cells, synthesize hormones, and repair damaged and worn out tissues. So how much do you need?

The protein lost from the body each day from shedding skin, sloughing intestine, and other miscellaneous losses is about 3 grams per day (0.05 grams/Kg).[3] Add to this loss other physiological requirements, such as growth and repairs. The final tally, based on solid scientific research, is: **your *total daily need for protein is about 20 to 30 grams.***[4,5] Plant proteins easily meet these needs.[6]

see page 3

So what are people consuming? Those living in many rural Asian societies consume about 40 to 60 grams from their diet of starch (mostly rice) with vegetables.[6] On the Western diet, typical food choices centered around meat and dairy products, "a well-balanced diet," provides about 100 to 160 grams of protein a day. A traditional Eskimo, eating marine animals, or someone on the Atkins diet, from various kinds of meat and dairy, might be consuming 200 to 400 grams a day.[7] Notice that there can be a 10-fold (1000%) difference from our basic requirements and the amount some people consume. The resilience of the human body allows for survival under conditions of incredible over-consumption.

Protein for the Human Body

Situation	Protein* (grams per day)	Protein (% of calories)
Daily Protein Loss	3 grams	Less than 1%
Minimal requirements	20 – 30 grams	4% - 5%
Breast-fed infant	3 – 12 grams	5% – 6.3 %
Asian Diet	40 - 70 grams	8% - 14%
McDougall Diet	35 – 75 grams	7% - 15%
Western Diet	75 – 160 grams	15% - 32 %
Zone Diet	150 grams	30%
South Beach Diet	100 – 250 grams	20% - 50%
Atkins Diet	150 – 350 grams	30% - 70%

*Calculations based on 2000 calories consumed for adults. Calories consumed on a weight loss program may often be less than 2000 per day. In this case the absolute amount of protein would be proportionally less. Breast-fed infants consume about 350 to 1100 calories a day.

Once the body's needs are met, then the excess must be removed. The liver converts the excess protein into urea and other nitrogen-containing breakdown products, which are finally eliminated through the kidneys as part of the urine.

The Fate of Excess Protein

There are two possible fates for excess nutrients: They can be stored, like excess dietary fats and oils are stored in the body fat (adipose) tissues of our belly, thighs, and buttocks - or the excesses can be eliminated. If protein were stored it would be deposited in our muscle tissues and people on the Western diet would all look like bodybuilders. Obviously, excess protein is not stored. Protein consumed in excess of the body's needs therefore must be eliminated, with some effort, and often at great expense to our health.

see page 4

Excess Protein Burdens the Kidneys and Liver

Processing all that excess dietary protein – as much as 300 grams (10 ounces) a day –causes wear and tear on the kidneys; and as a result, on average, 25% of kidney function is lost over a lifetime (70 years) from consuming the Western diet.[8,9] Fortunately, the kidneys are built with large reserve capacity and the effects of losing one-quarter of kidney function are of no consequence for otherwise healthy people. However, people who have already lost kidney function for other reasons – from an accident, donation of a kidney, infection, diabetes, and hypertension – may suffer life-threatening consequences from a diet no higher in protein than the average American consumes.[10,11]

The time-honored fundamental treatment for people with failing kidneys is a low-protein diet. End-stage kidney failure, requiring dialysis, can usually be postponed or avoided by patients fortunate enough to learn about the benefits of a low-protein diet.[10-13]

People suffering with liver failure are also placed on diets low in protein as fundamental therapy – short of a liver transplant, this is the most important therapy they will receive. During the end stages of liver failure, patients will often fall into a coma from the build-up of protein breakdown products (hepatic coma). A change to a cost-free, very low-protein diet can cause these dying people to awaken. Well planned, plant-food based diets are particularly effective with both kidney and liver disease.[14,15]

Excess Protein Damages the Bones = Osteoporosis

Worldwide, rates of hip fractures (and kidney stones) increase with increasing animal protein consumption (including dairy products). For example, people from the USA, Canada, Norway, Sweden, Australia, and New Zealand have the highest rates of osteoporosis. [15,16] The lowest rates are among people who eat the fewest animal-derived foods (these people are also on lower calcium diets) – like the people from rural Asia and rural Africa.[15,16]

Osteoporosis is caused by several controllable factors; however, the most important one is the foods we choose – especially the amount of animal protein and the foods high in acid.[17-19] The high acid foods are meat, poultry, fish, seafood, and hard cheeses – parmesan cheese is the most acidic of all foods commonly consumed.[20] This acid must be neutralized by the body.[21] Carbonate, citrate and sodium are alkaline materials released from the bones to neutralize the acids. Fruits and vegetables are alkaline and as a result a diet high in these plant foods will neutralize acid and preserve bones. The acidic condition of the body caused by the Western diet also raises cortisol (steroid) levels. [22] Elevated cortisol causes severe chronic bone loss – just like giving steroid medication for arthritis causes severe osteoporosis.

Consequence Two: Kidney Stones

Once materials are released from the solid bone, the calcium and other bone substances move through the blood stream to the kidneys where they are eliminated in the urine. In an effort to remove the overabundance of waste protein, the flow of blood through the kidneys (glomerular filtration rate) increases – the result: calcium is filtered out of the body. Naturally, the kidneys attempt to return much of this filtered calcium back to the body; unfortunately, the acid and sulfur-containing amino acids from the animal foods thwart the body's attempts to conserve calcium. See page 5

Compare the acid load of various foods:

(Renal Acid Load per 100 calories) [23,24]	
Beef	6.3
Chicken	7.0
Fish (Cod)	9.3
Cheddar Cheese	10.0
Potato	-5.0
Peas	1.0
Wheat Flour	1.0
Banana	-6.0
Apples	-5.0
Spinach	-56.0
Tomatoes	-18.0

(A positive value indicates acidic, whereas a negative value indicates alkaline.)

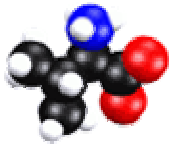
The final result is each 10 grams of dietary protein in excess of our needs (30 grams daily) increases daily urinary calcium loss by 16 mg. Another way of looking at the effects is: doubling protein intake from our diet increases the loss of calcium in our urine by 50%.[25] Plant proteins (plant food-bases) do not have these calcium and bone losing effects under normal living conditions.

Once this bone material arrives in the collecting systems of the kidney it easily precipitates into sold formations known as kidney stones.[27] Over 90% of kidney stones found in people following a high-protein, Western diet are formed primarily of bone-derived calcium. Following a healthy diet is the best way to prevent kidney stones.[28]

Toxic Sulfur Distinguishes Animal Foods

The *qualities* of the proteins we consume are as important as the quantities. One very important distinction between animal and plant-derived protein is that animal proteins contain very large amounts of the basic element *sulfur*. This sulfur is found as two of the twenty primary amino acids, *methionine and cysteine*. Derived from these two primary sulfur-containing amino acids are several other sulfur-containing amino acids – these are keto-methionine, cystine, homocysteine, cystathionine, taurine, cysteic acid.

see page 6



Methionine **Valine**

The yellow sphere represents the element sulfur.

Compare the relative amounts of the sulfur containing amino acid, methionine, found in these common foods (based on the same number of calories): [24]

- Beef provides 4 times more than pinto beans
- Eggs have 4 times more than corn
- Cheddar cheese has 5 times more than white potatoes
- Chicken provides 7 times more than rice
- Tuna provides 12 times more than sweet potatoes

Even though sulfur-containing amino acids are essential for our survival, an excess of these amino acids beyond our needs places a critical burden upon our body and detracts from our health in six important ways:

1) Amino *acids*, as the name implies are acids; the sulfur-containing amino acids are the strongest acids of all, they breakdown into powerful *sulfuric acid*. Excess acid, as discussed above, is a primary cause of bone loss leading to osteoporosis and kidney stone formation.[29]

2) Methionine is metabolized into homocysteine – animal foods are the major source of the amino acid, homocysteine, in people – the more meat in the diet, the higher a person's blood level of homocysteine. A diet high in fruits and vegetables lowers the levels of this amino acid. Epidemiological and clinical studies have proven homocysteine to be an independent risk factor for heart attacks, strokes, closure of the arteries to the legs (peripheral vascular disease), blood clots in the legs (venous thrombosis), thinking problems (cognitive impairment), and even worse mental troubles, like dementia, Alzheimer's disease, and depression.[30]

3) Sulfur feeds cancerous tumors. Cancer cell metabolism is dependent upon methionine being in the diet; whereas, normal cells can grow on a methionine-free diet (feeding off of other sulfur-containing amino acids). This methionine-dependency has been demonstrated for breast, lung, colon, kidney, melanoma, and brain cancers.[31,32] Increasing methionine in the diet of animals promotes the growth of cancer.[33]

There is also evidence of cancer promoting effects of methionine mediated through a powerful growth stimulating hormone, called i*nsulin-like growth factor* - 1 (IGF-1).[34] Meat and dairy products raise IGF-1 levels and promote the growth of cancers of the breast, colon, prostate, and lung.[35]

4) Sulfur from sulfur-containing amino acids is known to be toxic to the tissues of the intestine, and to have deleterious effects on the human colon, even at low levels.[36] The consequence of a diet of high-methionine (animal) foods may be a life-threatening inflammatory bowel disease, called ulcerative colitis.[37-38] see page 7

5) Sulfur restriction prolongs life.[39] Almost seventy years ago, restricting food consumption was found to prolong the life of animals by changing the fundamental rate at which aging occurs.[40] Restriction of methionine in the diet has also been shown to prolong the life of experimental animals. By no coincidence, a diet based on plant foods is inherently low in both calories and methionine – thus the easiest and most effective means to a long and healthy life.

6) Possibly a stronger motivation to keep protein, and especially methionine-rich animal protein, out of your diet is foul smelling odors – halitosis, body odor, and noxious flatus – akin to the smell of rotten eggs – are direct results of the sulfur (animal protein) you eat.[41,42]

Do Not Waste Your Health Away

Animal foods, full of protein waste, promote poor health and early death by accelerating the aging process and increasing the risk of diseases, like heart disease, diabetes, and cancer, that in their own right, cause premature death. From now on, think of the excess protein you consume ***as garbage*** that must be disposed of in order to avoid toxic waste accumulation. Obviously, the best action is to avoid the excess in the first place and this is most easily accomplished by choosing a diet based on starches, vegetables, and fruits. Within a few days of changing to a healthy diet, most of the waste will be gone and the damaged tissues will begin healing.
Unfortunately, you will find little support for such an obvious, inexpensive, and scientifically-supported approach – especially when the common masses of people worldwide are ignorant of the truth – most are gobbling down as much protein as they can stuff in their mouths – and the food industry is supporting this behavior by advertising their products as “high-protein” and "Atkins-approved" – as if this was somehow good for the body. This paradox is age-old, and because it is ruled by emotions, rather than clear thinking, a change in mind-set in your lifetime, should not be expected.

Two thousand years ago, in this Bible passage, Paul asked for tolerance between meat eaters and vegetarians (Romans 14:1-2). “One man’s faith allows him to eat everything, but another man whose faith is weak, eats only vegetables. The man who eats everything must not look down on him who does not, and the man who does not eat everything must not condemn the man who does...” Do not wait for a consensus before you take action.

References:

1) Chittenden, R. H. (1905). Physiological economy in nutrition, with special reference to the minimal protein requirement of the healthy man. An experimental study. New York: Frederick A. Stokes Company.
2) Fire Retardant Treated Plywood: http://www.nexsenpruet.com/library/docs/NPCOL1_624753_1.pdf
3) Calloway DH. Sweat and miscellaneous nitrogen losses in human balance studies.
J Nutr. 1971 Jun;101(6):775-86.
4) Hegsted DM.. Minimum protein requirements of adults. *Am J Clin Nutr.* 1968 May; 21(5): 352-7.
5) Dole V. Dietary treatment of hypertension: clinical and metabolic studies of patients on the rice-fruit diet, *J Clin Invest,* 1950; 29: 1189-1206.
6) Millward DJ. The nutritional value of plant-based diets in relation to human amino see page 8

acid and protein requirements. *Proc Nutr Soc*. 1999 May;58(2):249-60.

7) Mazess RB. Bone mineral content of North Alaskan Eskimos. *Am J Clin Nutr.* 1974 Sep; 27(9): 916-25.

8) Brenner BM. Dietary protein intake and the progressive nature of kidney disease: the role of hemodynamically mediated glomerular injury in the pathogenesis of progressive glomerular sclerosis in aging, renal ablation, and intrinsic renal disease. *N Engl J Med.* 1982 Sep 9; 307(11): 652-9.

9) Meyer TW. Dietary protein intake and progressive glomerular sclerosis: the role of capillary hypertension and hyperperfusion in the progression of renal disease. *Ann Intern Med.* 1983 May; 98(5 Pt 2): 832-8.

10) Hansen HP. Effect of dietary protein restriction on prognosis in patients with diabetic nephropathy. *Kidney Int.* 2002 Jul; 62(1): 220-8.

11) Biesenbach G. Effect of mild dietary protein restriction on urinary protein excretion in patients with renal transplant fibrosis. *Wien Med Wochenschr.* 1996; 146(4): 75-8.

12) Pedrini MT. The effect of dietary protein restriction on the progression of diabetic and nondiabetic renal diseases: a meta-analysis. *Ann Intern Med.* 1996 Apr 1;124(7):627-32.

13) Cupisti A. Vegetarian diet alternated with conventional low-protein diet for patients with chronic renal failure. *J Ren Nutr*. 2002 Jan;12(1):32-7.

14) Bianchi GP. Vegetable versus animal protein diet in cirrhotic patients with chronic encephalopathy. A randomized cross-over comparison. J Intern Med. 1993 May; 233(5): 385-92.

15) Abelow B. Cross-cultural association between dietary animal protein and hip fracture: a hypothesis. *Calcific Tissue Int* 50:14-8, 1992.

16) Frassetto LA . Worldwide incidence of hip fracture in elderly women: relation to consumption of animal and vegetable foods. *J Gerontol A Biol Sci Med Sci*. 2000 Oct;55(10):M585-92.

17) Maurer M. Neutralization of Western diet inhibits bone resorption independently of K intake and reduces cortisol secretion in humans. *Am J Physiol Renal Physiol.* 2003 Jan;284(1):F32-40.

18) Remer T. Influence of diet on acid-base balance. *Semin Dial.* 2000 Jul-Aug;13(4):221-6.

19) Frassetto L. Diet, evolution and aging--the pathophysiologic effects of the post-agricultural inversion of the potassium-to-sodium and base-to-chloride ratios in the human diet. *Eur J Nutr.* 2001 Oct;40(5):200-13.

20) Remer T. Potential renal acid load of foods and its influence on urine pH. *J Am Diet Assoc.* 1995 Jul;95(7):791-7.

21) Barzel US. Excess dietary protein can adversely affect bone. *J Nutr.* 1998 Jun;128(6):1051-3.

22) Maurer M. Neutralization of Western diet inhibits bone resorption independently of K intake and reduces cortisol secretion in humans. *Am J Physiol Renal Physiol.* 2003 Jan; 284(1): F32-40. Epub 2002 Sep 24.

23) Remer T. Potential renal acid load of foods and its influence on urine pH. *J Am Diet Assoc.* 1995 Jul;95(7):791-7.

24) J Pennington. Bowes & Church's Food Values of Portions Commonly Used. 17th Ed. Lippincott. Philadelphia-New York. 1998.

25) Massey LK . Dietary animal and plant protein and human bone health: a whole foods approach. *J Nutr.* 2003 Mar; 133(3): 862S-865S.

26) Jenkins DJ. Effect of high vegetable protein diets on urinary calcium loss in middle-aged men and women. *Eur J Clin Nutr.* 2003 Feb;57(2):376-82.

27) Lemann J Jr. Relationship between urinary calcium and net acid excretion as determined by dietary protein and potassium: a review. *Nephron.* 1999; 81 Suppl 1: 18-25.

28) Delvecchio FC. Medical management of stone disease. *Curr Opin Urol.* 2003 May; 13(3): 229-33. see page 9

29) Remer T. Influence of diet on acid-base balance. *Semin Dial.* 2000 Jul-Aug; 13(4): 221-6.

30) Troen AM. The atherogenic effect of excess methionine intake. *Proc Natl Acad Sci U S A*. 2003 Dec 9; 100(25): 15089-94.

31) Cellarier E. Methionine dependency and cancer treatment. *Cancer Treat Rev.* 2003 Dec; 29(6): 489-99.

32) Epner DE. Nutrient intake and nutritional indexes in adults with metastatic cancer on a phase I clinical trial of dietary methionine restriction. *Nutr Cancer.* 2002; 42(2): 158-66.

33) Paulsen JE. Growth stimulation of intestinal tumours in Apc(Min/+) mice by dietary L-methionine supplementation. *Anticancer Res.* 2001 Sep-Oct; 21(5): 3281-4.

34) Stubbs AK. Nutrient-hormone interaction in the ovine liver: methionine supply selectively modulates growth hormone-induced IGF-I gene expression. *J Endocrinol*. 2002 Aug; 174(2): 335-41.

35) Yu H. Role of the insulin-like growth factor family in cancer development and progression. *J Natl Cancer Inst.* 2000 Sep 20;92(18):1472-89.

36) Levine J. Fecal hydrogen sulfide production in ulcerative colitis. *Am J Gastroenterol.* 1998 Jan;93(1):83-7.

37) Roediger W. Sulphide impairment of substrate oxidation in rat colonocytes: a biochemical basis for ulcerative colitis? *Clin Sci (Lond).* 1993 Nov;85(5):623-7.

38) Christl S. Effect of sodium sulfide on cell proliferation of colonic mucosa. *Gastroenterology* 1994; 106:A664 (abstr).

39) Zimmerman JA. Nutritional control of aging. *Exp Gerontol*. 2003 Jan-Feb; 38(1-2): 47-52.

40) McCay C. The effect of retarded growth upon length of lifespan and upon ultimate body size. *J Nutr.* 1935; 10: 63-79.

41) McDougall J. Halitosis Is More than Bad Breath . McDougall Newsletter. January 2002 at www.drmcdougall.com.

42) McDougall J. Bad Farts? Meat Stinks! *McDougall Newsletter*. August 2002 at www.drmcdougall.com.

Favorite Five
My Favorite Five Articles from Last Month's Medical Journals

Mammography Is Fraud Promoted by Industry and Governments

Everyone should read this entire free article in the *British Medical Journal:*

Full Text: http://bmj.bmjjournals.com/cgi/content/full/328/7432/148

PDF File: http://bmj.bmjjournals.com/cgi/reprint/328/7432/148

Presentation on websites of possible benefits and harms from screening for breast cancer: cross sectional study, by Karsten Juhl Jorgensen in the January 17, 2004 *British Medical Journal* found poor and severely biased information on the Internet that is harmful to women about breast cancer screening. The 13 sites from advocacy groups and the 11 from governmental institutions all recommended mammographic screening, whereas the three from consumer organizations questioned screening. The organizations were from Australia, Canada, Denmark, New Zealand, Norway, Sweden, the United Kingdom, and the United States. All of the advocacy groups accepted industry funding – apparently without restrictions. In contrast, the three consumer organizations acknowledged the risk of bias related to industry funding, and two of them did not accept such funding at all. Advocacy groups and governmental organizations presented a positive view on screening with little concern for major harms of screening, such as over-diagnosis and over-treatment. The authors summarize, "The present situation is that a woman customer who visits a 'screening shop' doesn't know what she is buying into, and most often the shopkeeper either doesn't know or doesn't tell. This is untenable."
I recommend against breast cancer screening because it does far more harm than good (plain and simple, it doesn't work, but creates a huge business with great profits). I realize this position will be unpopular with many people – but it is the truth. For more on my views on this subject read my book, "The McDougall Program for Women," and my February 2002 newsletter article, "Mammography is Unjustified – A Letter Few Newspapers Will Print."

Jorgensen KJ, Gotzsche PC. Presentation on websites of possible benefits and harms from screening for breast cancer: cross sectional study. *BMJ*. 2004 Jan 17; 328(7432): 148.

High Carbohydrate* Diet Causes Effortless Weight Loss

Effects of an Ad Libitum (without restriction on amount) **Low-Fat, High-Carbohydrate Diet on Body Weight, Body Composition, and Fat Distribution in Older Men and Women: A Randomized Controlled Trial** by Nicholas P. Hays in the January 26, 2004 issue of the *Archives of Internal Medicine* found when older men and women consumed as much as they wanted of high-carbohydrate, low-fat foods they lost weight without "dieting." Over 12-weeks, participants on the recommended diet lost about 7 pounds without cutting calories and without exercising – and almost 11 pounds with 45 minutes of stationary bike-riding, four times weekly. The control group lost no weight. The experimental diet was 63% carbohydrate and 18% fat – the McDougall diet is even more effective because it is 80% carbohydrate and 7% fat. This article is an excellent review of the principles discussed in my book, the McDougall Program for Maximum Weight Loss, first published in 1994 (just to point out, not much has happened in 10 years for better nutrition). The authors provide many recent scientific references establishing why a high carbohydrate diet effortlessly and effectively results in weight loss without hunger in overweight people, and provides a lifetime of trim body weight maintenance.

See page 11

The scientific explanations for why a diet of starches, vegetables and fruits is the real solution for obesity in the Western world are found in this article. These are the three main principles:

1) High carbohydrate foods are very low in calorie density – very bulky, so they fill the stomach with fewer calories than the Western (American) diet.

2) The fat you eat is the fat you wear – fat is effortlessly stored – excess carbohydrates are not turned into fat under normal living conditions – excess carbohydrate is simply burned off.

3) Carbohydrates satisfy your hunger drive – fats leave you unsatisfied and looking for food (carbohydrate). You act like an "Obsessive Compulsive Overeater" – like you have some kind of emotional-mental disorder, and all you really are is hungry for carbohydrates.

This article would be well worth a trip to your local library (hospital, university, or community) for a copy to help you explain to family and friends why they are on the wrong track. This article may also be purchased for $12 (US) over the Internet at: http://archinte.ama-assn.org.

Nicholas P. Hays; Raymond D. Starling; Xiaolan Liu; Dennis H. Sullivan; Todd A. Trappe; James D. Fluckey; William J. Evans. Effects of an Ad Libitum Low-Fat, High-Carbohydrate Diet on Body Weight, Body Composition, and Fat Distribution in Older Men and Women: A Randomized Controlled Trial *Arch Intern Med*. 2004;164:210-217.
* Please understand that when I write about carbohydrates I mean starches, vegetables, and fruits – ***not*** donuts, cookies, cake, potato chips, and French fries.

Formula (Bottle) Feeding Causes Infant Brain Damage

Exclusive breastfeeding of healthy term infants for at least 6 weeks improves neurological condition by H. Bouwstra in the December 2003 issue of the *Journal of Nutrition* found poorer neurologic function in children breast-fed less than 6 weeks compared to those who had more breast milk. General movements are a sensitive marker for the condition of the nervous system, and when assessed at three months they are a powerful predictor of future neurologic function. With breast-feeding, 47% of infants were rated "normal-optimal," but only 18% who were not breast-fed received this rating. Furthermore, there was 4 times the risk of receiving a "mildly-abnormal" score if they were formula-fed rather than breast-fed (47% vs.12%). The presence of "mildly-normal" generalized movements at 3 months is associated with an increased risk of minor neurological dysfunction and attention problems at school age.
It has long been recognized that the intelligence of a child improves with duration of breast-feeding, but this study shows actual physical impairment of the nervous system in children deprived of the advantages of human breast milk. The most likely reason for the difference is the nutritional qualities of breast milk, which cannot be replicated by drug companies as synthetic infant formulas.
When I become Surgeon General of the United States the first action I will take is to make infant formulas available only with a doctor's prescription – and any doctor who writes such a prescription will be held accountable to a medical review board more stringent than the one that now looks over narcotic-drug prescribing habits of doctors. You can learn more about the importance of breast-feeding in "The McDougall Program for Women" book. See page 12

Bouwstra H, Boersma ER, Boehm G, Dijck-Brouwer DA, Muskiet FA, Hadders-Algra M. Exclusive breastfeeding of healthy term infants for at least 6 weeks improves neurological condition. *J Nutr.* 2003 Dec; 133(12): 4243-5.

Cow's Milk Causes Multiple Sclerosis

Antibody cross-reactivity between myelin oligodendrocyte glycoprotein and the milk protein butyrophilin in multiple sclerosis by Johannes Guggenmos in the January 1, 2004 issue of the *Journal of Immunology* found evidence that multiple sclerosis is caused from the consumption of cow's milk – by causing attacks by the immune system through a process known as *molecular mimicry.*

You may have heard that multiple sclerosis (MS) is an *autoimmune disease* or "a disease where the body attacks itself" – and in this case the nervous system is attacked. Molecular mimicry is a natural response of the immune system against foreign proteins misdirected to our own tissues. In this case, cow-milk proteins enter the bloodstream through the intestinal wall. Antibodies are made against this foreign substance. Because of the similarity between the structure of these proteins and the ones that make up the nervous system tissues, these antibodies attack and destroy parts of the human brain (myelin). The resulting injury causes loss of vision, hearing, sensation and strength. Within 10 years of diagnosis, half of the victims of MS are wheel-chair bound, bed-ridden, or dead – unless they are fortunate enough to change their diet. With a healthy diet the risk of getting worse over the next 35 years is less than 5% (See on my home page the interview with Dr. Roy Swank on MS).

Guggenmos J, Schubart AS, Ogg S, Andersson M, Olsson T, Mather IH, Linington C. Antibody cross-reactivity between myelin oligodendrocyte glycoprotein and the milk protein butyrophilin in multiple sclerosis. *J Immunol.* 2004 Jan 1; 172(1): 661-8.

Hot Tubs Are Safe for People with High Blood Pressure

Are hot tubs safe for people with treated hypertension? By Tae Won Shin in the December 9, 2003 issue of the *Canadian Medical Association Journal* found spending 10 minutes in a hot tub is safe for most people who are receiving drug treatment for high blood pressure. Twenty-one people on blood pressure medications were compared to 23 people without hypertension after being in a hot tub at 40 degrees centigrade (104 F). There was a little more than a 25% drop in blood pressure in both groups. The fall in pressure was due to dilation of blood vessels caused by the heat. A review presented in this paper of the published literature reveals almost no scientific support for the warning, "People with high blood pressure should not use the hot tub."

Of course, the best course of action for anyone with an elevated blood pressure and/or on medications for this blood pressure would be to improve the health of the cardiovascular system with a healthy diet, lifestyle (no coffee or alcohol), and exercise program. See my August 2002 newsletter article "Take Blood Pressure at Home - Get off Your Medications," for more help.

Shin TW, Wilson M, Wilson TW. Are hot tubs safe for people with treated hypertension? *CMAJ*. 2003 Dec 9; 169(12): 1265-8.

By the next Christmas, he couldn't walk, talk, or do anything for himself.[5] Then it was Anna's turn, then Michelle's. Michelle Bowen, age 29, died in a coma three weeks after giving birth to her son via emergency cesarean section. Then it was Alison's turn. These were the first five named victims of Britain's Mad Cow epidemic. They died from what the British Secretary of Health called the worst form of death imaginable, Creutzfeldt-Jakob disease, a relentlessly progressive and invariably fatal human dementia.[6] The announcement of their deaths, released on March 20, 1996 (ironically, Meatout Day[7]), reversed the British government's decade-old stance that British beef was safe to eat.[8] It is now considered an "incontestable fact" that these human deaths in Britain were caused by Bovine Spongiform Encephalopathy (BSE), or Mad Cow disease.[9] Bovine means "cow or cattle," spongiform means "sponge-like," and encephalopathy means "brain disease." Mad Cow disease is caused by unconventional pathogens called prions--literally infectious proteins--which, because of their unique structure, are practically invulnerable, surviving even incineration[10] at temperatures hot enough to melt lead.[11] The leading theory as to how cows got Mad Cow disease in the first place is by eating diseased sheep infected with a sheep spongiform encephalopathy called scrapie.[12] In humans, prions can cause Creutzfeldt-Jakob disease (CJD), a human spongiform encephalopathy whose clinical picture can involve weekly deterioration into blindness and epilepsy as one's brain becomes riddled with tiny holes. We've known about Creutzfeldt-Jakob disease for decades, since well before the first mad cow was discovered in 1985. Some cases of CJD seemed to run in families; other cases seemed to just arise spontaneously in about one in a million people every year, and were hence dubbed "sporadic." The new form of CJD caused by eating beef from cows infected with Mad Cow disease, though, seemed to differ from the classic sporadic CJD. The CJD caused by infected meat has tended to strike younger people, has produced more psychotic symptoms, and has often dragged on for a year or more. The most defining characteristic, though, was found when their brains were sampled. The brain pathology was vividly reminiscent of Kuru, a disease once found in a New Guinea tribe of cannibals who ate the brains of their dead.[13] Scientists called this new form of the disease "variant" CJD.
Other than Charlene, a 24 year old woman now so tragically dying in Florida, who was probably infected in Britain, there have been no reported cases of variant CJD in the U.S.[14] Hundreds of confirmed cases of the sporadic form of Creutzfeldt-Jakob disease, however, arise in the United States every year,[15] but the beef industry is quick to point out these are cases of sporadic CJD, not the new variant known to be caused by Mad Cow disease.[16] Of course, no one knows what causes sporadic CJD. New research, discussed below, suggests that not hundreds but thousands of Americans die of sporadic CJD every year, and that some of these CJD deaths may be caused by eating infected meat after all.

Although the fact that Mad Cow disease causes variant CJD had already been strongly established, researchers at the University College of London nevertheless created transgenic mice complete with "humanized" brains genetically engineered with human genes to try to prove the link once and for all. When the researchers injected one strain of the "humanized" mice with infected cow brains, they came down with the same brain damage seen in human variant CJD, as expected. But when they tried this in a different strain of transgenic "humanized" mice, those mice got sick too, but most got sick from what looked exactly like sporadic CJD! The Mad Cow prions caused a disease that had a molecular signature indistinguishable from sporadic CJD. To the extent that animal experiments can simulate human results, their shocking conclusion was that eating infected meat might be responsible for some cases of sporadic CJD in addition to the expected variant CJD. The researchers concluded that "it is therefore possible that some patients with [what looks like]... sporadic CJD may have a disease arising from BSE exposure."[17] see page 14

Laura Manuelidis, section chief of surgery in the neuropathology department at Yale University comments, "Now people are beginning to realize that because something looks like sporadic CJD they can't necessarily conclude that it's not linked to [Mad Cow disease]..."[18]

This is not the first time meat was linked to sporadic CJD. In 2001, a team of French researchers found, to their complete surprise, a strain of scrapie--"mad sheep" disease--that caused the same brain damage in mice as sporadic CJD.[19] "This means we cannot rule out that at least some sporadic CJD may be caused by some strains of scrapie," says team member Jean-Philippe Deslys of the French Atomic Energy Commission's medical research laboratory.[20] Population studies had failed to show a link between CJD and lamb chops, but this French research provided an explanation why. There seem to be six types of sporadic CJD and there are more than 20 strains of scrapie. If only some sheep strains affect only some people, studies of entire populations may not clearly show the relationship. Monkeys fed infected sheep brains certainly come down with the disease.[21] Hundreds of "mad sheep" were found in the U.S. in 2003.[22] Scrapie remains such a problem in the United States that the USDA has issued a scrapie "declaration of emergency."[23] Maybe some cases of sporadic CJD in the U.S. are caused by sheep meat as well.[24]
Pork is also a potential source of infection. Cattle remains are still boiled down and legally fed to pigs (as well as chickens) in this country. The FDA allows this exemption because no "naturally occurring" porcine (pig) spongiform encephalopathy has ever been found. But American farmers typically kill pigs at just five months of age, long before the disease is expected to show symptoms. And, because pigs are packed so tightly together, it would be difficult to spot neurological conditions like spongiform encephalopathies, whose most obvious symptoms are movement and gait disturbances. We do know, however, that pigs are susceptible to the disease--laboratory experiments show that pigs can indeed be infected by Mad Cow brains[25]--and hundreds of thousands of downer pigs, too sick or crippled by injury to even walk, arrive at U.S. slaughterhouses every year.[26]

A number of epidemiological studies have suggested a link between pork consumption and sporadic CJD. Analyzing peoples' diet histories, the development of CJD was associated with eating roast pork, ham, hot dogs, pork chops, smoked pork, and scrapple (a kind of pork pudding made from various hog carcass scraps). The researchers concluded, "The present study indicated that consumption of pork as well as its processed products (e.g., ham, scrapple) may be considered as risk factors in the development of Creutzfeldt-Jakob disease." Compared to people that didn't eat ham, for example, those who included ham in their diet seemed ten times more likely to develop CJD.[27] In fact, the USDA may have actually recorded an outbreak of "mad pig" disease in New York 25 years ago, but still refuses to reopen the investigation despite petitions from the Consumer's Union (the publishers of Consumer Reports magazine).[28] Sporadic CJD has also been associated with weekly beef consumption,[29] as well as the consumption of roast lamb,[30] veal,venison, brains in general,[31] and, in North America, seafood.[32,33] The development of CJD has also, surprisingly, been significantly linked to exposure to animal products in fertilizer,[34] sport fishing and deer hunting in the U.S.,[35] and frequent exposure to leather products.[36]

We do not know at this time whether chicken meat poses a risk. There was a preliminary report of ostriches allegedly fed risky feed in German zoos who seemed to come down with a spongiform encephalopathy.[37] Even if chickens and turkeys themselves are not susceptible, though, they may become so-called "silent carriers" of Mad Cow prions and pass them on to human consumers.[38] Dateline NBC quoted D. Carleton Gajdusek, see page 15

the first to be awarded a Nobel Prize in Medicine for his work on prion diseases,[39] as saying, "it's got to be in the pigs as well as the cattle. It's got to be passing through the chickens."[40] Dr. Paul Brown, medical director for the US Public Health Service, believes that pigs and poultry could indeed be harboring Mad Cow disease and passing it on to humans, adding that pigs are especially sensitive to the disease. "It's speculation," he says, "but I am perfectly serious."[41]
The recent exclusion of most cow brains, eyes, spinal cords, and intestines from the human food supply may make beef safer, but where are those tissues going? These potentially infectious tissues continue to go into animal feed for chickens, other poultry, pigs, and pets (as well as being rendered into products like tallow for use in cosmetics, the safety of which is currently under review[42]). Until the federal government stops the feeding of slaughterhouse waste, manure, and blood to all farm animals, the safety of meat in America cannot be guaranteed.

The hundreds of American families stricken by sporadic CJD every year have been told that it just occurs by random chance. Professor Collinge, the head of the University College of London lab, noted "When you counsel those who have the classical sporadic disease, you tell them that it arises spontaneously out of the blue. I guess we can no longer say that."

"We are not saying that all or even most cases of sporadic CJD are as a result of BSE exposure," Professor Collinge continued, "but some more recent cases may be--the incidence of sporadic CJD has shown an upward trend in the UK over the last decade... serious consideration should be given to a proportion of this rise being BSE-related. Switzerland, which has had a substantial BSE epidemic, has noted a sharp recent increase in sporadic CJD."[43] In the Nineties, Switzerland had the highest rate of Mad Cow disease in continental Europe, and their rate of sporadic CJD doubled.[44]
We don't know exactly what's happening to the rate of CJD in this country, in part because CJD is not an officially notifiable illness.[45] Currently only a few states have such a requirement. Because the Centers for Disease Control (CDC) does not actively monitor the disease on a national level,[46] a rise similar to the one in Europe could be missed.[47] In spite of this, a number of U.S. CJD clusters have already been found. In the largest known U.S. outbreak of sporadic cases to date,[48] five times the expected rate was found to be associated with cheese consumption in Pennsylvania's Lehigh Valley.[49] A striking increase in CJD over expected levels was also reported in Florida[50] and New York (Nassau County)[51] with anecdotal reports of clusters of deaths in Oregon[52] and New Jersey.[53]

Perhaps particularly worrisome is the seeming increase in CJD deaths among young people in this country. In the 18 years between 1979 and 1996, only a single case of sporadic CJD was found in someone under 30. Whereas between 1997 and 2001, five people under 30 died of sporadic CJD. So five young Americans dying in five years, as opposed to one young case in the previous 18 years. The true prevalence of CJD among any age group in this country remains a mystery, though, in part because it is so commonly misdiagnosed.[54]

The most frequent misdiagnosis of CJD among the elderly is Alzheimer's disease.[55] Neither CJD nor Alzheimer's can be conclusively diagnosed without a brain biopsy,[56] and the symptoms and pathology of both diseases overlap. There can be spongy changes in Alzheimer's, for example, and senile Alzheimer's plaques in CJD.[57] Stanley Prusiner, the scientist who won the Nobel Prize for his discovery of prions, speculates that Alzheimer's may even turn out to be a prion disease as well.[58] In younger victims, CJD is more often misdiagnosed as multiple sclerosis or as a severe viral infection.[59]

see page 16

Over the last 20 years the rates of Alzheimer's disease in the United States have skyrocketed.[60] According to the CDC, Alzheimer's Disease is now the eighth leading cause of death in the United States,[61] afflicting an estimated 4 million Americans.[62] Twenty percent or more of people clinically diagnosed with Alzheimer's disease, though, are found at autopsy not to have had Alzheimer's at all.[63] A number of autopsy studies have shown that a few percent of Alzheimer's deaths may in fact be CJD. Given the new research showing that infected beef may be responsible for some sporadic CJD, thousands of Americans may already be dying because of Mad Cow disease every year.[64] Nobel Laureate Gajdusek, for example, estimates that 1% of people showing up in Alzheimer clinics actually have CJD.[65] At Yale, out of a series of 46 patients clinically diagnosed with Alzheimer's, six were proven to have CJD at autopsy.[66] In another study of brain biopsies, out of a dozen patients diagnosed with Alzheimer's according to established criteria, three of them were actually dying from CJD.[67] An informal survey of neuropathologists registered a suspicion that CJD accounts for 2-12% of all dementias in general.[68] Two autopsy studies showed a CJD rate among dementia deaths of about 3%.[69,70] A third study, at the University of Pennsylvania, showed that 5% of patients diagnosed with dementia had CJD.[71] Although only a few hundred cases of sporadic CJD are officially reported in the U.S. annually,[72] hundreds of thousands of Americans die with dementia every year.[73] Thousands of these deaths may actually be from CJD caused by eating infected meat.

The incubation period for human spongiform encephalopathies such as CJD can be decades.[74] This means it can be years between eating infected meat and getting diagnosed with the death sentence of CJD. Although only about 150 people have so far been diagnosed with variant CJD worldwide, it will be many years before the final death toll is known. In the United States, an unknown number of animals are infected with Mad Cow disease, causing an unknown number of human deaths from CJD. The U.S. should immediately begin testing all cows destined for human consumption, as is done in Japan, should stop feeding slaughterhouse waste to all farm animals (see http://organicconsumers.org/madcow/GregerBSE.cfm), and should immediately enact an active national surveillance program for CJD.[75]

Five years ago this week, the Center for Food Safety, the Humane Farming Association, the Center for Media & Democracy, and ten families of CJD victims petitioned the FDA and the CDC to immediately enact a national CJD monitoring system, including the mandatory reporting of CJD in all 50 states.[76] The petition was denied.[77]

The CDC argued that their passive surveillance system tracking death certificate diagnoses was adequate. Their analysis of death certificates in three states and two cities, for example, showed an overall stable and typical one in a million CJD incidence rate from 1979 to 1993.[78] But CJD is so often misdiagnosed, and autopsies are so infrequently done, that this system may not provide an accurate assessment.[79]

In 1997, the CDC set up the National Prion Disease Pathology Surveillance Center at Case Western Reserve University to analyze brain tissue from CJD victims in the U.S. in hopes of tracking any new developments. In Europe, surveillance centers have been seeing most, if not all, cases of CJD. The U.S. center sees less than half. "I'm very unhappy with the numbers," laments Pierluigi Gambetti , the director of the Center. "The British and Germans politely smile when they see we examine 30% or 40% of the cases," he says. "They know unless you examine 80% or more, you are not in ouch."[80] "The chance of losing an important case is high."[81] see page 17

One problem is that many doctors don't even know the Center exists. And neither the CDC nor the Center are evidently authorized to reach out to them directly to bolster surveillance efforts, because it's currently up to each state individually to determine how--or even whether--they will track the disease. In Europe, in contrast, the national centers work directly with each affected family and their physicians.[82] In the U.S., most CJD cases--even the confirmed ones--seem to just fall through the cracks. In fact, based on the autopsy studies at Yale and elsewhere, it seems most CJD cases in the U.S. aren't even picked up in the first place.

Autopsy rates have dropped in the U.S. from 50% in the Sixties to less than 10% at present.[83] Although one reason autopsies are rarely performed on atypical dementia cases is that medical professionals are afraid of catching the disease,[84] the primary reason for the decline in autopsy rates in general appears to be financial. There is currently no direct reimbursement to doctors or hospitals for doing autopsies, which often forces the family to absorb the cost of transporting the body to an autopsy center and having the brain samples taken, a tab that can run upwards of $1500.[85]

Another problem is that the National Prion Disease Pathology Surveillance Center itself remains underfunded. Paul Brown, medical director for the National Institutes of Health, has described the Center's budget as "pitiful," complaining that "there isn't any budget for CJD surveillance."[86] To adequately survey America's 290 million residents, "you need a lot of money." UK CJD expert Robert Will explains, "There was a CJD meeting of families in America in which... [the CDC] got attacked fairly vigorously because there wasn't proper surveillance. You could only do proper surveillance if you have adequate resources."[87] "I compare this to the early days of AIDS," says protein chemist Shu Chen, who directs the Center's lab, "when no one wanted to deal with the crisis."[88]

Andrew Kimbrell, the director of the Center for Food Safety, a D.C.-based public interest group, writes, "Given what we know now, it is unconscionable that the CDC is not strictly monitoring these diseases."[89] Given the presence of Mad Cow disease in the U.S., we need to immediately enact uniform active CJD surveillance on a national level, provide adequate funding not only for autopsies but also for the shipment of bodies, and require mandatory reporting of the disease in all 50 states. In Britain, even feline spongiform encephalopathy, the cat version of Mad Cow disease, is an officially notifiable illness. "No one has looked for CJD systematically in the U.S.," notes NIH medical director Paul Brown. "Ever."[90]

The animal agriculture industries continue to risk public safety, and the government seems to protect the industries' narrow business interests more than it protects its own citizens. Internal USDA documents retrieved through the Freedom of Information Act show that our government did indeed consider a number of precautionary measures as far back as 1991 to protect the American public from Mad Cow disease. According to one such document, however, the USDA explained that the "disadvantage" of these measures was that "the cost to the livestock and rendering industries would be substantial."[91]

Plant sources of protein for farm animals can cost up to 30% more than cattle remains.[92] The Cattlemen's Association admitted a decade ago that animal agribusiness could indeed find economically feasible alternatives to feeding slaughterhouse waste to other animals, but that the they did not want to set a precedent of being ruled by "activists."[93] Is it a coincidence that USDA Secretary Veneman chose Dale Moore, former chief lobbyist for the see page 18

National Cattlemen's Beef Association, as her chief of staff?[94] Or Alison Harrison, former director of public relations for the Cattlemen's Association, as her official spokeswoman?[95] Or that one of the new Mad Cow committee appointees is William Hueston, who was paid by the beef industry to testify against Oprah Winfrey in hopes of convicting her of beef "disparagement"?[96] After a similar conflict of interest unfolded in Britain, their entire Ministry of Agriculture was dissolved and an independent Food Safety Agency was created, whose sole responsibility is to protect the public's health. Until we learn from Britain's lesson, and until the USDA stops treating this as a PR problem to be managed instead of a serious global threat,[97] millions of Americans will remain at risk.

REFERENCES:

(Full text of specific articles available by emailing article-request@DrGreger.org)

1 Spokesman Review. 22 September 2003
http://www.organicconsumers.org/madcow/putnam92203.cfm

2 HealthDayNews. 26 September 2003 http://www.healthday.com/view.cfm?id=515265

3 Reuters. 27 December 2003
http://www.organicconsumers.org/madcow/cjd122703.cfm

4 Moyes, Jojo. "Depression Leads to Painful Death." Independent 21 March 1996: 1.

5 "Victims' Families Cry Cover-Up by Protecting Beef Industry, Government Cost Lives, They Say." Miami Herald 26 March 1996: 7A.

6 PA News 30 November 1998.

7 http://meatout.org/

8 Brown, Paul. "Beef Crisis." Guardian 26 March 1996a: 7.

9 British Medical Journal 322(2001):841.

10 Journal of Infectious Diseases 161 (1990): 467-472.

11 Bentor, Yinon. Chemical Element.com - Lead. Jun. 3, 2003.
http://www.chemicalelements.com/elements/pb.html

12 British Medical Journal 322(2001):841.

13 Bulletin of the World Health Organization 70 (1992): 183- 190.

14 http://www.organicconsumers.org/madcow/florida1304.cfm

15 Journal of the American Medical Association, November 8, 2000; 284(18).

16 http://www.bseinfo.org/dsp/dsplocationContent.cfm?locationId=1267

17 "BSE prions propagate as either variant CJD-like or sporadic CJD-like prion strains in transgenic mice expressing human prion protein." EMBO Journal, Vol. 21, No. 23, 6358-6368, 2002.
http://emboj.oupjournals.org/cgi/content/full/21/23/6358

18 United Press International. 29 December 2003.
http://organicconsumers.org/madcow/CJD122903.cfm

19 Proceedings of the National Academy of Sciences 98(2001):4142.

20 "BSE may cause more CJD cases than thought New Scientist 28 November 2002.

21 Journal of Infectious Disease 142(1980):205-8.

22 http://www.aphis.usda.gov/vs/nahps/scrapie/yearly_report/yearly-report.html

23 March 17, 2000 Federal Register (Volume 65,:Page 14521).

See page 19

http://www.mad-cow.org/00/apr00scrapie.html
24 "Sheep consumption: a possible source of spongiform encephalopathy in humans." Neuroepidemiology. 4(1985):240-9.
25 The Veterinary Record 127(1990):338.
26 National Hog Farmer. 15 February 2002.
27 American Journal of Epidemiology Vol. 122, No. 3 (1985), pgs. 443-451.
28 http://www.consumersunion.org/food/psecpi301.htm
29 Creutzfeldt-Jakob disease surveillance in the UK: sixth annual report 1997. Edinburgh, Scotland: National CJD Surveillance Unit,1998.
30 American Journal of Epidemiology Vol. 122, No. 3 (1985), pgs. 443-451.
31 Creutzfeldt-Jakob disease surveillance in the UK: sixth annual report 1997. Edinburgh, Scotland: National CJD Surveillance Unit, 1998.
32 Quarterly Journal of Medicine 93(2000):617.
33 American Journal of Epidemiology 98(1973):381-394.
34 Lancet 1998; 351:1081-5.
35 American Journal of Epidemiology 122(1985)443-451.
36 Lancet 1998; 351:1081-5.
37 Schoon, H.A., Brunckhorst, D. and Pohlenz J. (1991) Spongiform Encephalopathy in a Red-Necked Ostrich, Tierartzliche Praxis, 19, 263-5
38 Journal of Virology 75(21):10073-89 (2001).
39 http://www.nobel.se/medicine/laureates/1976/gajdusek-lecture.html
40 NBC Dateline 14 March 1997.
41 Pearce, Fred. "BSE May Lurk in Pigs and Chickens." New Scientist 6 April 1996: 5.
42 http://organicconsumers.org/madcow/tallow123103.cfm
43 "BSE May Have Caused Some Cases Of CJD As Well As vCJD." The Guardian. 29 November 2002.
44 Lancet 360(2002):139-141.
45 Neuroepidemiology 14 (1995): 174-181.
46 http://www.cdc.gov/ncidod/diseases/cjd/bsecjdqa.htm
47 Altman, Lawrence K. "U.S. Officials Confident That Mad Cow Disease of Britain Has Not Occurred Here." New York Times 27 March 1996: 12A.
48 Flannery, Mary. "Twelve - Fifteen 'Mad Cow' Victims a Year in Area." Philadelphia Daily News 26 March 1996: 03.
49 Neurology 43 (1993): A316.
50 Neurology 44 (1994): A260.
51 Annals of Clinical and Laboratory Science 31(2001):211.
52 Boule, Margie. "Despite Anecdotal Evidence, Docs Say No Mad Cow Disease Here." Oregonian 16 April 1996: C01.
53 Burlington County Times 23 June 2003.
http://www.phillyburbs.com/pb-dyn/news/112-06232003-112425.html
54 Philip Yam. The Pathological Protein: Mad Cow, Chronic Wasting, and Other Deadly Prion Diseases. New York: Springer-Verlag Press, 2003.
55 British Journal of Psychiatry 158 (1991): 457-70.
56 Neurology 38 (1989): 76-79.

see page 20

57 Neurology 39 (1989): 1103-1104.
58 New England Journal of Medicine 310 (1984): 661-663.
59 "Brain Disease May Be Commoner Than Thought -Expert." Reuter Information Service 15 May 1996.
60 http://www.cdc.gov/mmwr/preview/mmwrhtml/00001820.htm
61 http://www.cdc.gov/nchs/fastats/alzheimr.htm
62 http://www.nimh.nih.gov/publicat/numbers.cfm
63 Neurology 34 (1984): 939.
64 The Lancet 336 (1990):21.
65 Folstein, M. "The Cognitive Pattern of Familial Alzheimer's Disease." Biological Aspects of Alzheimer's Disease. Ed. R. Katzman. Cold Spring Harbor Laboratory, 1983.
66 Alzheimer Disease and Associated Disorders 2 (1989): 100-109.
67 Teixeira, F., et al. "Clinico-Pathological Correlation in Dementias." Journal of Psychiatry and Neuroscience 20 (1995): 276-282.
68 British Journal of Psychiatry 158 (1991): 457-70.
69 Mahendra, B. Dementia Lancaster: MTP Press Limited, 1987: 174.
70 Archives of Neurology 44 (1987): 24-29.
71 Neurology 38 (1989): 76-79.
72 http://www.cdc.gov/ncidod/diseases/cjd/bsecjdqa.htm
73 Dementia and Normal Aging, Cambridge University Press, 1994.
74 Neurology 55 (2000):1075.
75 Lancet Infectious Disease. 1 August 2003.
76 http://www.mad-cow.org/jan99_petition.html#ddd
77 http://www.centerforfoodsafety.org/li/CDCrspn1.html
78 Morbidity and Mortality Weekly Report 12 April 1996: 295-303.
79 Neurology 43 (1993): A316.
80 The Wall Street Journal. 30 November 2001.
81 Beacon Journal (Akron). 5 June 2001.
http://www.organicconsumers.org/madcow/CJD6501.cfm
82 New York Times 30 January 2001.
83 http://abcnews.go.com/sections/living/Healthology/HS_autopsydearth_03130.html
84 Altman, Lawrence K. "Four States Watching for Brain Disorder." New York Times 9 April 1996.
85 http://www.medicomm.net/Consumer%20Site/tp/tp_a15.htm
86 http://www.organicconsumers.org/madcow/fact43001.cfm
87 Case Western Reserve University Magazine - Summer 2001.
88 Case Western Reserve University Magazine - Summer 2001.
89 USA Today. 7 January 1999.
90 Philip Yam. The Pathological Protein: Mad Cow, Chronic Wasting, and Other Deadly Prion Diseases. New York: Springer-Verlag Press, 2003.
91 Rampton, S and J. Stauber. Mad Cow USA: Could the Nightmare Happen Here? Common Courage Press; (September 1997):149-50. Full text available free online at http://prwatch.org/books/madcow.html see page 21

92 Food Chemical News 25 March 1996: 30.

93 Food Chemical News 5 July 1993: 57-59.

94 http://www.philly.com/mld/inquirer/5884855.htm

95 http://organicconsumers.org/madcow/usda1204.cfm

96 http://www.prwatch.org/prwissues/1998Q1/oprah.html

97 "World Health Organization says BSE is a major threat" http://www.organicconsumers.org/madcow/BSE7601.cfm

--

Michael Greger, M.D.

Chief BSE Investigator for Farm Sanctuary

http://www.nodowners.org

Mad Cow Coordinator for the Organic Consumers Association

http://www.organicconsumers.org/madcow.htm

(617) 524-8064

(206) 312-8640

mhg1@cornell.edu

185 South St #6

Boston, MA 02130

For periodic updates on the Mad Cow crisis send a blank email to

mailto:DrGregerMadCowUpdates-subscribe@lists.riseup.net

The First McDougall Introductory Weekend
Don't Miss the Next Event!

Sixty people traveled – some more than 3500 miles – from as far away as Alaska, Georgia, and Pennsylvania – to discover the excitement of the McDougall Program during the weekend of January 23 to 25, 2004. Possibly the biggest surprise for them was to have John and Mary McDougall with them all weekend – why would the McDougalls miss the fun, the pleasure of meeting nice people, and the great meals? The dessert table was the most popular, with chocolate brownies, Soy Dream ice cream, carrot cake, and apple crisp with vanilla cream sauce. However, no one got dessert until they finished their minestrone soup, lasagna, salad, sweet potatoes, and salad on Friday evening; and their mashed potatoes, portobello mushrooms, stuffing, green beans, salad, and stuffed mushrooms on Saturday evening.

For lunch on Saturday they enjoyed soup, pizzas and pasta salads; and Sunday lunch was delicious burgers, chili, and grain and bean salads. Even the children who attended loved the meals!

All of the lecture material was new to attendees. Most people had heard other lectures by John and Mary before – so they knew they were going to be well entertained and educated. However, the most pleasant surprise was Dr. Doug Lisle (a doctor of psychology with an important message and a fabulous sense of humor). His three lectures on the "Pleasure Trap" gave participants a clear understanding of why they have been making destructive choices at the dinner table. Our dietitian, Jill Nussinow, presented two outstanding food demonstrations on the basics of the McDougall Diet. Her delicious samples left many people too full to appreciate lunch.

Overall, the weekend was such a big success that most people commented they would be attending the next Alumni Weekend on May 14,15,and 16, and the next Introductory Weekend on July 9,10, and 11 – there is so much to learn and enjoy. Plus, where else can you get such an inexpensive weekend in Northern California? You might have paid this much for one night's room only at a similar resort.

see page 23

Don't Delay! You can get started on a better, trimmer life with the McDougall Program by joining us for a 10-day live-in program with medical care by Dr. McDougall. Dates (2004): February 6-15, March 12-21, April 23 to May 2, June 11-20, and August 13-22.

Or an Introductory Weekend: July 9 to 11, 2004.

Then once you are an alumnus, join us for an Alumni Weekend: May 14-16, 2004.

For prices and more information visit www.drmcdougall.com or call (800) 941-7111 or (707) 538-8609.

Featured Recipes

MINESTRONE SOUP

This is our favorite vegetable soup. It makes a wonderful meal with a loaf of fresh bread. It also reheats well and we like to have it for several lunches during the week.

Preparation Time: 30 minutes

Cooking Time: 3 hours

Servings: 8

1 ¼ cups dried red kidney beans
8 cups water
1 onion, chopped
1 teaspoon minced garlic
1 stalk celery, sliced
1 carrot, sliced
6-8 fingerling potatoes, chunked
½ cups fresh green beans, cut into 1½ inch pieces
1 cup tomato sauce
¼ cup parsley flakes
1½ teaspoons basil
1½ teaspoons oregano
½ teaspoon marjoram
¼ teaspoon celery seed
¼ teaspoon ground black pepper
1 15 ounce can garbanzo beans, drained and rinsed
1 15 ounce can chopped tomatoes
1 zucchini, chopped
1 ½ cups shredded cabbage
½ cup uncooked whole wheat elbows

Place the beans in a large pot with water to cover. Bring to a boil, cook for 2 minutes, turn off heat and let rest for 1 hour. (To eliminate this step, soak beans overnight.) Drain off water. Add onion, garlic and 8 cups of fresh water. Bring to a boil, reduce heat, cover and cook for 1 hour. Add celery, carrot, potatoes, green beans, tomato sauce and all the seasonings. Return to a boil, reduce heat and cook for 45 minutes. Add the garbanzo beans, canned tomatoes and zucchini. Cook for another 30 minutes. Then add the cabbage and pasta and cook for an additional 30 minutes.

Hints: I have many varieties of minestrone that I make throughout the fall and winter months, but this one is our favorite. You may use any type of uncooked pasta that you like, we also like spaghetti broken into 2 inch pieces in this soup. To change the flavor of this soup a bit, stir in a tablespoon or two (or more) of oil free pesto. See the recipe below for a delicious version of Pesto created by the McDougall Program dietitian, Jill Nussinow.

see page 25

PESTO

By Jill Nussinow, RD

Preparation Time: 10 minutes

Servings: makes about ½ cup

2 cups chopped fresh basil leaves
1 cup chopped flat leaf parsley
2 tablespoons pine nuts
1 slice white or sourdough bread or ¼ cup dry bread crumbs
3 cloves minced garlic
1-2 tablespoons light miso (to taste)
2 tablespoons nutritional yeast
¼ to 1/3 cup water

Place everything except the water in a food processor. Pulse until finely minced. With machine running, slowly add water until desired consistency is reached.

Hints: Coarsely chop the basil and parsley because the food processor will mince them later. Add some blended silken tofu to make a creamy pesto sauce for pasta.

POTATO SOUP

By Marilyn Borton

Marilyn is a past participant in The McDougall Program in Santa Rosa, CA. She sent me this recipe and said it is an old favorite that she has been making for years. I made it a few days ago for John and myself and we loved it! The potatoes break up a bit while cooking to make a rich, creamy broth. We also enjoyed the leftovers for lunch over the next couple of days.

Preparation Time: 15 minutes

Cooking Time: 45-60 minutes

Servings: 6-8

6 cups vegetable broth
1 onion, chopped
1-2 cloves garlic, minced
6 medium russet potatoes, peeled and chopped
2 carrots, chopped
2 stalks celery, chopped
1 zucchini, chopped
½ cup chopped fresh parsley
1 teaspoon dried dill weed
¼ to ½ teaspoon freshly ground black pepper
dash salt

see page 26

Place a small amount of the vegetable broth in a large soup pot. Add the onion and garlic. Cook, stirring occasionally for 5 minutes. Add the remaining ingredients, mix well, bring to a boil, reduce heat and simmer for about 45 minutes, until vegetables are tender and potatoes have started to break apart. Serve with some fresh bread to dunk into the flavorful broth.

CARIBBEAN VEGETABLE STEW

This spicy bean stew reheats well so it can be made ahead and refrigerated until needed for mealtime.
Preparation Time: 35 minutes
Cooking Time: 60 minutes
Servings: 8-10

1/3 cup water
1 large onion, chopped
1 red or green bell pepper, chopped
3 cups peeled & chunked sweet potatoes
1 15 ounce can tomato sauce
1 15 ounce can diced tomatoes with jalapenos
1 20 ounce can pineapple chunks with juice
2 cups chunked green apples (unpeeled)
1 4 ounce can chopped green chilies
½ cup vegetable broth
1 15 ounce can pinto beans, drained and rinsed
1 15 ounce can black beans, drained and rinsed
1 15 ounce can kidney beans, drained and rinsed
2 tablespoons brown sugar
2 teaspoons chili powder
1 teaspoon ground cumin
1 teaspoon ground oregano
¼ teaspoon cinnamon

Place the water in a large pot. Add onion and bell pepper. Cook, stirring occasionally, for 5 minutes. Add sweet potatoes, tomato sauce, tomatoes, pineapple, apples, chilies and vegetable broth. Bring to a boil, reduce heat, cover and cook over low heat for 45 minutes, stirring occasionally. Add beans and seasonings, mix well and continue to cook over low heat for another 10 minutes, stirring occasionally. Serve over brown rice or other whole grains.

BARBECUE TOFU WRAPS

This may be made ahead and reheated just before serving. It is wonderful as a leftover for lunch the next day or two.
Preparation Time: 20 minutes
Cooking Time: 10 minutes
Servings: 8-10

see page 27

16 ounces firm, water packed tofu (not silken)
2 teaspoons ground cumin
2 teaspoons chili powder
3 teaspoons rice vinegar
½ cup vegetable broth
1 onion, chopped
1 red bell pepper, chopped
1 cup frozen corn kernels, thawed
1 15 ounce can black beans, drained and rinsed
1½ cups cooked brown rice
1 cup barbecue sauce
8-10 fat free flour tortillas

Drain the tofu and cut into small cubes. Place in a shallow bowl and sprinkle with cumin, chili powder and vinegar. Stir gently to mix. Set aside, mixing occasionally.

Place the vegetable broth in a large non-stick frying pan. Add onion and bell pepper. Cook, stirring occasionally, for 5 minutes. Stir in corn, black beans, brown rice and barbecue sauce. Cook for an additional 2 minutes. Add tofu and continue to cook about 3 minutes longer, stirring gently when needed.

Warm tortillas. Spoon about ½ to ¾ cup of the mixture down the center of the tortilla. Fold up bottom of tortilla, roll up sides and eat.

Optional: Set out bowls of shredded lettuce, chopped tomatoes, chopped onions and additional barbecue sauce or salsa. Another option would be a small amount of bean guacamole (Quick & Easy Cookbook, page 262) or broccomole (New McDougall Cookbook, page 116). Top the tofu-bean mixture with any of these items before rolling up.

Variation: Use pinto or white beans instead of the black beans.