A WORD FROM THE NEWLY WISE!

I don't like to use scare tactics, but I'm putting out warnings on aspartate (found in the sweetener NutraSweet® and monosodium glutamate (MSG). The 1994 book EXCITOXINS by Russell Blaylock, M.D. has truly stirred up a storm (Health Press, PO Box 1388, Santa Fe NM 87504). Blaylock, a neurosurgeon, offers evidence that glutamate and aspartate in the unnatural amounts consumed today are destroying neurons. They're perfectly okay amino acids as parts of food molecules. Commercial development, however, has encouraged their consumption in very large amounts as separate amino acids, and that's where trouble begins. MSG became a major product in Japan in this century after chemists isolated the molecule in Kombu, a seaweed, that makes everything taste better. Kombu itself had been added to dishes for hundreds of years without harm. The Ajinomoto Co. sells MSG worldwide now. After WW II, U.S. food giants began adding it to their products, including baby foods, for its taste-enhancing effects.

Beginning in 1957, some unexpected, scary results showed up when this 'harmless' amino acid was given to infant mice: it destroyed all of the nerve cells in the retina of the eyes. Later experiments by John W. Olney, M.D., a neuroscientist, showed that a single dose of MSG destroyed brain cells in the hypothalamus of infant mice. Blaylock writes: "the concentrations of MSG found in baby foods was equal to that used to create brain lesions in experimental animals."

Although the FDA refused to act on his findings, Dr. Olney's testimony before a Congressional committee caused food manufacturers to agree to remove MSG from baby foods.

They replaced it with "hydrolyzed vegetable protein," a term familiar to us all and having only benign connotations, right? It so happens that the same Ajinomoto Co. makes most of the world's supply. It's a brown powder containing "three known excitotoxins -- glutamate, aspartate, and cysteic acid... It is then added by the food industry to everything from canned tuna to baby food." Many "health foods" are loaded with it! The fear expressed by Blaylock and other neurologists is that widespread use of "naked" excitotoxins may be contributing not only to brain damage in children but aggravating adult brain disorders such as Parkinson's and Alzheimer's disease.

Aspartate also makes up 50 percent of NutraSweet® (Equal® or aspartame). "Like glutamate, aspartate is a powerful brain toxin which can produce similar neuron damage," Blaylock says. As we know, NutraSweet is now the calorie-free sweetener of choice in soft drinks consumed by adults and children.

It also forms methyl alcohol, i.e., wood alcohol, especially in hot liquids. When people drink wood alcohol by mistake, they can go blind, because it destroys the optic nerves. Julian Whitaker, M.D., in his newsletter Health & Healing, December 1994, tells of a pilot who "experienced such severe blurred vision while flying that he couldn't even read the instrument panel and barely averted a crash landing. This occurred two hours after he inadvertently drank two cups of aspartame-sweetened hot chocolate. He has consumed no aspartame since, nor has he had any blurred vision." (The book Aspartame (NutraSweet®): Is It Safe? by H. J. Roberts, M.D., Charles Press, 1980, documents hair-raising cases, ranging from vision loss to seizures.)

That evening, my sight returned to normal. Until I read the December Health & Healing, I didn't have a clue. After I read it, I threw out my large, economy-sized boxes of NutraSweet in the trash. (Soon after, I gave up coffee, but that's another tale, told in FL#81.)

"Optic nerve decompression surgery," is commonly performed for a type of sudden vision loss that strikes up to 6,000 Americans yearly, according to a recent news item. While useful for certain other eye problems, the surgery may cause worse vision in this particular condition. This was the conclusion of a study by the National Eye Institute, reported in JAMA, Feb. 22, 1995. This type of optic nerve damage is "the most common cause of sudden vision loss in the elderly," the report stated, "usually striking people in their 60s or 70s, producing sudden blurring or blind spots."

Do you suppose this could be related to too many years of consuming excitotoxins? I wonder.

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TRANS-FATS, COURTESY OF CSPI

For thousands of years, people ate coconut and palm kernel oils. The fats were safe, plentiful and easy to extract, e.g., in Southeast Asia, palm fruit was boiled in large pots of water and the oil skimmed off the top. Sure, they were saturated, but folks got their essential w6 and w3 poly's from greens, seeds, and seafood. Food processors in the 20th century used coconut and palm kernel fats freely because they were cheaper than butter and less apt to go rancid.
Enter the American Soybean Assoc. and Michael Jacobson of Center for Science in the Public Interest (CSPI). They joined in a campaign about 15 years ago to replace the tropical fats with polyunsaturated soybean oil -- for the heart's sake. *Nutrition Action*, the CSPI magazine, went after tropical fats with a fury, proclaiming victory after victory as, one by one, major food processors and restaurant chains switched from coconut and palm kernel to "polyunsaturated" oils. Dietetic and medical spokespeople applauded these efforts as "heart-healthy."

The upshot is that it may be decades before the food industry goes back to trans-free tropical fats. I'm feeling some urgency about this, ever since learning from Dr. Enig of their health-giving qualities. Tropical oils do not cause a rise in blood cholesterol when added to normal diets. They have a large complement of easily digested fats known as medium chain triglycerides (MCTs). MCTs are given to premature infants and patients who can't digest ordinary fats. In addition, tropical fats are rich in a fatty acid, lauric acid, which has natural antiviral, antibacterial properties. (Nature puts lots of lauric acid into breast milk.) Of course, they don't supply w3s/w6s, which I hope we're smart enough to get from foods like flaxseed, flaxseed oil, whole grains, walnuts, and plenty of seafood!

Tropical fats don't get rancid, are excellent for frying, and make pie crusts flaky and crackers crispy. For your home bakers, health food stores sell organic coconut butter. The food industry would be doing people a favor if they dumped hydrogenated fats and reinstated the tropicals. Granted, nobody benefits from habitually eating fat-filled commercial foods. At the very least, though, these foods should be trans fat-free, to lower the potential for harm!

"Ernest, let's talk about your carbohydrates."

Mary G. Enig, Ph.D., probably knows more about trans-fatty acids than any scientist on earth. She knew that the polyunsaturates being substituted for tropical fats were being hardened by chemical means--hydrogenation--in order to provide the same desirable characteristics as before to cookies, crackers, fried foods, etc. Polyunsaturates were being transformed into trans fats. During her years at the University of Maryland, she and her research group did tedious evaluations of trans fats in margarines, shortenings, and thousands of commercial foods. They tested animals, gathered statistics, and started the long, uphill, unpopular task of warning the medical community that trans meant Trouble with a capital T. Naturally, it went over like a wet balloon.

Enlightenment came late, mostly through solemn European studies that broke in major newspapers, linking these machine-made fats to increased heart disease and cancers. Long after trans fats had become a dirty word, though, *Nutrition Action* went on denying trans were anything but benign. Only when everybody, even stalwart margarine champion Jane Brody, came down hard on trans, did CSPI concede they were bad, but blamed the food industry for using hardened shortening instead of pure oils -- which was a crock, because they'd gone along with this approvingly for years!

Women are especially at risk of EFAI. The huge social pressure for women to be slim combined with their lower metabolic rates (and potential greater EFA needs with pregnancy), lead many women to diets very low in EFAs.

"The problem of EFAI... is compounded by the use of hydrogenated oils, leading to elevated concentrations of circulating trans fatty acids, which we have shown to be associated with the risk of coronary heart disease."

(Am J Clin Nutr, Dec 1994; 70:973)

Remember, folks, a diet that's low in w6 EFA is bound to be awesomely deficient in w3s.

SICKLE CELL, 1932 & TODAY

A strange silence about thiocyanate therapy blankets the sickle cell medical community. I feel like Don Quixote tilting at windmills as I endeavor to whip up interest in testing thiocyanate's potential in treatment.

As described in earlier newsletters (FLs 71, 72, 75), children and adults in the U.S. who've inherited sickle cell (SC) genes from both parents may have severe anemia and be hospitalized often for infections and great pain. Yet studies as far back as the 1950s had no explanation for the enormous variations in seriousness of the disease, which affects all races and, oddly enough, provides some protection from malaria. I learned this when I prepared a report for a food chemistry class in 1977, my last year at U.C. Berkeley. In a stunning paper, Robert G. Houston (Amer J Clinical Nutrition, 26, Nov 1973) had proposed a nutritional connection to SC, explaining why symptoms could be severe, mild, or nonexistent. Wherever people with a double set of SC genes ("homozygotes") ate traditional indigenous foods that naturally generated thiocyanate in their bodies, he said, SC illness tended to be mild or even absent. These foods kept their blood thiocyanate level high -- as much as 40 times higher than in SC homozygotes who didn't eat these foods and had severe sickling illness. Thiocyanate, which normally is found in everyone's plasma and saliva, looked like Nature's antisickler of choice!
Dr. Linus Pauling, whose early pioneering work on the SC hemoglobin molecule set the stage for subsequent studies on hemoglobin disorders, had suggested to Houston in 1973 that, indeed, thiocyanate should be an effective antiscisker. By virtue of its ability to break apart hydrogen bonds, thiocyanate could prevent hemoglobin molecules from forming the rigid crystal polymers in red blood cells that characterize sickle cell disease.

In 1977, I chased down Huston’s references and accumulated dozens more, all leading me towards the same observation: Thiocyanate was safe and looked like a winner. (Years later, I found papers by anthropologists whose field work in tropical countries confirms his theory in spades.) It was only a matter of time, I thought, before formal studies would begin if they hadn’t already. Soon, SC patients would be encouraged to eat tasty thiocyanate-yielding foods daily (See lists in F1s 69, 71, 72, 75). Seriously ill patients would get potassium thiocyanate as medication. Potassium thiocyanate was commonly prescribed in the 1930s and 40s for high blood pressure and certain kinds of dysentery. Although blood levels needed to be checked periodically, the medication had a good safety record. I gave my report to the class, stored my stack of files in the basement, and went on to other projects.

I got a wake-up call in 1993. Newspaper stories about a “promising” new treatment for SC, a butyrate compound developed by researchers at Children’s Hospital in Oakland, stressed the hopeless aspects of the disease, the suffering it caused in children, let alone adults. A year later, Bay area newspapers and television stations carried a plea for donors of a rare blood type to save the life of a boy desperately ill with SC for whom no treatment was effective. Now, in 1995, the “new breakthrough drug” being touted is hydroxyurea. Reports describe grim side effects, saying it must be used with constant medical monitoring and only in people with severe disease.

W ait a minute, guys! I thought you scientists had solved the problem 15 years ago with thiocyanate medication and diet! I hit the paper trail again, scoured the U.C. libraries and the lists of studies on a variety of experimental therapies (including sodium cyanate, a molecule related to thiocyanate but far more toxic), but nothing

on thiocyanate. Eventually, I linked into a small network of good people whose work I’ve described earlier. They have seen amelioration of anemia and illness in SC homozygotes when a daily high thiocyanate-yielding diet is eaten, a potassium thiocyanate + iodine supplement is given, or a combination of supplement and diet is used. No side effects, either.

In general, treatment for sickle cell then, as now, was mostly palliative, including the use of transfusions, liver extracts, and drugs to relieve pain. (Some doctors were removing spleens, but the authors note that this approach had not proven useful in the long run.) During the young man’s last hospital stay in 1931, every measure to relieve his pain was used, including placing him in an oxygen tent, administering salicylates, nitroglycerin, atropine sulphate, etc. “Throughout his period of residence in the hospital and during his attacks of periodic pain, it was difficult to make this patient comfortable, even by the use of morphine sulphate in fair-sized doses given at frequent intervals.”

Now comes the novel approach, born of desperation. The doctors knew that RBC’s in vitro promptly sickle if the solution is made more acidic. To prevent systemic acidity in the patient, they were giving him daily doses of potassium citrate and bicarbonate of soda, even though it didn’t stop the pain. (As a matter of fact it makes sense, for the simple reason that hemoglobin itself is an important acid-base buffer. In the case of diminished or sickled hemoglobin, there would be a real loss of acid buffering power.)

The decision to use “sulphocyante” (an early name for thiocyanate) was a gamble, based on animal experiments where thiocyanate administered after anesthesia permitted tissues to return much more rapidly “to its previous normal colloidal state,” they wrote.

Beginning on February 19, 1931, the patient received large daily doses of potassium citrate and sodium bicarbonate. He had previously received these two preparations with no favorable results, but now there was added one and one-quarter grains of potassium sulphocyanate given three times a day. On February 23, this dosage was increased to two and one-half grains three times a day and this medication was continued until March 6, 1931. During this time he was entirely free from pain and discomfort and was up and about the ward. He remained free from pain until March 11, 1931, when he again complained of pain in the shoulders and abdomen. Immediately on receiving the potassium sulphocyanate therapy there occurred again a prompt relief of symptoms....He felt unusually well and on being denied the privilege of leaving the hospital in the regular way, he absconded April 16, 1931.”

The doctors conclude: “Although the matter would seem to rest on some highly theoretical grounds, there seems to be some justification for believing that there is a rationale in using a thiocyanate salt with other salts of sodium and potassium under the circumstances which we assume to present themselves during the critical abdominal attacks as they occur in sickle cell anemia. …Further trials and observation are necessary before any conclusions may be reached.”

1. Since each red blood cell (RBC) is packed with hemoglobin molecules, the accumulation of abnormally long hemoglobin crystals distorts the round or oval RBC into a weird, elongated sickle shape. RBCs are normally ‘deformable,’ i.e., elastic, to ensure safe voyage through tortuous networks of tiny capillaries. Sickled RBCs, in contrast, are too brittle and fragile for the journey. They disintegrate early, leading to chronic anemia. They also tend to clag blood vessels, causing damage to tissues by depriving them of oxygen. Hemoglobin, as we know, is the oxygen carrier, via the bloodstream, to every cell in the body.

2. William E. Richardson, M.D., runs the Atlanta Clinic of Preventive Medicine, 1718 Peachtree St. N.W., Suite 2, Atlanta, Georgia 30309. Tel: 404/607-0570. Biochemist Dr. Oji Agbai Chima wrote Sickle Cell Anemia: A Solution at Last. It can be ordered from Dr. Abagai for $30. Write to Biochemical Research Institute, 2010 S. Nogales, Tulsa, Oklahoma 74107. Chef Dawud Ujmaan wrote a wonderful cookbook of thiocyanate-yielding foods, Back to our Roots, Cooking for Control of Sickle Cell Anemia, and has a video series as well. Write him for information at 103 Eastwyck Rd., Decatur, Georgia 30032; or call him at 404/243-1316.
Plant lignans protect seeds against fungi, viruses, and bacteria and may also exert these effects in the human intestine. Flaxseed is the richest known producer of mammalian lignans.

The authors say there's been some concern that cyanogenic glycosides in flaxseed may increase serum thiocyanate concentrations and thus pose a health threat, but then assure us that cyanide exposure in response to doses of raw flaxseed as high as 60 grams "is not hazardous to healthy individuals."

Dear people, potassium thiocyanate was a medical treatment not only for high blood pressure but for certain forms of dysentery in the 1930s and 40s, as noted. I suspect more health problems come from too few, rather than too many "cyanogenic glycosides," i.e., thiocyanate-generating foods, in the many fabricated concoctions that pass for vituals today!

Canada's Contributions

Our good neighbor to the north has been putting out marvelous work. Stephen Cunnane, Ph.D., and colleagues at the University of Toronto tested the effects in 10 healthy young men and women of consuming 50 grams of flaxseed a day (Am J Clin Nutr. Jan 1995; 61:62-8). (Ooh, that's a lot of flaxseed! My gut rumbles just thinking about it.) In addition to their normal diet, for four weeks the subjects consumed either two muffins a day containing 25 grams of flaxmeal each, or two identical "control" muffins minus flaxmeal. After a muffin-free "washout" period, they switched sides and continued eating muffins for another four weeks.

The following benefits were seen during the flaxseed but not the control period:

- Total and LDL blood cholesterol decreased 6% and 9% respectively.
- Bowel movements per week increased by 30%. This may have helped to lower cholesterol by causing more bile acids, which are formed from cholesterol, to be excreted.
- Urinary lignan excretion rose more than fivefold—an indication of a huge rise in mammalian lignan production.

To test flaxmeal's effects on w3 fatty acid blood levels, the subjects were not allowed to eat any fish for two weeks before and throughout the experiment. As expected, because of flaxseed's very high alpha-linolenic acid (ALA) content, this w3 fatty acid rose significantly in the blood during flax-muffin time, while declining during control periods. EPA and another important long-chain w3 derived from ALA also increased. However, no rise in w3 DHA was seen. (Fish, of course, contribute to desirable higher DHA levels.)

Fears about increased lipid peroxidation in tissues (because of consumption of ALA at about nine times the North American intake) were put to rest. The lignans from flaxseed actually may have contributed to antioxidant protection.

All in all, the authors were satisfied that flaxseed, in the large amounts used in the study, demonstrated "beneficial nutritional attributes" as well as being safe. This is all to the good because, they say, "high production of mammalian lignans by humans may lead to protection against cancer and perhaps other diseases." Examples from recent animal studies include reduction by flaxseed of precancerous abnormalities in gut cells in rats, flaxseed inhibition of "cell proliferation and nuclear aberration in rat mammary epithelial cells," and reduced tumor size at the tumor-promoting stage of cancer.

If all of this pushes your buttons, you may wish to send away for a little pamphlet by The Flax Council of Canada: "A Taste of Flax: The Cookbook." Recipes for muffins, cookies, scones and bread all have flaxmeal as a star ingredient. Send request to them at 465-167 Lombard Avenue, Winnipeg, Manitoba, Canada R3B OT6. (Postage from U.S. is 40 cents.)

Bay area folks: Save Sunday afternoon on May 21. John R. Lee, M.D. will be talking about Natural Progestrone: The Multiple Roles of a Remarkable Hormone. His book and lectures are responsible for a phenomenal grass roots movement that's beginning to reach other clinicians. The talk, beginning at 1 p.m. with question period afterwards, is sponsored by the Alameda League of Women Voters. It's at the Alameda High School Little Theater, Walnut & Central in Alameda, opposite the Veteran's Bldg. $5 donations to the League accepted. See you there!

Illustrations by Clay Geerdes and other artists as noted.

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