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Is the Bugle Blowing Taps for Sickle Cell Anemia???

Well, sure, if I were a sickle cell scientist I'd be plenty miffed too, if some upstart biochemist from Tulsa threated to collapse my research edifice like a house of cards! I'm not naive enough to expect the medical folks to embrace with rapture the somewhat unnerving concepts laid out by Oji Agbai PhD ND at the 27th annual conference of the Sickle Cell Disease Assoc. of America in Cleveland, Ohio, September '99.

.But something wonderful happened there. Aficionados of a hi-tech approach to sickle cell treatment may yet be nudged, albeit grudgingly, into a U-turn to the past--to a Philadelphia hospital in the 1930s where an ingenious discovery was made by two doctors. More on this later.

Sickle Cell in Brief

HERE'S A quick summary of this hereditary condition, found in all races all over the globe: Each red blood cell (rbc) in your body is packed with about *1 million hemoglobin molecules which deliver oxygen to your tissues and organs.* (Hemoglobin also is the major acid-base buffering agent in blood.) Sickle cell babies inherit two genes--one from each parent--coding for an amino acid substitution (valine instead of glutamic acid) in hemoglobin, *thereby altering its electric charge.* No small matter -- we're talking about an effect taking place in *one million hemoglobins per rbc.**

Electrophoretic mobility of hemoglobin--its motion in aqueous solution in an applied electrical field--is measurable and is in fact one way to analyze blood to identify who is or isn't a sickler. One result of the change in electric charge: in common physiological conditions such as high acidity or low oxygen, the sickler's hemoglobin may polymerise into long, rigid crystals.

*Humongous numbers here: total blood volume is around 5000 milliliters (over 5 quarts). Each milliliter of blood has about 5 million rbc's, and each rbc contains a million hemoglobins. You're welcome to do the multiplying!

This is Linus Pauling's description in 1952 of sickled rbc's:[1]
"They are twisted into crescent or sickle-like shapes, with longest dimension considerably greater than that of the normal cell...and they are quite rigid -- the normal cell is almost jelly-like in its flexibility, but when sickling occurs the cell loses this flexibility, so that it has been described as appearing to be as rigid as a crystal of ice...These distorted cells, which seem also to be sticky, have difficulty in passing through capillaries, many of which are so small as just to allow passage of normal erythrocytes in single file. When sickling becomes enhanced in a crisis of the disease, the capillaries become jammed with red cells, and the flow of blood is prevented..."

Sickled rbc's are fragile, tending to disintegrate in as little as *10 to 12 days,* instead of the usual 120. Stark anemia ensues, creating heavy overwork for the bone marrow's blood-making function. The list of catastrophic disorders engendered by any or all of the above effects is impressive.

So little progress!

The wheels of medical research sometimes move with agonizing slowness, but I'm hard put to understand how a cheap, noninvasive treatment for sickle cell anemia which worked spectacularly in the case below, documented in 1932, has been ignored ever since by authorities in the field.

It's not as if they've found abiding answers in the intervening decades. Somber reviews in journals continue to stress high morbidity, agonizing pain, and crippling strokes inflicted on sicklers *beginning in infancy.* Even after escaping the high mortality prevalent in early childhood, sicklers have a slim life expectancy: 42 years for men, 48 years for women.

To stave off frequent life-threatening infections, daily antibiotics commonly are given to infants and young children. Unfortunately this encourages new resistant strains of pathogens. Biweekly blood transfusions are standard therapy for many young sicklers. Current treatment favorites--hydroxyurea, bone marrow transplantation, gene therapy--offer benefits peppered with risks and/or require hi-tech, invasive, costly procedures.

A White-Coated Black Knight!

Enter Dr. Agbai, an assimilator of information gathered by MDs, anthropologists, and social scientists[2-8] as well as gleaned from his own background growing up in West Africa, a member of the Igbo people. Although many home folks were homozygous for sickle cell, i.e., had the gene from each parent, he tells me crippling disability and early death were rare.

A diet connection started to make sense to him after he came to the U.S. and learned how severe symptoms were for African-Americans, whose standard foods had almost no relationship to those in his native land. *There, foods providing thiocyanate and/or sublethal amounts of cyanide, such as African yam, cassava, millet, and sorghum grain, were traditional staples.*

Looking further for answers to the puzzle, he found a 1968 paper by Graham Serjeant MD et al[2] describing relatively benign aspects of sickle cell in Jamaica despite homozygous sickle genes in 11% of the population. Agbai knew that manioc (cassava), indigenous to the West Indies as well as Central and South America, was a major food along with true yams (*Dioscorea* species) and a variety of beans, all contributing to high yet nonlethal plasma levels of thiocyanate in the population. A common incident, puzzling to Serjeant's group, involved Jamaicans who became sick and anemic only after they emigrated to the States where their sickle cell disease was then diagnosed. On returning home to the West Indies [and, Agbai assumed, resuming their former diet], most recovered without further need for treatment.

Thiocyanate Experiments

He was a chemistry professor at Univ. of Arkansas at Pine Bluff in the late 1980s when he began speaking at health conferences, community groups, churches, etc. about an effective approach to sickle cell anemia. *His case studies showed consistent improvement in sicklers who regularly consumed nutrients known to raise thiocyanate levels.* In test-tubes, he proved that potassium thiocyanate itself completely blocked sickling of rbc's.

Thiocyanate [thio = sulfur from the Greek *theion*] *is a normal constituent of plasma, saliva, urine, etc.* Apparently many of earth's creatures, including ourselves, possess skillful enzymatic mechanisms to hang a sulfur molecule onto sublethal amounts of cyanide, transforming cyanide into thiocyanate. This is not a trivial protection, since at least 1000 species of plants are known to be capable of releasing cyanide when consumed, i.e., are cyanogenic.

Thiocyanate alone is found in many plant foods, e.g., broccoli, collard greens, cabbage, while a great many edible plants and seeds contain both cyanide and thiocyanate. (Flaxseed is a good source. I ate at an Ethiopian cafe in Oakland that offers a number of traditional dishes in which flaxseed is one of the ingredients.)

At optimal levels thiocyanate not only isn't toxic, but appears to play useful but currently under-explored roles in the body.

Back in the Philadelphia hospital almost 70 years ago....

Two MDs, Edward Torrance and Truman Schnabel,[8] tried everything they could to relieve their 26-year-old "Negro" sickle-cell patient's "excruciating pain" during his fourth admission in 5 years, including placing him in an oxygen tent, administering salicylates, nitroglycerin, atropine sulphate, etc. Even morphine "in fair-sized doses given at frequent intervals" didn't help enough.

Pickles/Brian Crane

To lower the patient's systemic acidity--a known stimulus for increased sickling--the doctors were giving him daily doses of potassium citrate and bicarbonate of soda, even though this didn't stop his pain Out of desperation, on February 19, 1931, the doctors *also added potassium thiocyanate three times a day* (they called it "potassium sulphocyanate"), continuing with a total of 500 milligrams daily until March 6.

"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."

My Paper-Chase Trail

In 1977, my last semester at UC Berkeley, I first learned of the Torrance & Schnabel study from Robert G. Houston's[3] trailblazing 1973 article while working on a report for a food chemistry class. If I was able to hunt it down in a university library, so could any researcher in the field. Why had it not been followed up???

But I didn't realize this was the case until 1993, when I first became aware of news releases about "promising" new treatments (hydroxyurea, bone marrow transplants, etc.) to ease the suffering of sicklers, most of whom were children and adolescents. Naive me -- I'd thought the problem had long been neatly solved by thiocyanate treatment!

When I then searched for medical reports on thiocyanate therapy in sickle cell, *I found not one since 1932.*

Anthropology studies produced further affirmation or acknowledgment of the thiocyanate theory. There was Haas & Harrison's[4] paper in 1977 describing a "possible role of cassava (manioc)-based diets in providing a buffer against the effects of sickle-cell anemia" in environments *where sickling provided protection against malaria.* Solomon H. Katz[5] in 1987 referred to medical studies where *cyanate* was an effective anti-sickler (Cerami, 1974)[6], noting that widespread intake of manioc (cassava) seemed to correlate with regions where sickle genes abounded and where malaria was a serious threat. He speculated that cyanate from manioc might ease sickling, as well as blunt the effects of malaria by interrupting the malarial parasite's life cycle in rbc's.

On Nov. 10, 1981, *S.F. Chronicle* science editor David Perlman wrote about 32-year-old Stanford Univ. anthropologist William H. Durham, whose forays into malaria-ridden lands of West Africa convinced him that strict cultural rituals connected to the planting and eating of African yam--a very rich source of thiocyanate--served to protect sicklers against both anemia and malaria.

Bay area newspapers in August of 1982 ran stories on two UC Berkeley faculty members, husband and wife Robert T. and Linda Collier Jackson, who led a field team of scientists and students in Liberia (West Africa) to probe a "cyanide link to health...Ms. Jackson said that cyanates from cassava appear to normalize sickled blood cells and may also prevent malaria parasites from thriving in the blood."

More Evidence

In 1988 Linda C. Jackson[7a] and fellow anthropologists at the Univ. of Florida at Gainesville did an unusual study on miniature pigs: they gave them *sublethal amounts of cyanide* along with balanced feed. Cyanate and thiocyanate both have the capacity to "carbamylate" hemoglobin, i.e., to bond with hemoglobin's terminal amino acids. Carbamylation has been shown to increase the life of rbc's and to decrease sickling, but the doses of cyanate used clinically for these effects were much greater than amounts available from ordinary intake of cyanogenic foods.

Their purpose was to find out "whether significant hemoglobin carbamylation could still occur in the presence of chronic dietary-derived cyanide at the sublethal levels commonly ingested by millions of Liberian (West African) children and adults."

The results? Yes, by gosh, carbamylation of hemoglobin in their rbc's took place readily when the little swine ingested small, nontoxic amounts of cyanate and thiocyanate. The piggies, whose systems closely resemble our own in major respects, suffered no harm from the doses.

A Theory on Why Sicklers Stayed Alive...

Later, in a complex 1990 paper Prof. Jackson[7b] suggested that survival rates for homozygous sicklers over a 400-year period might be related to long-established varied intakes of cyanate- & thiocyanate-rich cassava (manioc) in different regions of Liberia. (Cassava was introduced in the 1600s from South America.) She proposed that sicklers may have had a slight edge in surviving malaria in areas of *low* cassava intake because the parasite *Plasmodium falciparum* can't complete its life cycle in sickled rbc's. As long as the sicklers avoided a disabling anemia by consuming enough cassava to keep an adequate percentage of their rbc's unsickled, she theorized that many could reach adulthood and pass on sickler genes to their kids.

...and Malaria Parasites Died!

On the other hand, Jackson suggested that *nonsicklers* may have learned to lessen malaria's morbidity by staying on the *very high* cassava intake that's traditional in specific regions of Liberia. It seems that high but sublethal cyanate-thiocyanate blood levels in people *are semitoxic for the parasite*, interfering with its survival!

Back to Cleveland and the Conference

Dr. Agbai's research paper was entitled "Anti-sickling Activity of Nutritional Thiocyanate." A deeply religious man, he explained to me in our phone conversation a few days later that before delivering the technical stuff, he told the audience that "God, or Divine Wisdom, known as 'Chima' in the Igbo language, provided sickle cell hemoglobin to protect humans from deadly malaria, and provided nutritional thiocyanate in so many tasty foods to protect sickle cell homozygotes from anemia and illness."

He then went on to tell attendees and fellow-presenters of his *in vitro* work, showing transparencies of scanning electron micrographs of sickled rbc's, contrasted with rbc's fully protected from sickling by potassium thiocyanate (KSCN). KSCN markedly increased solubility of sickler hemoglobin, inhibiting formation of liquid crystals that distort rbc's into sharp sickle shapes.

He showed KSCN to be more effective in this than cyanate, which had been explored extensively in the early 1970s by Cerami, Manning, Gillette, etc.[6], but discarded because longterm use caused serious nerve damage in some patients. (These same scientists, for unfathomable reasons, never went on to explore the possibility that *detoxified cyanate, i.e., thiocyanate,* might accomplish the same goals *safely.*)

Pickles/Brian Crane

Now at last we come to the small miracle that took place in the Cleveland auditorium. Lennette Benjamin, M.D.* convention chair and new chief medical officer of the Sickle Cell Disease Assoc. of America (SCDAA), had planned the 27th annual convention with a special focus: it was to be a forum for *patients and parents*, as well as health care providers, administrators, advocates, researchers, etc. *(SCDAA Sickle Cell News,* Summer 1999.) I'd received their publications since 1993 and never noticed any discussion of thiocyanate-based nutrients, either as desirable foods or as treatment possibility. Apparently, Dr. Agbai's paper was to be a first.

An Unprecedented Outpouring

Maybe his findings were new information to the medical folks at the meeting, but it seems it wasn't to many in the large contingent of sicklers and their families there. When he finished his talk, a startled Dr. Agbai and other panel members witnessed one person after another from the audience getting up and taking the microphone, first to relate bleak medical histories, their own or their children's, despite conventional treatments, and then to tell what happened after they had finally heard, read, or been told of Agbai's work. They were there to testify, in many cases, to *years* of painfree, productive life since making a daily habit of thiocyanate nutrients.

While these public testimonials came as a pleasurable shock to Agbai, in actuality he had been reaching community, church, and educational groups since the late 1980s. Over the years he'd received many letters from educators, leaders, and, above all, recovered sicklers or their parents. Some very moving ones are in his book, ***Sickle Cell Anemia: A Solution At Last,**** first published in 1987 and periodically updated. In it are scanning electron microphotographs of red blood cells (from patients) sickling in a saline solution, then of rbc's protected from sickling by KSCN in the solution. The book explains thiocyanate's protective actions, combining theory with test tube evidence *plus* convincing real life stories.

*Sheis associate professor of medicine and clinical director of Albert Einstein College of Medicine, Montefiore Hospital Medical Center, Bronx, NY.

Chef Ujamaa's Remarkable Cookbook

Not only had Agbai's many talks over the years gotten favorable writeups in local newspapers, but his work had inspired Chef Dawud Ujamaa, television star of "The Cooking Man Show" in Atlanta, Georgia, to produce a book and video series in 1993. ***Back To Our Roots: Cooking for Control of Sickle Cell Anemia & Cancer Prevention***** is an enchanting mix of scientific theory and down-to-earth how-to's on identifying and preparing high-thiocyanate foods to keep the illness at bay -- for life. Believe me, there's nothing like this invaluable guide anywhere.

An MD who treats with thiocyanate

Another quiet hero in our story is William E. Richardson MD of Atlanta, a clinical physician who wrote a foreword and several sections in Ujamaa's book and for years has been steering sickle cell patients towards the nutrients he too firmly believes are provided by nature to stave off anemia and illness.

Five years ago, after I found in anthropology and biochemistry papers intriguing backup for Agbai's concepts, I wrote to a bevy of sickle cell researchers. The few who bothered to reply were immersed in their own work and had no desire to get involved in thiocyanate studies.

**For *Sickle Cell Anemia: A Solution At Last,* send $25 to Natural Health Research Institute, 2010 S. Nogales Ave., Tulsa, Oklahoma 74107-1840.

***For the cookbook, send $18.95 plus $3 shipping to.Chef Dawud Ujamaa, 3955 Emerald North Circle, Decatur, Georgia 30035.

One seasoned biochemist told me frankly *it would be tough to get funding for research on an unpatentable nutrient substance.* That's probably a key to why thiocyanate as antisickler is one of the best-kept secrets in medical annals!

I did manage to get an appointment in May 1994 with Elliott Vichinsky, M.D., chief of haemotology/oncology and sickle cell at Children's Hospital Oakland. He explained that coping daily with transfusions and other life-and-death matters with infants and children left him few research options. Also, he said he had treated several of the patients who developed nerve damage from prolonged sodium cyanate treatment in the 1970s trials, and dismissed any possibility of better outcomes if *thiocyanate* were used. No published studies on thiocyanate (since 1932) was proof enough for him.

"Well, yes, nothing in the medical journals, but here's an unpublished one, " I countered, offering him Dr. Richardson's 1991 paper, "Hematological analysis of sickle cell patients in a clinical trial with potassium thiocyanate (KSCN)."

Five sicklers received 500 milligrams daily of a KSCN nutritional supplement in Richardson's clinic, while one patient didn't -- the control. Within as little as five days, significant blood improvements took place in all but the control. Ordinarily it takes heroic measures like transfusions to raise hemoglobin and hematocrit levels.

Vichinsky was impressed enough by Richardson's study (also by the fact that I had coauthored a book by an MD) to give me the name of a doctor in San Francisco who he thought might be interested in doing a thiocyanate trial. Soon after, I had a friendly talk with the prospective researcher, sent him literature, but never heard from him again.

More on the Orthodox Medical Attitude

Alas! the course of true love, e.g., Clara's investigative passion, seldom runs smoothly. But, to coin another phrase, where there's life, there's HOPE. Now we're back at the September '99 conference. There, patient after patient took the mike to talk about recoveries, fueled not only by one another's stories but by the satisfaction of seeing Dr. Agbai's concepts getting a forum.

But then a former medical officer of SCDAA who had presented a paper on transfusions a day or so before rose in the audience. Dr. Agbai tells me his attitude was distinctly peevish as he took the mike and cautioned the assembly about "this new enthusiasm" for products that deliver cyanate or thiocyanate. "Before you rush out to buy the African yams or cassava," he warned sternly, "remember, they're not subject to the same scrutiny by the FDA as drugs!"

At this, several in the audience responded spontaneously, walking to the microphone to say that since using these antisickling foods they were free of sickling crises, pain, and no longer needed transfusions.

By now openly hostile, the doctor challenged Dr. Agbai: "I want to ask you a question. Isn't your method *dangerous*, since it's known that increasing hemoglobin levels rapidly can bring on sickling crisis and even stroke?

At the podium Agbai had responded to the doctor's warning about cyanate- & thiocyanate-yielding foods, saying these have been staples for centuries in countries where malaria was, and often still is, a big threat. [Not coincidentally, sickle cell genes *also* exist in people of all races in these countries, including Africa, Saudi Arabia, India, Egypt, Greece, Sicily, Turkey, Philippines, the West Indies, etc. CF] Thus, people all over the world learned a long time ago to process such foods to minimize toxicity.

Now in answer to the doctor's challenge he pointed out that the 1932 thiocyanate study disproved the danger of crisis, as did his own case studies. "It's true that a sudden rise in hemoglobin may present a danger *if* the new hemoglobin is going to form sharp crystals that cause rbc's to sickle," he explained. "It's *not* a danger for persons who continue to eat the protective nutrients, for their rbc's now *resist* sickling.

A Special Protective Function

"Furthermore," Agbai continued, "an optimally high level of thiocyanate in the blood, besides making hemoglobin more soluble and less apt to form crystals, *serves also as a natural dilator of blood vessels,* thereby lessening risk of crisis, and probably of stroke." [In part because of this attribute potassium thiocyanate was used medically in the 1930s and '40s to lower high blood pressure. CF]

He again referred to "the wisdom of Divine Guidance or 'Chima' that provides us with such elegant solutions to our problems. "

The auditorium became very quiet as people turned to look at the MD, awaiting his answer, but he sank to his seat without a word!

Thoughts and Hopes

I'd be surprised if this international gathering of scientists, health workers, and patients wasn't affected by the spontaneous outpouring of success stories, in a disease noted mainly for its grim outcomes. Maybe the chances are zilch for a cheap nutrient to challenge head-on the techno-pharmaceutical-surgical forces holding center stage.

But remember, dear people, few initially believed vitamin C was the answer to deadly scurvy, or in this century that a single B vitamin, niacin, would prevent pellagra. And closer to our time--1981, in fact--there was only a handful of takers when Donald O. Rudin MD [9] offered a comprehensive theory on "modernization diseases" [ranging from dandruff to schizophrenia and heart failure] arising in good part, he said, because of profound systemic imbalances engendered by a huge 20th century drop in intake of -- yep, you guessed it -- omega-3 fats.

These were all unpatented substances found in natural foods.

I'm delighted to report, by the way, that the stodgy psychiatric establishment at long last has begun to embrace Rudin's views without, of course, crediting him. Journals are publishing new studies of omega-3 oils easing sufferings of manic-depressives and schizophrenics -- just as Rudin predicted 20 years ago from results of his pilot study.

Is sickle cell a deficiency disease?

The concept of natural foods as healers is as old as humankind. Robert Houston's[3] remarkable synthesis more than 25 years ago never ceases to amaze me when I reread it.

He drew upon Linus Pauling's and Ernst Krebs' work, among others, for the theory that if people with sickler genes were freed from anemia and morbidity on traditional daily diets generating high blood thiocyanate levels; and conversely, if they manifested all the morbid aspects of sickle cell anemia when

not eating such foods while demonstrating *low* blood thiocyanate levels, then **"sickle cell anemia might be characterized as a genetically determined nutritional deficiency anemia."**

Dr. Agbai writes, carrying Houston's torch gratefully: "Just as eating foods that are rich in iron eliminates iron deficiency anemia, it is proposed here that by eating foods rich in thiocyanate, the thiocyanate deficiency anemia (sickle cell anemia) will be effectively controlled."

If this premise holds up under widespread formal studies, not just the physical suffering but much of the disease onus surely will be lifted from children with sickle cell genes, as well as the burden of guilt from their parents.

While waiting for the medical bigwigs to catch up with tribal wisdom, sicklers can jump the gun by reading Agbai's and Ujamaa's books and tanking up on the foods described so lovingly. (You don't have to be a sickler -- they're delicious, I eat them all the time.)

So I'm impatient but also optimistic that sickle cell therapy with thiocyanate, as food and affordable supplements, will find its place in the sun. Maybe not tomorrow, but soon. ❑

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The Norm/Michael Jantze

CURE YOURSELF OF DIETING!

Every woman I've known in my life, my generation or younger, has been trying to achieve perfection by losing 15 pounds. The only exceptions have been the truly bounteous who're trying to lose 50, and the 2% who want to stop looking like toothpicks. Your savvy editor before she wisened up struggled in the same trap.

Because of this peculiarly 20th century obsession (created and fueled early by movies and the growing advertising business, later reinforced on an hourly basis on TV), women and men equate female beauty with full breasts on a skinny boy-body. Nature doesn't make most of us ladies this way. I'm writing this in case you haven't been to a public pool lately.

In ***The Diet Cure*** (1999, Viking, Penguin Group, New York, NY) Julia Ross writes: "This is not going to be like any diet book you have ever read. I won't mention calories except to forbid you to eat too *few!* ....We know now that dieting leaves us in worse shape than before we started. Our health, energy, mood, and weight have all deteriorated because of dieting. *And yet we can't quit.* We know no other escape from the weight gain that was triggered by our last diet! What we really need now is to be cured of dieting. We have to find an entirely new way to deal with our weight."

Julia Ross M.A., director of Recovery Systems* in Marin, CA, is a tall, very goodlooking, *naturally thin* (g-r-r-r!) woman who helps people who are troubled with destructive addictions. I've written about the clinic's unusual success rate in freeing clients *permanently* from their substance abuse (*FLs 68, 73, 98-99*), building on the concepts Joan Mathews Larson Ph.D. pioneered in her Minnesota clinic -- essentially by *focusing on healing the client's screwed-up biochemistry.*

A Means to Break the Cycle

A funny thing happened along the way: Julia Ross discovered the same approach, with modifications, worked for their *eating disorders*. Her clinic's success in reversing these permanently has been so stunning it propelled the writing of the book. From case after case she learned an extraordinary but well-hidden truth: plain old everyday dieting for weight loss *is the primary cause of eating disorders, some of which turn deadly.*

No, not low self-esteem, anxiety, early childhood abuse, super-control needs, and all the other psychological rationalizations around compulsive overeating, bulimia, or anorexia. No, just dieting itself. Sure, plenty of new emotional problems make their debut and old ones reinforced, once the eating disorders take hold.

She writes: "'So why can't I lose weight?' my clients ask. I point out that they usually *have* lost weight -- dozens, sometimes hundreds of times. Truly, there is nothing harder than dieting. Starvation by any name--famine, drought, or Optifast--is a frightful ordeal. Most of those critical spouses and family members could never stand the course of even *one* diet."

More grim news: "More than 95 percent of dieters gain back any weight they lose within two years after a diet. But many have gained more than they ever lost to begin with. It is typical for dieters to become progressively heavier than they ever would have been had they never dieted."

How the Body Copes With 'Famine'

There are sound reasons for this. Dieting (starvation, really) causes loss of muscle, but it's *muscle* tissue that burns most calories. The body, correctly bracing for more famine, lowers its metabolism. Many ex-dieters actually get fatter on shockingly few calories. Their battered metabolic machinery now uses calories to make and store *fat* instead of muscle, in a desperate ploy to cope with the next 'famine.' And very importantly, dieters have played havoc with their brain chemicals--key neurotransmitters that can only be biosynthesized adequately when there's ample protein, fats, calories, and all major nutrients in daily intake.

Magic Mood Molecules!

Julia tells the discouraged 'career dieter': *"It's not because you are weak willed, it's because you're low in certain brain chemicals...."*"These brain chemicals are thousands of times stronger than street drugs like heroin. And your body has to have them. If not, it sends out a command that is stronger than anyone's willpower: 'Find a druglike food or a drug, or some alcohol, to substitute for our missing brain chemicals. We cannot function without them!' Your depression, tension, irritability, anxiety, and craving are all symptoms of a brain that is deficient in its essential calming, stimulating, and mood-enhancing chemicals."

Your brain relies on protein--the only food source of amino acids -- "to make all of its mood-enhancing chemicals." Prolonged stress can short-change output of 'feelgood' neurotransmitters. So can druglike foods: bread, bagels, pasta, etc. can create 'fake' pleasure chemicals that inhibit the real ones. Low moods may be partly hereditary -- they seem to run in families, but then so do poor food habits!

Self-imposed Starvation

But for the average dieter, *it's the dieting itself* that knocks out production of the 'feelgoods.'

In fact, if you've been a regular dieter, your average day's calories "may have dropped below the amount provided at the dreaded Nazi camp at Treblinka: 900 per day. When I give this figure to female high school and college students they gasp. They think of 900 calories as generous and regard 2,500 calories per day as 'gross.'"

Yet 2,500 calories, as determined by Dept. of Agriculture standards, is the *minimum* amount an adolescent or adult woman has to have to cover basic nutritional needs. The World Health Organization established "that *starvation begins under 2,100 calories per day.* But half of U.S. women "eat fewer than 1,500 calories per day."

During the critical growth period of puberty and adolescence, young bodies require at least 2,500 high-quality calories, "yet many girls, if not most, at this age try to limit themselves to fewer than 1,000 calories a day, and often those are junk-food calories. This starvation diet can quickly develop into compulsive eating, bulimia, and anorexia. In fact, two 14-year-old anorectic girls came to the Recovery Systems clinic recently. Their eating disorders had started after their very first diets."

Fuel for Neurotransmitters

Julia describes the basic known neurotransmitters that create our moods as:
"1. dopamine/norepinephrine, our natural energizer
"2. GABA (gamma amino butyric acid), our natural sedative
"3. endorphin, our natural painkiller
"4. serotonin, our natural mood stabilizer, sleep promoter, and mind-focusing chemical."

Of all the amino acids needed to make feel-good molecules, *tryptophan* is the least available in foods and depleted the fastest by weight-loss dieting. Its loss quickly is reflected in increased depression, sleep problems, compulsive carbo cravings, etc. Why? Serotonin, the great mood enhancing molecule, is made by the body from trytophan. So is melatonin, the sleep regulator. Prozac and similar widely prescribed drugs work by keeping your serotonin working longer, but not by helping you to make new serotonin, as tryptophan does.

Eight Key Imbalances

A questionnaire in the book similar to one administered to Recovery Systems clients helps readers to identify their problem area(s). Is it depleted brain chemistry? malnutrition due to low-calorie dieting? unstable blood sugar? unrecognized low thyroid function? addiction to foods you're actually allergic to? hormones out of balance? systemic yeast overgrowth or parasites? fatty acid deficiency?

The book devotes a chapter to each of these eight critical imbalances, explaining their destructive impact on a person's wellbeing, then *another* chapter on each explaining what to do to restore homeostasis. Medical help is suggested where it's appropriate, lab tests where needed; and resources are listed throughout.

Recommended correction measures usually include specific amino acids and other repair nutrients; home testing for food allergies; lab and home tests for thyroid imbalance and exhausted adrenal glands [commonly overlooked health-robbers!]; foods to avoid as well as foods for repair.

Don't Leave Home Without A Hi-Pro Breakfast!

You won't find the following concepts in most weight-loss guides:

"Do not undereat...My clients thrive on 2,500 or more calories per day....Be sure to eat 3 meals *minimum* per day;... 20 grams protein or *more* per meal..." Above all, "***Deprogram yourself about dieting. Stop weighing yourself and counting fat grams and calories. Stop kidding yourself that low-calories dieting will help you lose weight---it will make you gain.***" [emphasis mine. CF]

Julia has observed for ten years how quickly and effectively the supplements she employs stop unstoppable food cravings. The next order of business for ex-dieters is to get those 2500-plus calories *primarily* from foods that build health. The book is a treasury of breakthrough information on good foods and good fats (yes, omega-3's too) and ways to include them daily -- the recipes and menus are do-able for working people.

They finally get off their duffs!

She's learned she can't force or cajole her ex-dieters into a regular exercise regimen, but many clients eventually find the vigor for it by dint of their rebalanced metabolism. Their 2500-plus calories go to make energy and rebuild muscle, which then helps them to burn up their old 'anti-famine' storage fat. Slowly but steadily, good things happen to her clients' weight, shape, and outlook. *Permanently*, for most.

The thoroughness of Julia's book and its groundbreaking concepts make it a one-of-a-kind reference for health professionals who treat grave food disorders. It should be read by their clients, too -- or even, for that matter, by anyone with run-of-the-mill weight battles, food cravings, mood swings, etc. -- which may mean about 90% of us! ❐

*To contact Julia Ross by mail: *The Diet Cure*, 775 E. Blithedale #402, Mill Valley CA 94941. Recovery Systems information: 415 458-8446.

CATCHING THE GOOD RAYS

In the last newsletter I wrote about Reinhold Vieth's superb review of vitamin D (*Am J Clinical Nutrition,* May 1999; 69; 842-856). His forceful conclusion is that great numbers of us in latitudes some distance north or south of the equator are not getting enough of the sunshine vitamin from sun *or* supplements to keep us healthy.

Longtime vitamin D researcher Robert Heaney in his editorial in the same issue says amen to that. One of the consequences he points to is "the worldwide epidemic of osteoporosis." Many factors may be involved in this bone density ailment, he writes, but even a mild degree of "vitamin D insufficiency" can produce it.

We Rely on Faulty Test Standards!

Now accepted as the best predictor of a person's vitamin D status is the metabolite in blood serum known variously as "25-hydroxyvitamin D," or "25 OH-cholecalciferol," or "25(OH)D." The reference range for it in standard lab tests is **45 to 90 nanomoles per liter**: below 45, you've too little vitamin D; over 90, you're in danger of toxicity.

Balderdash and poppycock! says Vieth. In early humans these concentrations "were surely far higher than what is now regarded as normal. Humans evolved as naked apes in tropical Africa. The full body surface of our ancestors was exposed to the sun almost daily." Now we expose hands and face--about 5% of body surface--not even every day. And slather on sunscreen, to boot.

Tough research challenges

I'd no idea until I'd waded thru a stack of studies how complex the job is for scientists to get a true handle on, for example, the quantity of vitamin D you can make per square inch of exposed skin; how long you have to be exposed; the factor of lightness or darkness of skin (dark requires lots more time); your age (older people may need four times more exposure time than young, etc., etc., let alone figuring out what's needed supplementwise to compensate for its scarcity in foods! (Nature clearly meant us to get D from sunlight on our bods; even breastmilk hasn't much because an early humankind baby soaked up rays along with its mom.)

It isn't a vitamin, really. Cholesterol in your skin grabs the sun's ultraviolet light rays to *make* the initial vitamin D_3 molecule, cholecalciferol. (We can't *make* any other vitamin.) After enzymatic transformations in liver and kidneys, what emerges in essence is a *hormone.* (A simplified name for this new molecule is calcitriol.) And scientists have yet to puzzle out all its roles, besides those of making calcium assimilable and building bones and teeth.

One very pivotal job for vitamin D, which doesn't get much press, is its responsibility for the *ionization* of calcium. About half of the calcium circulating in plasma throughout the body has to be ionized by vitamin D. The ionized form alone is required for "most functions of calcium in the body, including the effect of calcium on the heart, on the nervous system, and on bone formation." (A.C. Guyton MD, *Textbook of Medical Physiology, 6th edition,* 1981, WB Saunders.)

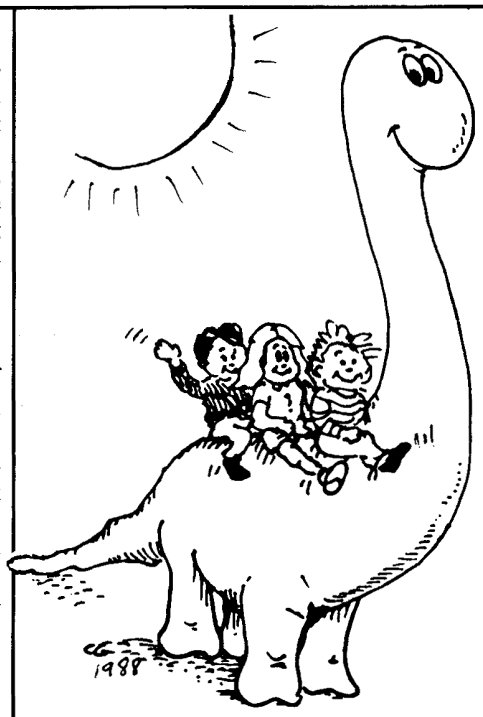
To produce the ATP "energy currency" molecules that fuel all cellular activities, the cell's "energy factories" (the mighty mitochondria) must use *ionized* calcium.

No Ca ions=no cell energy!

Carl Reich MD of Calgary premised over 30 years of clinical practice on this unique function of vitamin D. Without enough ionized calcium, he said, "cell energy starvation" takes place. To combat this the body *acidifies* its cellular fluids, in a desperate effort to ionize whatever calcium it can. [See *FL 85.]*

Reich used saliva pH tests to confirm widespread ***chronic systemic acidity*** in his patients, including children. He insisted that this and low cell energy are prime instigators of all kinds of health nuisances: fatigue, headaches, muscle cramps, allergies, gut problems, etc. If vitamin D deficiency persists and the body's adaptive processes become exhausted, he said the small complaints evolve into serious ones: asthma, hypertension, osteoporosis, arthritis, etc., depending on which organ or system is most vulnerable.*

*He proved his theory by treating patients with calcium, magnesium and zinc; vitamin A, and vitamin D (4800 to 8800 IU daily for adults; 2400 to 5200 IU daily for adolescents; 1,400 IU for children). He also recommended an alkaline-producing diet high in vegetables and fruit. Periodic blood and urine tests showed no abnormally high calcium levels. He lowered the doses as patients improved. And, yes, he saw very consistent improvements and recoveries in his patients!



Vitamin D insufficiency: A link to common ailments?

I think the medical world may be catching up to his ideas. Vieth points out successful experimental use of UVB light *to lower blood pressure* in moderately hypertensive women. That's in line with the fact that "prevalence of hypertension in a population increases with distance north or south of the equator." Not coincidentally, the women's 25(OH)D levels rose from **58** to **150** nmol/liter.

He tells of arthritis researchers recommending that persons with *osteoarthritis of the knee* take whatever measures needed to get their serum 25(OH)D up to at least 75 nmol/L, because of good results seen at the higher 25(OH)D levels.

So we've got strong correlations between too-low serum 25(OH)D, hypertension, and arthritis. To these we can add what Heney refers to as "the worldwide epidemic" of *osteoporosis* (poor bone mineral density). (The other related vitamin D deficiency disease, *osteomalacia* (softening and bending of bones, known as rickets in children), was prevented or treated for centuries with fish liver oil as folk medicine.)

Hypertension, arthritis, and osteoporosis are among the commonest afflictions of older people in latitudes away from the equator. Vieth says an important way to fight these is to get people's serum 25(OH)D levels ***higher than 100 nmol/L*** -- just like levels of folks exposed to sun much of the year in latitudes nearer the equator.

Stop the Presses!!

It must be fate. The new *Lancet* came in the mail just as I was finishing the newsletter pasteup to take to the print shop. The lead article was "High blood pressure and bone-mineral loss in elderly white women: a prospective study" (F.P. Cappuccio et al.,Vol 354, Sept 18, 1999, pp 971-5). "High blood pressure," they write, "is associated with abnormalities in calcium metabolism." They mapped bone density changes in 3676 women (66 to 91 years of age) living mostly in higher U.S. latitudes.

Only the femoral neck was tested, a vulnerable section of thigh bone near the hip socket. Sure enough, retesting after 3 to 5 years showed high blood pressure to be "associated with increased bone loss at the femoral neck...[which] may reflect greater calcium losses with high blood pressure, which may contribute to the risk of hip fractures."

D'you suppose these "calcium losses" and resulting osteoporosis have anything to do with ***loss of ionized calcium because of vitamin D insuffficiency??***

New Vitamin D Dietary Guidelines

Official medical policy-setters recently have taken the unprecedented step of *tripling the recommendation to 600 IU* for those over 70 years, while retaining the same, skimpy 200 IU per day for all other adults.

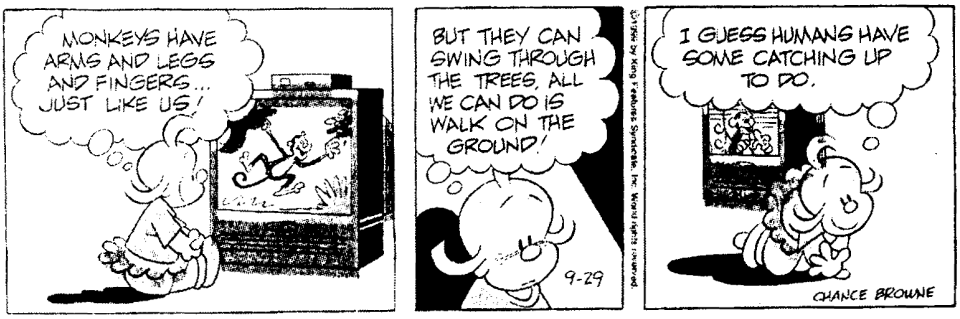
Vieth suggests a beneficial amount is more likely to be **800 to 1000 IU a day**, "on the basis of bone density measurements and fracture prevention in the elderly." Even with 1000 IU, people would still have to get about another 3000 IU *from UV sunlight,* to fulfill their needs which he says is about 4000 IU per day. It takes at least that to achieve protective serum 25(OH)D levels, by his evidence.

Fear of toxicity has always governed these decisions. Reich pooh-poohed the toxicity alarrums years ago, citing studies showing it took daily doses of 50,000 IU to make trouble. Vieth (of Toronto, another Canadian--it's getting to be my favorite country) finds key toxicity reports to be poorly documented, not well substantiated, incorrectly interpreted, etc.

Lessons from our beginnings

Heney believes it's no longer cool to equate vitamin D sufficiency "with the absence of rickets or osteomalacia." The vitamin is needed for a lot of other yet-to-be explored good deeds. For instance, Vieth says a number of population studies show that higher serum 25(OH)D levels, or greater sun exposure, *are associated with lower rates of breast, ovarian, prostate, and colorectal cancers, and less multiple sclerosis.*

Hi and Lois/Brian and Greg Walker



The very complexity of vitamin D's actions and absorption patterns compels wise scientists to look for simpler models as they try to come up with realistic guides. Vieth points once again to evidence from human evolution:

"....modern society in general is vitamin D-deprived compared with prehistoric humans. The concentrations of 25(OH)D observed today are arbitrary and based on contemporary cultural norms (clothing, sun avoidance, food choices, and legislation) and the range of vitamin D intakes being compared may not encompass what is natural or optimal for humans as a species."

For his part, Heney says the *evolution of our own physiology surely is related to the high ultraviolet light exposures that early humans experienced.* He challenges the typically bass-ackward research mindset on this by suggesting that rather than have all the burden fall on establishing the efficacy of these exposures, "one would think that the burden would be on establishing the safety and adequacy of the often much lower contemporary exposures."

Incidentally, too much sun can burn you, but it won't cause any more vitamin D to be made than your body needs. The excess made in the skin is degraded, creating equilibrium, with no toxicity. Nature's really something!

Trumpets, please!

Dear readers, a light bulb just lit over my head: I thought of Carl Reich's theory of cell energy starvation from vitamin D deficiency *in terms of sicklers.* Their hemoglobin is known to be especially vulnerable to sickling under both low oxygen pressure *and acidic cellular conditions.* Could a factor in the severe anemia and frequent crises in African Americans, or in emigrants from the West Indies, be *chronic underexposure to UV light???* Dark-skinned people need **3 to 6 times more exposure time** than light-skinned ones to make ample D. If, as Vieth and Heney say, whole populations in U.S. and Canada suffer from unrecognized vitamin D insufficiency, this will be much greater in black and brown Americans of any race, especially when they live in the northerly latitudes. If Reich is right, they may be prone to chronic systemic acidity -- meaning, for those with sickle cell genes, more sickled rbc's, anemia, and crises.

Check your nanamoles, everybody!

All of you, including sickle cell folks, please ask your health advisors to include 25(OH)D serum levels* with your routine lab tests. **If you test below 100 nanamoles per liter [nmol/L], get more sun on your bod and raise your supplement dose, *dye'hear*??** I'd hate to think I'm doing all this journal-chasing for nothing! ❑

*A subscriber wrote to tell me she got her test results for 25(OH)D in different units. Here's the translation: **100 nanamoles per liter** is equivalent to **40 nanograms per milliliter.**
And here's the arithmetic: nmol/L = ng/mL **multiplied by 2.5.** Or the other way 'round: ng/mL = nmol/L **divided by 2.5.**

Illustrations are by the late Clay Geerdes and other artists as noted.

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