

# The FELIX Letter

A COMMENTARY ON NUTRITION

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We're doing *something* right: heart attack deaths are down, as are deaths from strokes. The unprecedented epidemic of deaths from coronary heart disease (CHD) began to appear in the 1920's, increased ominously between 1940 and 1960, reached a plateau between 1960 and 1967... then, for the first time, began decreasing. The decline is about 2 percent per year, an encouraging trend which appears to be continuing. On public television's MacNeil-Lehrer Report, December 9th, I listened to a group of physicians attributing the changes to these factors:

- Blood pressure awareness in the public
- Dietary salt restriction
- Medical control of high blood pressure
- Weight control
- Dietary restriction of fats and cholesterol
- Reduction of smoking
- Improved ways of lessening chronic emotional tension
- Increased exercise and physical fitness programs
- Better medical care for patients with CHD, e.g., improved emergency care, anti-coagulant and lipid-lowering medication, pacemakers, and so on.

With few variations, the medical consensus in professional journals and releases to the public is essentially summed up by the above. I am not arguing with it — but I feel it is lacking a key factor. Maybe I missed the journal article or TV discussion I was hoping to see, but I'm inclined to believe it didn't take place: an acknowledgement by medicine of the role the "back-to-whole-foods" movement and the *greatly expanded use of vitamin and mineral supplements* have played in this phenomenon.

## Nutrients and the Heart

Common sense tells me it cannot be otherwise. For a long time I've seen studies which, fragment by fragment, add up to a still-fuzzy picture whose meaning gets clearer each year: *the health of the cardiovascular system is dependent on a*

*steady flow of nutrients and trace elements*, some not even recognized as essential until a few years ago. In the early 1900's, chemists and biologists were starting to investigate elements in food which had uniquely remedial effects on illnesses like beriberi and rickets. Only in the 1930's was vitamin B1 (thiamin) isolated and its structure determined by Dr. R. R. Williams who, in 1910, first became interested in the mysterious substance found in the outer coat of rice which alleviated polyneuritis in chickens, similar to human beriberi. In addition to destruction and paralysis of leg muscles, beriberi affects the *heart and cardiovascular system* severely.



If man weren't such an ingenious inventive creature, we wouldn't have beriberi in the first place, because we'd still be eating whole brown rice or other grains, like our animal and bird friends who aren't smart enough to invent milling machines. With all due respect to the wise men of medicine, there is at least as much evidence to link the epidemic of CHD with the magnitude of changes in our food brought about by the new technology as there is with the diet nemesis of the 1970's: hard fats and cholesterol. Degenerative atherosclerotic changes in arteries are the underlying factor in most heart attacks and strokes today, but until

the 1920's, heart attacks and strokes from this (or any) cause were much rarer. Then, hearts damaged by rheumatic fever or congenital anomalies were the ones that failed. Until the turn of the century, we were still a mostly rural nation, eating from our own farms or kitchen gardens. More cities grew and so did the technology to feed them. The introduction of steel rollers into milling in the 1880's produced a whiter flour than ever before; the new sugar refining machinery in the 1860's began to make sugar so abundant and cheap that per capita consumption shot up six-fold from 16 pounds a year (in 1815) to over 90 pounds — a quarter of a pound a day — at the turn of the century. (*This* is the U.S. consumption figure constantly referred to as "the norm," as if the thousands of prior centuries without refined sugar had been a curious anomaly that, happily, was now being rectified.)

## CHD and the New Technology

We're learning a lot about the "lag" theory in cancer — the period of time that may lapse between the introduction of a noxious substance and the appearance of the ultimate insult to the cells. So it may be with degenerative diseases like atherosclerosis. The illness may take twenty or thirty years to manifest itself though the process can be happening all along. The serious beginning of our heart attack epidemic in the 1930's and 40's could thus date from the turn of the century, the victims often being middle-aged men. Fat consumption had increased somewhat, but sugar was being consumed in amounts unknown in human chronicles. At the same time, the steel-roller milling of grains had diminished a major source of trace minerals like chromium and selenium, the B-vitamins (including the "heart" vitamins, thiamin and B6), vitamin E, and fiber. For insulin to carry glucose (from sugar or starch) effectively into our cells after digestion, the trace element chromium is needed — a fact only learned in the 1960's; and to metabolize sugar into energy or fat, our system requires an impressive array of vitamins and minerals, among them magnesium, iron, sulfur, thiamin, niacin, pantothenic acid, riboflavin, and biotin.

Although sugar beets and cane have all of these nutrients, refined sugar has none, and the body must rifle its own stores to metabolize it. A compounded indignity to tissues occurs when the diet itself no longer contains adequate amounts.

Excellent laboratory and population studies continue to show clear connections between lack of a particular vitamin or trace element and the appearance of cardiovascular symptoms in man. Examples:

*chromium deficiency* - impaired glucose tolerance; elevated serum lipids; atherosclerosis

*selenium deficiency* - cardiomyopathy

*magnesium deficiency* - increased deaths from heart attacks

*low vitamin C levels in white blood cells* - coronary artery disease

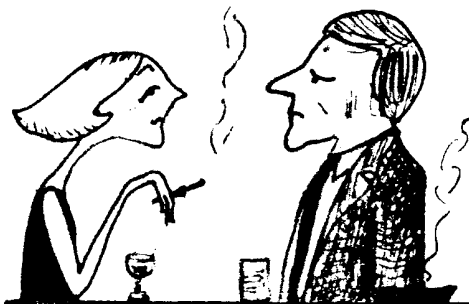
*vitamin E deficiency* - marked alterations in heart function; leg pains (intermittent claudication)

Unfortunately, there is another "lag" aspect we are faced with: the time lapse between the appearance of studies like these . . . and the awareness, acceptance, and implementation of this knowledge into clinical medical practice. Having observed this discouraging phenomenon more times than I care to remember, I've developed a philosophy about information which may have direct bearing on health and well-being: if enough corroborative studies exist, and if my intuitive feelings are strong, I'll act on the material. By no means have I batted 1000 but, all in all, the benefits have far outweighed the failures — besides allowing me to feel righteously smug when, some twenty years after I may have first incorporated it into my regimen, the academicians come out with a hot, new policy on nutrition.

### The Mythical Vitamin E

One of my hunches or educated guesses was about vitamin E. Side by side with the popular literature touting it 25 years ago were thoughtful studies by sober research groups. If vitamin E existed in every seed to protect its oils from rancidity and peroxidation, and was needed by laboratory animals to maintain normal reproductive function. . . then, by George! it was good enough for me and my family. There was convincing evidence of its clinical benefit in coronary disease as described by the Canadian physician-brothers Wilfrid and Evan Shute, who treated thousands of heart patients beginning in 1945. The fact that the American Medical Association scorned the Shutes' work and the value of vitamin E was, by then, almost a point in its favor for me. The vitamin, the AMA said, couldn't possibly be responsible for

the multitude of symptoms attributed to its lack, especially since it was plentiful in our diets. Their view was reflected in the staid world of academic nutrition at least as late as 1973 in the 9th edition of a standard nutrition text, *Nutrition and Physical Fitness*, by Bogert, Briggs and Calloway: "Naturally occurring vitamin E deficiencies in adult man in the United States are extremely unusual," although the same chapter noted: "The vitamin E activity of foods may be considerably reduced in processing, storage, and packaging. For instance, as much as 80 percent or more may be lost in converting whole wheat [the grain] to white bread" [emphasis mine].



"IF GOD WANTED US TO HAVE VITAMIN E,  
HE'D'VE PUT IT IN VALIUM!"

The National Research Council had belatedly acknowledged the essentiality of vitamin E in 1968 by establishing a recommended dietary allowance (RDA) of 30 International Units for adult males, based solely on estimates of vitamin E available in average diets. They penitently cut it down to 15 IU in their 1974 and 1980 recommendations. Their reasoning? The amount of E available in diets in the United States "is considerably less" than estimated in 1968, they said.

*Inasmuch as there is no clinical or biochemical evidence that vitamin E status is inadequate in normal individuals ingesting balanced diets, the vitamin E activity in average diets is considered satisfactory.*

We were still in the midst of a heart disease explosion in 1974, when the RDA for the vitamin was halved. The connection however between possibly inadequate vitamin E levels in our diet and the epidemic of atherosclerotic arteries was too abstract for the Council to make, and it has remained so to this day in nutrition/medical policy-making circles.

### Cell Membranes and E

Meanwhile, back at the lab, evidence continued to pile up. It served to demonstrate a *unifying principle* explaining the puzzling wide-ranging effects of the vitamin which, in the late 1970's was finally accepted by the conservative nutrition establishment: by "soaking up" and "defusing" harmful oxidizing agents produced both by the environment and in normal cell metabolism, *vitamin E serves as a major protector of cell membranes.*

When I was a life science major my first time around academically in the 1940's, human cells viewed by our microscopes were seemingly simple units: a fluid mass of granules called "protoplasm," nucleus, a few cell vacuoles — all enclosed within the cell's membrane. The great electron microscope changed everything: those tiny dots in protoplasm proved to be intricate organelles with production and assembly line functions of their own, and *they* had membranes, too. Later, we learned that in each membrane, both within and enclosing the cell, molecules of vitamin E existed in a certain ratio to the unsaturated fatty acid molecules that formed part of each cellular membrane. Since our tissues and organs — including our hearts and arteries — and our whole selves are formed from billions of these very cells, it strikes me as reasonable to assume, at the very least, that a deficiency of vitamin E would have far-reaching consequences.

### A New Role for E

Now at last, in addition to vitamin E's role as a stabilizer of membranes, the mystery of its specific effect on cardiovascular health is finally being unraveled. The biological catchword of the '70's and '80's has been "prostaglandins," and newly-discovered prostacyclin (PGI<sub>2</sub>) is a prostaglandin with remarkable properties: it is naturally produced in the heart and arteries, where it dilates the arteries and prevents aggregation of blood platelets. "Sticky" blood platelets are a big factor in abnormal clot formation, constricted arteries cause high blood pressure, and together they can lead to myocardial infarction (heart attack) and stroke. The new awareness (since 1976) that in prostacyclin our bodies produce a natural inhibitor of platelet clumping and a dilator of arteries has created a lot of optimism in medical circles. In the lead article in the November *American Journal of Clinical Nutrition* (1981, Chan & Leith), scien-

tists are now saying that *the synthesis of prostacyclin in arteries may be dependent on vitamin E*. The authors suggest that "a defect in focal production of PGI<sub>2</sub> may be the underlying pathogenesis of atherosclerosis and moreover, diseased vessels may benefit from increased PGI<sub>2</sub> production." A deficiency of the vitamin, they found, depressed prostacyclin synthesis; while "refeeding vitamin E to the deficient animals promptly restored PGI<sub>2</sub> back to control levels."

I'd say that's jolly good news for all of us when, again, a simple food factor comes up roses. This may explain why a few nonorthodox surgeons have used vitamin E successfully to prevent abnormal clotting in patients after surgery without the danger of hemorrhage that anticoagulant drugs can create.

Will this new information affect the nutrition/medical policy makers? I doubt it. Their argument primarily is no longer with the need for vitamin E in human nutrition but with the presumption that a widespread deficiency of the vitamin exists. It *doesn't*, they say, and if there *does* prove to be a clear relationship between vitamin E and cardiovascular health, the current decline in heart attack deaths is only further evidence that a deficiency never existed.

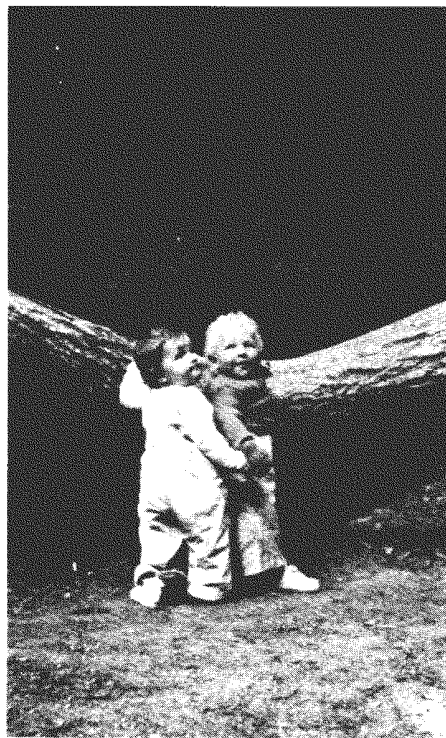
### The Great Coincidence

My original hunch about vitamin E grew from the observation that our living ancestral cousins in the wilds — the apes — consume a great deal more of it than we do, even before we became a nation of fast-fooders. The turn-of-the-century dietary changes were perhaps only the straw that broke the camel's back. The effects of the accelerated loss of fiber, trace elements, and vitamins, including E, *on a scale incompatible with cardiovascular health* began to show up strongly in the 1930's and grew until CHD was our leading cause of death. With whatever other effective therapeutic interventions have developed, coinciding with the turning of the heart-attack tide at the end of the 'sixties has been the phenomenal rise in consumption of nutritional supplements, including vitamin E. More than 700 million dollars worth of vitamins a year are now sold here.

### Leaving the Garden of Eden

It takes a special kind of determination on the part of the medical community to ignore this rather astonishing correlation, but I suspect this may arise from a conviction that to attribute to nutritional supplements an effect of such magnitude would be altogether too simplistic. I, on the other hand, feel there is nothing

simplistic in the possibility that restoration of missing nutrients may have profound effects, just as I feel there is nothing simplistic about the evolvement of human beings in an environment which originally met their nutrient needs with exquisite rationality . . . as it did those of all surviving species. HOWEVER. . . we are the *only* living creature that doesn't simply eat its food: we mill, strip, polish, cook, pulverize, extrude, fractionate, reshape, and decorate it . . . and a lot is lost in the translation! The last hundred years of frenetic technology have only carried these impertinences to their inevitable outcome: our food has lost its evolutionary logic and we are feeling the consequences. Replacing lost values with little supplements in bottles is an absurd way to go, but it's a stopgap and may be saving lives. If the declining rate of heart attacks and the rising sale of vitamin and mineral supplements are more than coincidence, there's cause for optimism, as well as incentive for a thoughtful review of where straying from our evolutionary food path has brought us. ■



### THE BEAN BREAKTHROUGH

Being a nutrition scientist may not be a carload of laughs, but I had a hard time picturing the usual solemn research conclaves when I ran across a piece recently in the *Journal of Food Science* (1980, Vol. 45, 1161-1164). Utah State University researchers compared the effect

different methods of preparing beans had on the amount of flatulence produced in laboratory rats eating the beans. Don't laugh! The hunt for a non-detonating bean is serious business. Dr. Horace Burr, my Food Science professor at U.C. Berkeley, told our class in 1977 that the U.S. Department of Agriculture — although understandably reticent about proclaiming it — had been seeking solutions for a long time to the "flatulence factor" in beans, but were seriously handicapped by the expense and difficulties of investigating this in *human* subjects. Fortunately, he told us, a breakthrough had occurred: they had learned that rats responded just as humans did to beans: with gas! Hosannah! The winds of progress began to blow lustily as researchers scaled down their flatus-collecting apparatus to fit the new pocket-sized subjects.

A small biochemical excursion: beans cause gas in humans and rats mainly because both species lack the necessary enzyme in the intestinal mucosa and cannot split the longer-chain sugars (principally verbascose, stachyose, and raffinose) which are major natural sugars in legumes. Having escaped the usual digestive process and subsequent absorption into the blood stream of "shorter" sugars like fructose and sucrose (disaccharides), these "long" sugars (oligosaccharides) remain in the intestinal tract, where bacteria can metabolize them (really, pig-out on them) to form large amounts of carbon dioxide and hydrogen gas, and small amounts of methane gas. Although intestinal transit time in man varies widely, the production of gases would appear to begin about 6 to 8 hours after ingestion and may continue for what seems like a VERY LONG TIME to some of us. The degrees of discomfort experienced, ranging from none to painful abdominal distention, may partly depend on how efficiently the bloodstream absorbs these gases through the intestinal membrane and neutralizes them, or eliminates them eventually through the *lungs*. Yes, the lungs. (Nature moves in mysterious ways.) The balance of the unabsorbed gases will then be expelled as flatus — and the sooner the better.

Before your credulity is strained visualizing a group of sober lab workers struggling to insert tiny enema tubes into rats in order to measure very small gas bubbles, LET ME HASTEN TO EXPLAIN that one of the more convenient laboratory finds of the last decade is that *hydrogen gas measured in the breath of humans* correlates significantly with their intestinal flatus volumes. The breakthrough that Dr. Burr was describing in the Department of Agriculture's work was the discovery that

hydrogen gas production in rats who were fed beans correlated significantly with breath hydrogen measurements and flatus gas volumes in *humans*, after the same kind of beans were eaten. In other words, men and rats were similarly affected by the indigestible sugars in legumes; thus, measurement of hydrogen production in rats could provide a simply bioassay predicting for man the "flatulence factor" in beans.

Kidding aside, beans are a wonderful food, exceptionally rich in potassium, phosphorus, calcium, trace minerals, folic acid, and fiber. Recent studies have shown them to be excellent food for stabilizing blood sugar in diabetics. They become a superior source of complete protein when eaten with rice, corn, or wheat, and make

resulted in the most gas production in the animals. Analysis of the beans prepared by this traditional method also showed essentially no reduction in the oligosaccharides responsible for intestinal gas.

But when the beans were *germinated* for 24 hours at room temperature, they produced about 80 percent less hydrogen gas in the rats than the cooked beans. Correspondingly, there was a big drop in the 'offending' oligosaccharides when the 24-hour germinated beans were analyzed (for verbascose, stachyose, and raffinose). These had declined from 60 percent of total sugars in raw beans to only 8 percent in the germinated beans.

Another biochemical note: what, you may ask, has happened to the 'long' sugars during this beginning germination?

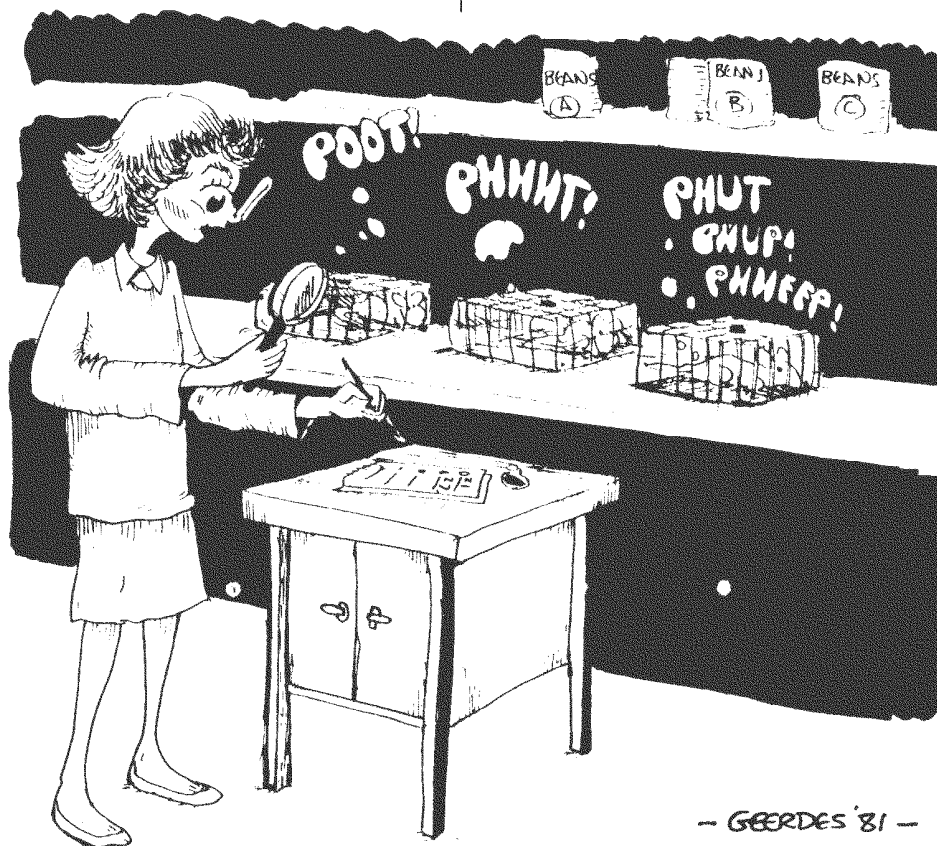
lengths of light. Dried beans have little vitamin C; bean sprouts are a fine source of it. Since ancient times, fresh bean and seed sprouts have been eaten by many cultures who undoubtedly found they sustained health when fresh greens and fruits were not available.

### Germinating Beans

I was so impressed by this unheralded study that I did a simple kitchen experiment of my own. Utah State's researchers had written: "black gram seeds were germinated for 24 hr. in petri dishes at 22° C by using sufficient distilled water." Did this imply that simply soaking the beans for 24 hours constituted germination, or did the term "germination" refer to visible burgeoning signs of sprouting? I chose the latter interpretation and used the techniques I have long found effective in growing alfalfa sprouts or mung bean sprouts: I soaked blackeyed peas in a jar of water overnight, drained and rinsed the peas, wrapped the jar and laid it on its side. (A warm dark place will speed up the process.) Two or three times during the next day I rinsed and drained them to keep them fresh-smelling, and continued the same procedure for 24 hours *after* I began to see the beginnings of sprouting (in a day or so). I then simmered the beans in fresh water for several hours with added herbs and seasoning until tender. (The beans took less cooking and water than usual.) The taste and texture were still beanlike — the sprouts were only tiny protruding tails. I did the same thing with garbanzo beans, except germination took longer to appear in these larger, denser beans. Again, taste and texture after cooking were not much different from non-germinated beans.

At this point, I must say, with all modesty, that I am an EXCEPTIONALLY sensitive subject for the detection of the F-factor in legumes, and it is with great joy that I report the experiment a success. The only qualifications: germination has to proceed in the *whole* batch of beans, so I have been continuing the sprouting/rinsing period to include all the little laggard beans who have taken their own sweet time to begin germinating, which can add another day to the process. Still, the procedure is easy, consumes little time, and allows me to eat beans with *dignity*. I recommend it to my readers. ■

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— GEERDES '81 —

a robust meat substitute for those inclined towards a greater degree of vegetarianism. Truly, to be able to eat this good natural food without painful or unsocial repercussions would be a benefit.

With all of this excellent theory as backdrop, the researchers in Utah State University's nutrition department proceeded to test hydrogen production in rats that had ingested beans prepared by several methods. Beans cooked for three hours (with sufficient water at 90° C)

They were split by natural enzymes into simple sugars to supply the needs of the sprouting rootlet and stem. As we know, beans, like most seeds, may be dormant for years but will convert their stored starch and sugar to simple sugars like glucose at an astonishing rate, to provide energy and building materials for the seedling. This is also a time when glucose is rapidly converted into ascorbic acid (vitamin C). The seedling will be breaking through to sunlight for the first time, and vitamin C is now needed to protect it from oxidative damage caused by certain wave-