SOUND THE BUGLES!

Dr. Colin Leakey, son of the late famed anthropologist Louis Leakey, is growing beans in a 1-1/2 acre plot behind his house in Girton, a village in England just outside Cambridge. These aren't just any old flatus-forming beans, they're his "Prim" bean—as in prim and proper. Dr. Leakey's focus began while working in Uganda in 1961, becoming as he describes it an "expert on flatulence" and in the process refuting conventional wisdom which holds that beans are prime toakers because of their hard-to-digest oligosaccharides, i.e., complex sugars, that end up being fermented by colon bacteria and forming billows of gas.

Instead, Dr. Leakey insists bean skins contain chemicals that inhibit enzymes we normally make to allow us to disperse gas into the bloodstream where it can exit gracefully through the lungs (instead of rumbling around in the gut and exiting in a somewhat ungraceful way.)

He insists the beans he has been breeding for more than a decade have far less of these enzyme inhibitors. His first low-flatulence bean was harvested in 1990, but sales of Prim beans through his company Peas & Beans Ltd. are modest, word having not yet gotten around that the beans are both easier to digest and very tasty. The April 1st article in Wall Street Journal said Peas & Beans may soon be marketing Prim as ready-to-eat beans in jars. Sounds interesting.

DUMPING ON OMEGA-3s

It was a long, tough fight for those of us who helped shove the neglected Omega-3 (w3) fats out of nutrition's grey zone into their rightful place as pivotal players. But no sooner was I settling into complacency, when odd and scary reports began drifting in. A series of articles in '93 and '94 medical journals warned that "high levels" (in the diet and in the blood) of the primary essential w3 fat—alpha linolenic acid (ALA)—were associated with men's risk of developing advanced prostate cancer. Soon, newspapers and popular magazines were trumpeting the dangers of consuming vegetable oils high in ALA.

The basic study, however, had specifically found animal fats from red meats to be the chief culprits. [E. Giovannucci, et al., J of the Nail Cancer Institute, Vol. 85, No. 19, Oct 6, 1993.] It depended solely on statistical analyses of food-frequency questionnaires, filled out in 1986 by over 47,000 men initially free of diagnosed cancer. By the beginning of 1990, follow-up questionnaires to the men revealed 300 cases of prostate cancer, 126 of these advanced.

From the men's reported food intakes, researchers concluded "animal fat, especially fat from red meat, is associated with an elevated risk of advanced prostate cancer." Then comes the truly strange part: the actual fatty acid they found to be "most strongly related to risk" was alpha-linolenic. That's because, believe it or not, they said red meat is "relatively high" in w3 ALA and low in the primary w6--linoleic acid. Or stated another way, "animal fat is a relatively good source of alpha-linolenic acid but a poor source of linoleic acid..."

I beg your pardon, Dr. Giovannucci et al. Red meat (beef, pork, and lamb) and its fat are poor sources of w6 linoleic, but are far poorer sources of ALA! Consult any reliable fatty acid table: typical cuts of beef, lamb, and pork contain from 2 to 13 times more w6 linoleic than w3 ALA.

(Note: Pork products contain about five times more w6 linoleic than lamb or beef, but still only a smidgin of ALA.)

In other words, the researchers goofed on their basic premise. Their tangled statistical analyses of the diets reported some years earlier "proved" that the fat in red meat responsible for advanced prostate cancer was w3 ALA, when there's little of it in red meats, and so much less of it than w6 linoleic. Moreover, they did not include w6 arachidonic acid content of red meats—a puzzling oversight.

The actual sources of w3 ALA in the diets of the men with advanced prostate cancer probably would have been, typically, soy oil in salad dressings and cooking oil—but the researchers explicitly stated it was not ALA from vegetable sources but only ALA from animal products that was associated statistically with prostate cancer.

Great statistics!

Incidently, the calculated levels of w3 ALA in the diets, according to the researchers, ranged from low to moderate. By my standards, based on work by careful scientists, even the highest levels were very low! In contrast, w6 linoleic acid intake (from salad dressings, mayonnaise, grains, etc., as well as from red meat) tended to be high, as is typical in U.S. diets. A high w6: low w3 intake ratio admittedly is a recipe for trouble, including cancer.

Dr. Stephen C. Cunnane of the University of Toronto in his chapter in Flaxseed in Human Nutrition (AOCS Press, 1995) questions the interpretation given to the data, in light of "strong evidence that ALA is not cancer promoting in animals..." and that "lean cuts of red meat contain almost undetectable ALA."

Processed red meats, i.e., sausage, lunch meat, etc., contain more fat, more ALA, but also more w6 linoleic--again, about 6 to 8 times more linoleic than ALA.

This muddled study, full of contradictory, oddly conceived conclusions, did not deserve the hoopla it got. Nobody in the press apparently checked it against food tables but swallowed it whole, issuing wholesale warnings about red meat and/or ALA. I remember in 1981 a similar to-do in medical journals about ghastly consequences of taking more than 100 to 300 I.U. of vitamin E. A nurse friend cautioned me sternly about it. For years afterwards I'd see anti-vitamin E admonitions in the medical media. I'll bet those doctors (and their patients) are sorry now, what with all the current kudos (finally!) in medical literature for vitamin E as a heart-saving, primo antioxidant.
Another area of concern expressed to me by many readers arose from warnings in Barry Sears' 1995 best-seller "The Zone" that targeted alpha linolenic acid along with such nasties as trans fats and viral infections as suppressors of an enzyme that forms the desirable w6 fatty acid GLA. An intake of "large amounts of alpha linolenic acid (ALA)" as found in "flax seeds, flax oil, and walnuts" is one of several "insidious ways to further reduce the production of this crucial enzyme, and thus limit the formation of GLA," he wrote.

In FLs 88/89 I voiced my objections based on historical evidence and sound biochemistry. Sears may have heard from other folks as well in the interim, because in his 1997 book "Mastering the Zone" he modifies his anti-ALA caveat to a suggestion to avoid consuming more than a tablespoon a day of flax oil. That's okay with me; few of us need more than that except for brief therapeutic intervals. [See Omega 3 Oils by Donald O. Rudin, M.D., & Clara Felix. Avery Publishing Group, 1996.]

We have to remember, though, that strict (nonfish-eating) vegetarians depend solely on ALA to form the vital w3 metabolites EPA and DHA. It's a complicated business, this churning down of factors involved in production or inhibition of w3 and w6 metabolites. These are highly polyunsaturated fatty acids, formed by action of specific enzymes in our cells after we eat the primary essential ones (w3 ALA and w6 linoleic acid). The metabolites eventually form long-chain fatty acids—the structural basis for membranes enveloping every cell in the body as well as each organelle within our cells. Cellular membranes control inflow and outflow of nutrients, enzymes, hormones, waste, etc. The wellbeing of our body's trillions of cells is linked closely to w3 and w6 fats from food.

A Fat-Based Control System!

Moreover, w3 and w6 metabolites also can become 20-carbon "activated" molecules known as "eicosanoids." Eicosanoids include prostaglandins, thromboxanes, leukotrienes, and lots more. They are regulators of critical human functions, serving as 'local' cellular hormones and working in sync with 'long-distance' bloodborne hormones such as insulin.

To achieve the "Zone" state of optimal health, Sears says good and bad eicosanoids must be in favorable balance. [I've no conflict with this.] The big push according to him is to raise levels of w6 DGLA and reduce those of w6 arachidonic acid: DGLA can produce "good" prostaglandins, while arachidonic acid can be the precursor to a bunch of "bad" ones.

Here his path and mine diverge. It's not arachidonic acid itself that needs to be suppressed, but unbalanced output of eicosanoids from it. Consumption of w3 EPA from fish which Sears highly recommends is a great "balancer," but w3 ALA intake from flax seeds, perilla seeds, walnuts, beans, etc. also works along the same lines.

Are organ meats no-no's?

Sears says arachidonic acid is so villainous we need to suppress its formation from w6 essential fats and also avoid ready-made sources, such as organ meats and egg yolks. He and I really part company here. The admonition against these foods, which also contain cholesterol, arose in the murky days beginning in the 1960s when the big medical push evolved to eat "heart-healthy" margarine and avoid "killer cholesterol" in foods. I don't buy any of that line. Margarine, of course, is full of harmful trans-fats, while cholesterol is an immensely valuable molecule: all mammals and birds go to a lot of trouble to make enough for cell membranes, brain function, and as the sole precursor for all steroid hormones. In the skin it's needed to make vitamin D. That's why an egg that's going to become a baby chick contains so much of it. Why should we fight nature and human history?

Starting with hunter-gatherers, organ meats were always the most valued parts of any animal or bird consumed, as they are for lions and other predators, and still are for people in less westernized cultures. As for arachidonic acid, all of us, even newborns, need lots of it for the brain as well as for cell structure in general. Infants grow less when there's none in their formula; breast milk, of course, will contain ample arachidonic acid plus the other desirable w3 and w6 metabolites, if the mother gets enough w3 and w6 in her daily diet. [Series of studies by Susan Carlson, Ricardo Uauy, Bernard Koletzko, Robert Gibson, etc. in Essential Fatty Acids & Eicosanoids, AOCS 1992.]

Inflammation, spasms in blood vessels, abnormal clotting, menstrual cramps, skin disorders, and so on that arise from out-of-control eicosanoids actually are distortions of normal protective functions. Eicosanoids are not "good" or "bad" in themselves; they're ingeniously designed for countless operations in cells and tissues. Apparently, we get into trouble when we routinely break dietary rules that came with our heritage.

Our Lifeline: Aquatic Foods

I believe one of these has to do with the fact that our ancestors in order to survive needed potable water from lakes, rivers, and streams, that many also lived near the sea, and that the easiest form of protein food to secure was, and still is, fresh- and saltwater shellfish. Clams, oysters, barnacles, crayfish, shrimp were there for the asking. Later, when we became smarter we learned to catch fish too. Skills for "mighty hunting" came much later. But even as hunters of big animals, our ancestors depended on aquatic foods including sea vegetables, another w3 source. Witness the Ohlone Indians of the San Francisco and Monterey Bay areas who, before the conquering Spaniards came, thrived on an abundance of game animals and birds and the weapons to catch them, but who ate so much shellfish that mounds of empty shells by their villages "were as much as thirty feet deep, some a quarter of a mile across--dramatic testimony to thousands of years of feasting on shellfish." (The Ohlone Way by Malcolm Margolin, 1978. Heyday Books, Box 9145, Berkeley CA 94709.)

In other words, it looks as if we developed as a species with special needs for lots of EPA and DHA--the main w3s in finfish and shellfish. When we eat these foods, even though they, too, contain plenty of cholesterol and arachidonic acid, the w3s keep a brake on eicosanoids that can make trouble. [Andrew Sinclair. "Was the hunter-gatherer diet prothrombotic?" In: Essential Fatty Acids & Eicosanoids, AOCS, 1992.]
A newer challenge has emerged regarding formulas for preterm infants whose "converting" enzymes are not yet well developed. While fish oil contains EPA and DHA, breast milk has little EPA compared with its DHA content—even the milk from mothers who eat lots of fish. Here’s where I’m dazzled by nature’s ingenuity. You see, EPA, but not DHA, diminishes arachidonic acid levels, and newer studies show that babies need lots of arachidonic in the brain and for growth. Also, the brain needs gobs of DHA but little EPA. So the mother’s marvelous milk-making machinery screens out excess EPA, yet keeps good levels of DHA (and, of course, arachidonic acid) in her milk.

The challenge for makers of formula for preterm infants is to see how close they can come to this brilliant bit of natural chemistry!

I know that lots of new breastfeeding mothers suffer from doubts about their milk—is it really nourishing, is it as good as 'scientific' formulas? Please don’t worry, ladies! They never will be able to come up with a formula that has the specific antibodies, mineral transport factors, ever-changing fatty acid mix to adjust to the baby’s needs, plus a host of other identified and not-yet-identified molecules that breast milk delivers so humbly, just as if it weren’t one of nature’s miracles.

\[ \text{FISH OIL & ASTHMA} \]

University of Wyoming scientists tested the effects of low fish-oil vs. high fish-oil supplementation on breathing capacities of a group of young asthmatics (ages 19-25). During the first four weeks, the ratio of Omega-3 (w3) EPA and DHA to Omega-6 (w6) fatty acids in their diets ('low fish-oil' period) was 1-to-10. The next four weeks, fish oil was increased more than fourfold ('high fish-oil' period), for a ratio of 1-to-2.

Nothing much happened during the low fish-oil period. But the subjects showed much increased breathing difficulty after challenge with a chemical, methacholine, compared with pulmonary function after the same challenge before the diets began.

But during high fish-oil weeks, breathing capacity began to improve markedly for 40% of those tested. The asthma patients who improved were termed "responders," in contrast to "nonresponders." Nevertheless, both responders and nonresponders showed a drop (during high fish-oil) compared with low fish-oil (month in a special group of eicosanoids made from w6 arachidonic acid, known as "4-series leukotrienes." These have long been associated, according to the researchers, with inflammatory conditions of the skin (psoriasis), lung (allergic asthma), and joints (rheumatoid arthritis).

Moreover, the scientists found a new connection between improvement in respiratory indexes and higher production of eicosanoids made from w3 EPA, known as "5-series leukotrienes." Even the nonresponders produced more of these during the high fish-oil period than before supplementation, but responders made a whopping 230% more 5-series leukotrienes than nonresponders.

In other words, a buildup of 4-series leukotrienes (from w6 arachidonic) is a recipe for trouble; while the opposite holds true for the 5-series' from w3 EPA.

The authors say 5-series competitively inhibit 4-series leukotrienes and do not elicit an asthmatic response. These findings raise the possibility that dietary supplementation with marine oils or highly enriched sources of w3 PUFAs [polyunsaturated fatty acids] may be another viable treatment modality for asthma." (K.S.Broughton et al., Am J Clin Nutr. Apr 1997; 65:1011-7.)

\[ \text{AN OLDER LADY'S WISDOM IS NEEDED, TOO!} \]

Beatrice Trum Hunter, ace nutrition journalist, sent me an article by Ann Gibbons in April 25 SCIENCE, with a note: "Happy Mothers’ and Grandmothers’ Day!" University of Utah anthropologist Kristen Hawkes and colleagues offer a novel theory as to why human females live for so many years after they stop reproducing—often as long as 40 years—in contrast to all other primates. In the year Hawkes’ group spent studying 300 Hadza hunter-gatherers in hilly northern Tanzania, they witnessed the Hadza moving from season to season and surviving almost entirely on wild resources. Women dug tubers and gathered berries and other fruit, while men hunted for game and honey.

They learned that younger children’s weight gain had to do with how much time their mothers could forage. With each new baby, a mother had less time to forage for her weaned youngsters. That’s when grandmothers, mostly in their 60s, stepped in the breach! They freed the mothers to nurse. The older women ("hardworking and incredibly fit," according to Hawkes) spent more time foraging than the young mothers and allowed their new grandbabies to nurse and thrive, and the older toddlers to stay nourished. Incidentally, Hawkes’ team found hunting by the men to be a less reliable source of food than the tubers grandmothers dug up.

So, one answer to why women have a long life after menopause may be that it made evolutionary sense for women to live to a ripe old age in order to make sure the grandbabies had enough to eat.

Gibbons writes that according to Hawkes, "such provisioning by grandmothers may allow human mothers to have babies closer together than other apes can...With grandmothers providing food, daughters can breast-feed infants for a shorter period and so bear more babies during their fertile years than primates without helpers do."

The "grandmother hypothesis" suggests that natural selection, by thus favoring a long life beyond a woman’s fertile years, also favored close family ties. Not all paleoanthropologists buy the hypothesis, saying more proof which is hard to come by would be needed. But it makes a lovely theory, doesn't it?
he'd draw cartoons for the mixed-race kids in a Japanese orphanage whenever his ship was in port. He helped his young wife of long ago overcome an impoverished background to go on to college and become a superb elementary teacher. I'm sure he was a catalytic force to students during his years as a radical young college English prof, but his real passion lay in photo-journalism which motivated him the rest of his life. He wrote both regularly and as a freelancer for L.A. Free Press, Coast Magazine, and a host of other now defunct, mostly radical publications, and also was a photo-publicist for colorful theatrical troupes. Geerdes was one of a handful of writers and cartoonists who recognized the unique cultural impact of comic strips and comic books on American life; in his articles he gave them the respectful scholarship they deserved. As the publisher of his own newsletter in that field for 22 years, he became a guru to young cartoonists, many of whom contributed logos that have become a kind of history of "underground" cartooning—in contrast to the "straight" Prince Valiant kind. He began publishing little 8-page "mini-comix," the cartoons coming in from young artists all over the country, most of whom had never been in print before. There was no money in it, just the pleasure for cartoonists of "jamming" on ideas and connecting with other artists. Clay never rejected any artist; he figured they'd gradually improve with experience. (Many did.)

When we became best friends I had reached a dead end careerwise. After a long struggle as a middle-aged re-entry that netted me a degree in nutrition science at Univ. of CA at Berkeley, I had one brief, unsatisfying job in the field, and now what?? It was Clay alone who cajoled, pushed, teased, encouraged, and finally inspired me into publishing the first Felix Letter in 1981. As you know, it's kept me in mischief ever since.

The last three years, Clay connected with a remarkable radical weekly, the Anderson Valley Advertiser*, published in Mendocino County by a rabid crew of justice-seekers, including editor and splendid curmudgeon Bruce Anderson, his

wife Ling, brother Rob, son Zack, and Mark Scaramella. The AVA suited Clay just fine. He contributed stories about life in Nebraska, Navy years, college teaching, book and movie reviews, sociological ramblings—a good mix. They published Clay's goodbye, "The Last Door," in the July 23rd edition, adding "emeritus" to his name on the Contributors' staff.

This issue of The Felix Letter is dedicated to Clayton Edward Geerdes, Jr. Long may he live in us through his writings; his cartoons which I'll continue to publish; his courage, integrity, and lifelong harvest of good deeds.

Dear Subscribers: I'm settling back into research and writing again on a regular basis, but subscriptions to the newsletter will be by the number of issues, rather than by the year. So, you may or may not get all six issues within a year (or 12 within two years), but you'll always get the number of issues you've paid for, okay?

Illustrations by the late Clay Geerdes and other artists as noted. 
THE FELIX LETTER, P.O. Box 7094, Berkeley CA 94707, has been published independently, irregularly, and improvidently by Clara Felix since 1981, supported solely by subscriptions. Descriptive list of available issues plus sample, $1. Six-issue subscription $12, 12-issue sub $22. U.S. & Canada (U.S. funds only). 
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