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the FELIX Letter

A COMMENTARY ON NUTRITION

2001

LET'S LOOK AGAIN AT THE CHOLESTEROL, SATURATED FATS, HEART DISEASE CONNECTION

You're seeing a big push by pharmaceutical giants to have statin drugs granted over-the-counter status, in sync with their campaign to persuade doctors and John & Mary Q Public that low cholesterol = freedom from heartache. I confess that while I never bought the notion that cholesterol is the villain in coronary heart disease (CHD), until recently I *did* feel concern when people showed so-called high LDL- and low HDL-cholesterol readings on blood tests. I even devoted a newsletter (*FL #36* in 1987) to ways of 'bettering' these values. (Rereading, I'm relieved the methods I pushed were sound, whether they did or didn't drop your cholesterol!)

My wake-up call comes from keen scientists who've done laborious homework. For one, there's Uffe Ravnskov MD, a Swedish clinician with a PhD in chemistry, whose book *THE CHOLESTEROL MYTHS,* was first published in his country in 1991 and updated in 2000 to include analyses of statin trials beginning in the late 1980s.[1]

The Lipid Hypothesis

Since the early 1950s, he says, researchers seeking the causes of atherosclerosis and heart disease have focused on "the diet-heart idea, sometimes called the lipid hypothesis." Its proponents say CHD is the third and final step of a three-step process:

Step 1. High blood cholesterol is caused by an atherogenic diet, i.e., one high in cholesterol and saturated fat (found mainly in animal products, such as meat, milk, eggs, but also in palm oil and coconut oil) and too little polyunsaturated fat.

Step 2. High blood cholesterol is the main cause of atherosclerosis.

Step 3. Atherosclerosis causes CHD by blocking the blood vessels of the heart.

Ravnskov writes: "At first glance, the diet-heart hypothesis does indeed appear simple, logical and well-founded. It is also an attractive idea, because it almost promises that death from coronary heart disease can be prevented. If animal fat [in diet] and high blood cholesterol are the villains, then cholesterol-lowering diets and cholesterol-lowering medicines appear to be wise choices."

Not Such Pure Science

The medical/nutrition world has been swept along by these concepts for close to 50 years, beginning with Ancel Keys' 1953 statistical 'proof' that high-fat food was *the* culprit in CHD. In undertaking painstaking reviews of hundreds of pertinent studies, Ravnskov has performed a rare enlightening labor not just for the public but for his fellow-medicos. Naive I'm not, but I confess to being shaken by his disclosures of obfuscation, outright errors, and/or misleading conclusions in an unseemly number of the supportive papers. The lipid hypothesis has proven to be unscientific and "hopelessly incorrect," Ravnskov says, yet somehow gifted with "eternal life"!

1. *THE CHOLESTEROL MYTHS. Exposing the fallacy that saturated fat and cholesterol cause heart disease.* NewTrends Publishing, Inc., Washington DC. www.NewTrendsPublishing.com Tel: 877-707-1776

Mysterious Scientese!

If you're like me, you blunder with a wing and a prayer through numbing graphs and statistics in typical research papers. I'm grateful to the good doctor for clarifying mumbo-jumbo -- explaining point by point what charts and graphs really mean, what terms like 'correlation coefficients' actually signify [see later], and how statistics commonly are manipulated to 'prove' a point.

"Risk factors," for instance. High cholesterol actually *is* a 'risk factor' for CHD, particularly for men (not women) **whose serum cholesterol is over 350 mg/dl.** But so are several hundred other risk factors, "including smoking, overweight, high blood pressure, lack of exercise, psychological stress, baldness, snoring and eating too much or too little of a steadily increasing number of various food items, *but the cause of the disease is still unknown* [my emphasis CF].... Factors that are statistically associated with a disease are called *risk factors.* A risk factor *may* be the cause of the disease, but most often it is not."

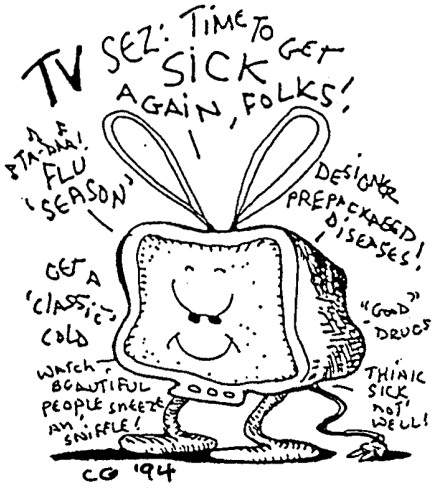
He writes: "By 1998, a total of 27 studies had been published including 34 groups (cohorts) of patients and control individuals.... In three of these 34 cohorts, patients with coronary disease had eaten more animal fat than the control individuals, and in one cohort they had eaten *less.* In the rest of the groups -- 30 in all -- investigators found no difference in animal fat consumption between those with heart disease and

those without. In three cohorts the patients had eaten more polyunsaturated vegetable oils than the control individuals, and in only one they had eaten less.

"In the studies mentioned above, the researchers try to press the figures down into the cholesterol shoe, but neither heels nor toes will fit...."

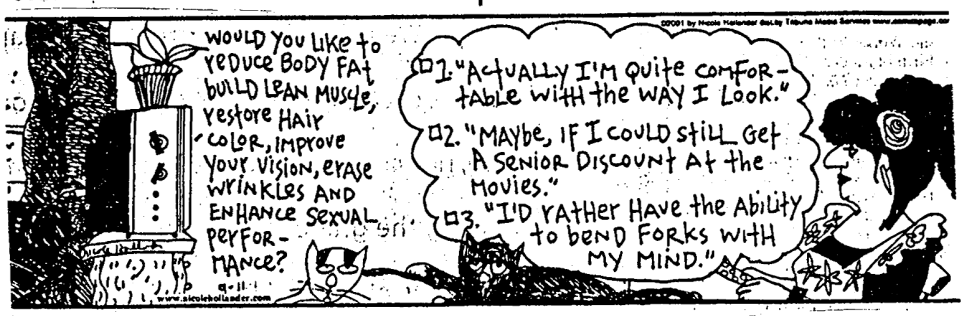
Here's one sample. A paper put out jointly by the American Heart Association [AHA] and the National Heart, Lung and Blood Institute [NHLBI] stated: "...showing the link between diet and CHD, particular impressive results [were produced in] the Western-Electric, the Honolulu Heart, the Zutphen and the Ireland-Boston studies."

"Yet the tables published in these studies," Ravnskov writes, "showed that only in the Honolulu heart study had the patients eaten significantly more saturated fat. But they also consumed significantly more polyunsaturated oils, just the opposite of what we have been led to expect."



High-cholesterol foods are to be avoided, according to the lipid hypothesis, because they, too, raise blood cholesterol. (Repeated studies refute this. As you eat more cholesterol, your body makes less.) Yet the doctor explains that in 10 major studies, the mean consumption of cholesterol actually was a little *lower* in subjects who developed CHD than in the lucky folks who didn't.

SYLVIA *Nicole Hollander*



Myth: That High Blood Cholesterol Causes CHD

Ravnskov hollows out leading studies supporting "Step 2," showing how statistic 'games' add up to fragile science.

For example, in the Framingham, Massachusetts study, longterm coronary and total mortality (death) ***increased by 11%*** for each **1% *reduction* i**n blood cholesterol. Yet the AHA and the NHLBI wrote in a 1990 joint statement: "The results of the Framingham study indicate that a 1% reduction...of cholesterol [corresponds to a] 2% reduction in CHD risk."

Ravnskov writes: "Most supporters of the diet-heart idea think the increased risk of [CHD] is present at all cholesterol levels...The truth, were it known, would send pharmaceutical stocks plunging. In most studies, the increased risk is present only above a level of cholesterol that includes just a small percentage of the total population. And women can stop worrying immediately because high cholesterol is not a risk factor for the female sex..."

More Good News

"In fact, it seems more dangerous for women to have low cholesterol than high. Dr. Bernard Forette and a team of French researchers from Paris, found that old women with very high cholesterol live the longest. The death rate was more than five times higher for women who had very low cholesterol. In their report, the French doctors warned against cholesterol lowering in elderly women, but they could as well have warned against cholesterol lowering in any woman or, to be more precise, in anyone at all.

... "Thus, high cholesterol is said to be dangerous for Americans but [based on medical studies] not for Canadians, Stockholmers, Russians or Maoris. High cholesterol is said to be dangerous for men, but not for women; it is said to be dangerous for healthy men, but not for coronary patients; and it is said to be dangerous for men of 30, but not for those of 48 [and older]. And high cholesterol may even be beneficial for older people. Such discrepancies indicate the association between high cholesterol and [CHD] is not due to simple cause and effect. The most likely interpretation is that high cholesterol is not dangerous in itself but a marker for something else."

More Evidence vs "Step 2"

It turns out a number of careful post mortem studies found no correlation between amount of cholesterol in blood, and actual degree of atherosclerosis, i.e., hardened 'sclerotic' plaque buildup in arterial walls of the deceased.

Arteries of those with low blood cholesterol were just as sclerotic as those whose cholesterol had been high!

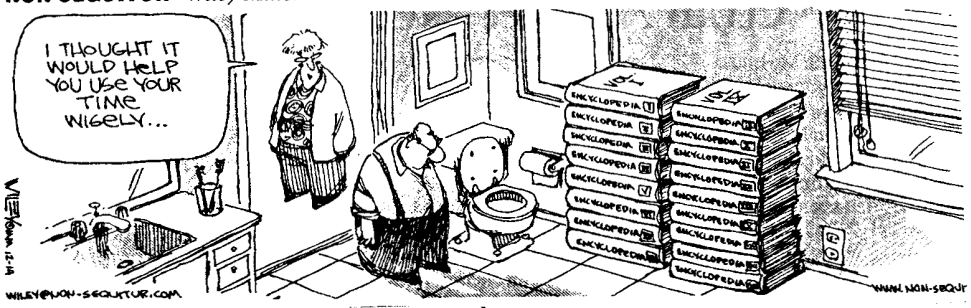
Statistics Demystified

A 'Correlation coefficient' expresses the strength of an association between any two variables. For instance, in the famous Framingham study, the two variables that were examined in 281 deceased participants were **blood cholesterol** and **degree of atherosclerosis**. If both were invariably associated, the **correlation coefficient would have been 1**.

Actually, Ravnskov writes, it "was only 0.36. Such a low coefficient indicates a desperately weak relationship" between cholesterol and atherosclerosis. Usually, scientists demand a much higher correlation coefficient before they conclude that there is a biologically important relationship between two variables."

In their report the Framingham researchers didn't feel it necessary even to comment on the low correlation coefficient. Not only "was the correlation coefficient trivial, but this study, funded with millions of taxpayer dollars by the National Institutes of Health, could have a major impact on

NON SEQUITUR *Wiley Miller*



national health care and the American economy. If there was no connection between cholesterol and atherosclerosis -- as all the previous studies had shown -- then there was no reason to bother about lowering cholesterol or changing the diet. Billions of taxpayer dollars could have been spent more wisely than in cholesterol-lowering measures for healthy people."

Ravnkov lets the air out of another lipid-hypothesis balloon: that lowering cholesterol in CHD patients *will halt or reverse atherosclerosis.* Study after study with the newer tool of coronary angiography allowing us to see inside blood vessels with X-rays reveal that coronary atherosclerosis "gets worse just as fast or faster when cholesterol goes down as when it goes up..."

We know that high cholesterol is one risk factor for CHD. "To prove that high cholesterol is the villain -- and not just an innocent bystander -- demands that a *change* in the cholesterol concentration in each individual is followed by a *change* in degree of atherosclerosis in the same direction. But in all studies these changes occur haphazardly."

Cholesterol: Villain or Friend?

Your body simply wouldn't go to the trouble to make cholesterol if it weren't basic to survival. This lipid is an essential structural component and stabilizer of your cell membranes, as well as precursor to vitamin D, bile salts, and all your corticosteroid and sex hormones. *It's also a repair molecule for injuries to your tissues, including those to blood vessel walls.* Mother's milk is especially rich in cholesterol to ensure the baby's good nerve and brain development. That's why egg yolk--the chick embryo's food--is rich in it too.

The question scientists should be asking is what turns this priceless sterol into a rogue? -- if indeed it really *is* playing a deadly role in your arteries.

Louis H. Krut, MD, of St. Louis Univ. Medical School says there "is a remarkable dearth of concepts on how cholesterol may promote atherogenesis, and even these few are incompatible."[2]

The bulk of the plaque in arteries, he writes, "is made up of fibrotic elements, with the lipid in its core [cholesterol, etc.] often contributing relatively little to its mass." It's the fibrous tissue that's most responsible for narrowing the lumen of arteries, which is the critical factor as far as compromised blood flow is concerned. "Cholesterol in plaque should therefore hold little interest for us unless it can be shown to be sclerogenic."

Krut continues: "The other pillar of the lipid hypothesis, namely that lowering plasma cholesterol will clear cholesterol from the plaque, thus promoting its regression and consequently preventing clinical sequelae, has been our *raison d'etre* for 4 decades. It must therefore be noted that cholesterol in plaque is contained in a 'gruel' of pultaceous necrotic debris....It is inconceivable how lowering plasma cholesterol could effect clearance of cholesterol from such a plaque with regression of its fibrous component. The fibrous layer surrounding the lipid core is generally free of lipid. It is therefore apparent that whatever the contribution of cholesterol to atherogenesis may be, this contribution must occur early in plaque formation. Once the fibrotic elements begin to generate, no further deposition of plasma lipid occurs, nor indeed is this required for fibrosis to progress."

Hmmm...

2. L.H. Krut. On the statins, correcting plasma lipid levels, and preventing the clinical sequelae of atherosclerotic CHD. *American J of Cardiology,* Vol 81, April 15, 1998.

Less Than Pure Motives?

In light of what Krut tells us and its confirmation in studies reviewed by Ravnskov, what's the justification for today's hugely publicized campaign to shrink everyone's cholesterol levels?

Here's what John R. Lee, MD, writes in his June 2001 *Medical Letter* :[3]

"Last month the big health story in the media was that the National Cholesterol Education Program (NCEP) has decided that 'normal' levels for cholesterol should be lowered even more, and that as a result, 20 percent of all American adults should be taking drugs to lower their cholesterol levels. **This has got to be one of the most blatant health scams I've seen in my more than 40 years in medicine.**

"First, you might ask, what is the NCEP? It's a branch of the National Heart, Lung, and Blood Institute (NHLBI), which is part of the Federal government's National Institutes of Health. The supposed mission of the NCEP is to get Americans to reduce their cholesterol levels in order to reduce the incidence of heart disease, even though a direct link between high cholesterol and dying from a heart attack has never been made.

"(**High cholesterol levels can be one of many symptoms of heart disease, just as a runny nose can be one of many symptoms of the flu.** Forcing cholesterol levels down with drugs without addressing the underlying conditions that caused the symptom in the first place, and then declaring that the drugs reduce heart disease, is like taking an antihistamine when you have the flu and then declaring that you've cured it because your nose stops running.)"

Dr. Lee adds: "I won't go into any detailed speculations about **who's paying whom to tell Americans that 20 percent of them need to be taking a cholesterol-lowering drug,** but suffice it to say that these recommendations have to do with money, and not with your optimal health..."

3. *The John R. Lee, M.D. Medical Letter* is one of my favorite health newsletters, and Dr. Lee is one of my medical heroes, not the least for pioneering the dialogue on perils of estrogen dominance and progesterone deficiency. PO Box 84900, Phoenix AZ 85071, Tel: 800-528-0559, or e-mail: www.johnleemd.com

The statin story is not improving. In August the German drug-maker Bayer AG withdrew its statin drug from the U.S. market after Baycol (cerivastatin) was found responsible for 31 deaths from a muscle-destroying side effect. Soon afterward, consumer watchdog Public Citizen petitioned our government "to require manufacturers to give warning brochures to the 12 million Americans who take those medicines -- statins -- telling them to quit the pills at the first sign of muscle pain or weakness" [*SF Chronicle,* Aug 21].

Loss of Coenzyme Q10

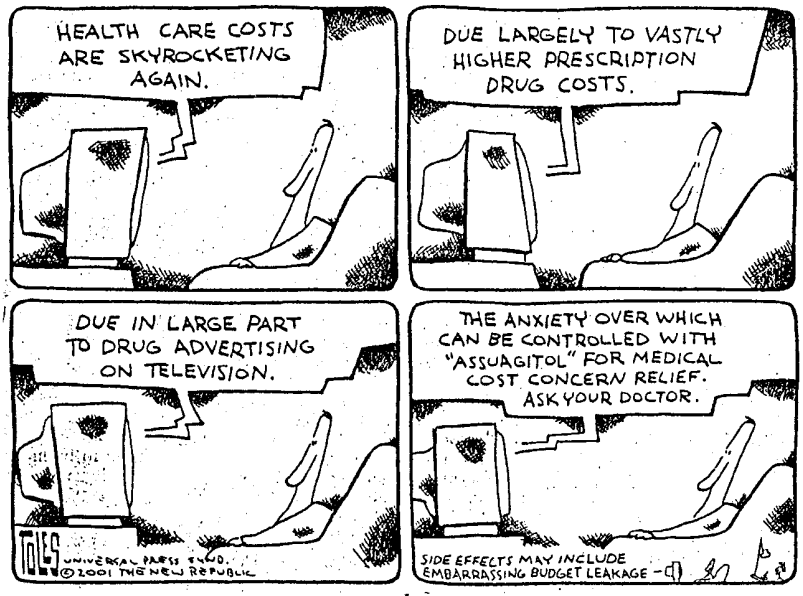
The *Chronicle's* medical writer countered on August 26th with a report about the hordes of heart disease patients being saved by statins. No mention was made that statin drugs inhibit your body's synthesis not just of cholesterol *but of a major energy-making molecule: coenzyme Q10.* (CoQ10 also is named 'ubiquinone' because normally it's ubiquitous to all your cells, not just to muscle.) Nasty effects from this inhibition are so well-documented that at least one statin-maker patented the inclusion of CoQ10 in its formula.

Emile G. Bliznakov MD writes that CoQ10, besides being used for energy production in each of your cells, also "stabilizes cell membranes, thus preserving cellular integrity and function."

Now hear this:

"A third, well-documented action is as a scavenger of reactive oxygen species, *thus preventing oxidative injury to DNA, lipids, proteins, and other molecules. This retards or prevents the development of many cardiovascular, neoplastic, and possibly neuro-degenerative disease states.* [My emphasis. CF]. Ironically, the attempt to reduce cardiovascular morbidity and mortality with statins is partially negated by lowering the CoQ10 level, which is essential for optimal cellular function.[4]"

4. E.G. Bliznakov & David J. Wilkins PhD. Biochemical and clinical consequences of inhibiting CoQ10 biosynthesis...*Advances in Therapy,* July/Aug 1998, Vol 15 No. 4. Reprint requests: EG Bliznakov, 2801 North Course Drive (H-205), Pompano Beach FL 33069.



A Price to Pay?

In six major trials in which statins were prescribed for about 4 to 6 years, Dr. Uffe Ravnskov points out that while there were fewer deaths from CHD in the statin-takers than in control patients, "there was no association between the degree of cholesterol lowering and the outcome." He offers the notion that *something* about the statins may work to benefit CHD, "but their effect is not due to cholesterol reduction." Actual numbers of reduced CHD deaths ranged from small to trivial; and in one trial (of healthy people with normal cholesterol) there were a few more all-cause deaths in the statin-takers.

Dr. Louis Krut[2] puts it another way. In the supposedly very successful simvastatin trial, where the calculated average extension of life in the treated CHD group was 0.065 years (24 days) in 5.4 years, he writes: "If we were to "set a very modest goal to extend their average life by only 1 year, it would require them to take simvastatin for 83 years."

In another 'successful' trial in Scotland of healthy people with high cholesterol, "their calculated average extension of life after 5 years on pravastatin is about 1 week. To extend their average life by 1 year would require them to take pravastatin for 260 years." (!)

The capper, Ravnskov says, is that statins may stimulate *cancer* (Newman TB, Hulley SB. *JAMA,* 1996, 55-60). "Their review showed that the blood levels that caused cancer in rodents were close to those seen in patients taking the statin drugs. Because the latent period between exposure to a carcinogen and the incidence of clinical cancer in humans may be 20 years or more, the absence of any controlled trials of this duration means that we do not know whether statin treatment will lead to an increased rate of cancer in coming decades....There is good reason to exercise caution...because in the CARE [pravastatin] study breast cancer was indeed more common among those who took the drug than in the control group."

CF Caution: Could diminished CoQ10 protection against neoplastic disease be a factor? Statin-takers, make sure your pills contain CoQ10 -- *and if not, take a daily supplement.* Food is not an adequate source. Nonprescription CoQ10 is recommended by alert clinicians in amounts from 50 to 300 milligrams daily.

And Now From Japan...

As I struggled to squeeze all my info-glut into *FL* dimensions, *Lancet* (August 4, 2001) arrived, bringing a topper. Beginning in 1965, the Honolulu Heart Program had periodically examined cholesterol, etc. in 8000 Japanese/American men, aged 45 to 68, living in Hawaii. During 1991-1993, changes in cholesterol, etc. between the 3rd and 4th exams were

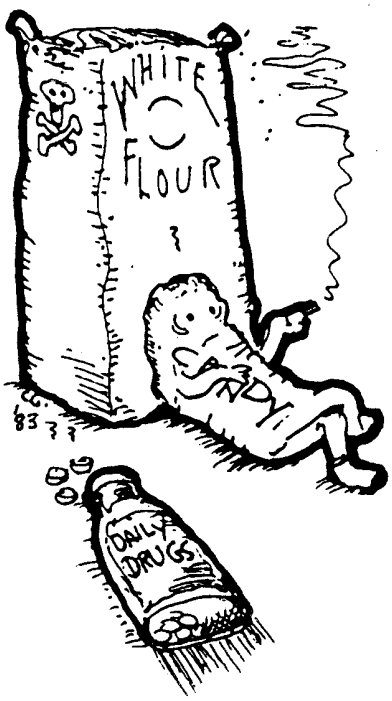
assessed in 3741 of the men, now aged 71 to 93. By 1996's end, 727 of this cohort had died from all causes. Here's what the doctors learned:

"Our data accord with previous findings of increased mortality [death] in elderly people with low serum cholesterol, and show that long-term persistence of low cholesterol concentration actually increases risk of death. Thus, the earlier that patients start to have lower cholesterol concentrations, the greater the risk of death...

"...There are few studies that have cholesterol concentrations from the same patients at both middle age and old age. Although our results lend support to previous findings that low serum cholesterol imparts a poor outlook when compared with higher [ones] in elderly people, our data also suggest that those individuals with a low serum cholesterol maintained over a 20-year period will have the worst outlook for all-cause mortality.

"...[I]n view of our data, and those of others, is there scientific justification for attempts to lower cholesterol to concentrations below 4.65 mmol/L [~180 mg/dL] in elderly people? We believe that until more information about these complex relations is available, prudence dictates a more conservative approach in this age group." [5]

5. I J Schatz et al, Cholesterol & all-cause mortality in elderly people from the Honolulu Heart Program: a cohort study. *Lancet* 2001; 358: 351-55.



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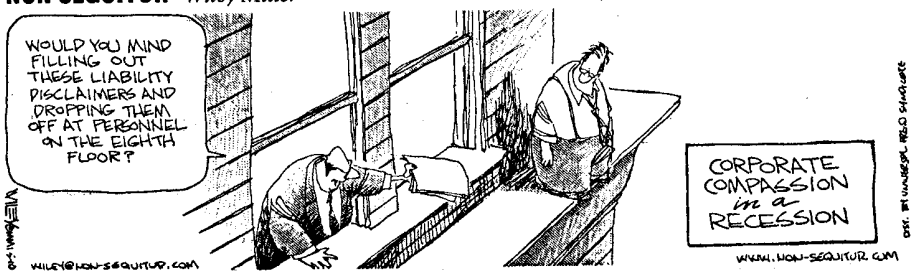


"Guts 'n Grease"

For 99% of the time that we inhabited the earth we were solely hunter-gatherers, cherishing wild plant foods *and* flesh food (fish and shellfish included), consuming all organs and fat -- even cracking leg bones and skulls of herbivores killed by animals, to get to the fatty marrow and brain. Closer to our time, first-person accounts tell how American Indians always sought out fatter game, utilizing every scrap of fat in traditional ingenious recipes.

In "Guts and Grease: The Diet of the Native Americans," [6] Sally Fallon and Mary G. Enig PhD offer a marvelous rundown of this preference, its implementation, and how it affected our First People. Prior to colonization the absence of degenerative diseases was notable; skeletal remains show "a virtual absence of tooth decay, arthritis and any other kind of bone deformity....The early explorers consistently described the native Americans as tall and well formed...The men could run after a deer for an entire day without resting and without apparent fatigue..."

Ye Gads, Maybe POLY-GLUT Is Doing Us In!

My home is one vast filing cabinet -- 'though blooms, paintings, photos, tapestries, etc. soften the stern effect. So there at arm's reach was anthropologist H. Leon Abrams' great paper: "Vegetarianism: An Anthropological/Nutritional Evaluation." This 35-page review, which appeared in the *J. of Applied Nutrition* (Vol. 32, No. 2) ***in 1980,*** rips great holes in the 'lipid hypothesis.' From our beginnings as a species, flesh food and its fats, together with plant foods, enabled us to thrive,

6. In *Wise Traditions,* Spring 2001, pp40-47. This no-holds-barred quarterly will educate, amuse, and sometimes infuriate you, but it's a *must* for all thinking people. Contact The Weston A. Price Foundation, PMB #106-380 4200 Wisconsin Ave NW, Washington DC 20016.

but Abrams insisted the same could never be said of the new, unprecedented consumption of 'naked' polyunsaturated vegetable oils, along with trans-fatty acids from partially hydrogenated oils and margarines, which the experts urge as substitutes for animal fats. Instead of 'saving' our hearts, he said, lavish intake of these substances is bringing on premature aging of cells -- even cancer.

The oils and margarines Prof. Abrams wrote about contained mostly omega-6 (w6) fatty acids -- still do. In 1980, research on w3 fatty acids, or on the mysterious prostaglandins derived solely from w6 and w3, had barely begun to make waves. Today we have solid evidence targeting polyunsaturate overkill as a player in CHD.

You can fill your polyunsaturate w6 requirements nicely from regular intake of seeds, whole grains, and nuts. Plus, you get built-in antioxidants that protected the seeds, etc. from rancidity.

When, instead, you consume lots of 'naked' oils stripped from seeds, your cellular membranes become increasingly unsaturated and less protected by antioxidants. *Polyunsaturated lipids are supervulnerable to peroxidation, i.e., rancidity;* rancid lipids can churn out highly reactive 'free radicals' (molecules with unpaired electrons). Free-radicals play havoc in tissues, and, yes, *free-radical damage in arteries is one factor in atherosclerotic plaque buildup.*

Saturated fats, by the way, are *not* subject to peroxidation and do *not* form free radicals in your tissues.

Protection - or Peril - from Prostaglandins

Just since the '80s, we learned certain localized hormones known as prostaglandins (also called eicosanoids) have a lot to do with the wellbeing of your cardiovascular system. Out of this has come one of the all-time great discoveries in nutrition: *you control how these powerful molecules act by the fats you eat.*

For instance, specific eicosanoids your body makes *from w6 fatty acids* can set off emergency actions: blood clotting, squeezing down arteries, and inflammation -- ingenious mechanisms to stop you from bleeding to death from cuts, and to thwart potential infection.

However, when people habitually eat too many w6s and too few w3s, the balance of this masterful system is knocked out.

For instance, the w3s squelch 'runaway' w6 eicosanoids. Without this critical regulation, blood clots can become boulders, and temporary inflammatory weapons against infection can turn into chronic, simmering inflammation in blood vessels (and other sites).

It's estimated our Paleolithic ancestors consumed small but about equal amounts of w6 and w3 fatty acids, along with plenty of saturated ones. *People in the USA today are taking in from 10 to 20 times more w6 than w3.* This finally is being recognized for what it is: a recipe for trouble -- and not just in the heart.

Harumi Okuyama[7] and colleagues' wrote a trailblazing paper in 1997 on the sharp upswing of 'Western-type' ailments in modernized postwar Japan, including allergies, bronchitis, cancer *and heart disease*, which they carefully trace to increased w6 oil consumption.

Before World War II, the Japanese consumed only **2.8 times more w6 than w3 fatty acids**. Now, they're eating over **4 times more**. If that small rise is enough to increase heart disease, etc., what can we say about the 10-20 times more w6 than w3 intake in the U.S.?

The w3s have been my 'beat' since I first began covering their story in 1983 (*FL#14*), but I just learned from a new study [there are many thousands now] that besides a balanced w6/w3 intake, we need enough *saturated fats* before our bodies can store and use w3 fatty acids effectively.

Our ancestors knew something we're having to learn all over again.

7. Harumi Okuyama et al. (1997) In *Progress in Lipid Research,* Vol 35, No. 4, pp 409-457. I reviewed this electrifying study in *FLs94/95.*

Pickles/Brian Crane

You Make Sat-fats 'Cause You Need 'Em

Meanwhile, scientists like Mary G. Enig PhD struggle to reinstate slandered saturated fats to the place of honor they always had in traditional diets -- ours, too, until 75 years ago. She writes: " ...the body needs saturated fatty acids for at least half, and sometimes much more, of the fatty acid part of the phospholipids that form the membranes of the cells. For those who don't understand why the human body makes saturated fatty acids -- it is because the saturated fatty acids are *required*...and for some people, the fats in the diet are either too polyunsaturated or too low."[8]

Mother's milk is almost 50% saturated fats, as the baby needs these fats to firm up cell membranes comprising its tissues. Too many polyunsaturated lipids in membranes make for limp, unstable tissues -- and vulnerable to free-radical damage besides.

Saturated fats that you eat, or make in your own tissues, come in a variety of carbon-chain lengths. Breast milk is rich in easily digested medium-chain ones, like *lauric acid* -- and so are the much-maligned saturated tropical fats -- coconut and palm kernel oil. It so happens lauric acid is noted for its superb antimicrobial properties -- one reason for breastfed babies' resistance to infections. Palm kernel oil and coconut, of course, have been giving people in the tropics this protection for thousands of years.

Results of the famous Framingham study, Enig writes, "have been interpreted by many in ways so as to link saturated fats to CHD." Yet here's what she tells us its director, Dr. William P. Castelli, wrote in 1992: "...In Framingham, for example, we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least, and were the most physically active." **Wow!!**

When Dr. Uffe Ravnskov's book became the subject of a television show in Finland in the early '90s, its attack on the lipid hypothesis so infuriated participants, they literally *burned* the book on television. If I've shaken you up as well, take a deep breath, calm yourself, try not to shoot the messenger.

Remember, saturated fats in food *and in your tissues* are immune to oxidative attack.

They're solid, man. ❐

8. *Know Your Fats: The Complete Primer for Understanding the Nutrition of Fats, Oils, and Cholesterol* by Mary G. Enig, PhD. Bethesda Press, Silver Spring MD. Tel: 301-680-8600. 1st printing April 2000.

VITAMIN D-(LIGHT-FULL)

I'm rhapsodic over good stuff that's pouring in about a vitamin most of us thought was old hat, i.e., necessary but boring. Prof. Nick J Reynolds et al. of England tell in the June 23, 2001 *Lancet* of substantial improvement in the common, stubborn skin disorder, **atopic eczema,** in most of their patients who received gradually increased whole-body exposure (twice a week for 12 weeks) to UVB phototherapy , but not in those who got UVA instead. Skin exacerbations and itching lessened, sleep improved, and these effects were maintained 3 months after phototherapy ended.

Only UVB rays make vitamin D in your skin. If patients' serum levels of D had been tested, I wonder how these might have correlated with the improvements? The UK lies between latitudes 50°-51° North at the Channel, to about 59° at the tip of Scotland. It's a battle to get enough D's even from summer's UVB rays at those latitudes. (It's not easy even here at latitude 38° in our Bay area, except in the middle of a cloudless summer day. UVB rays don't penetrate clouds, fog, smog, or glass, alas.) Researchers in Helsinki, Finland (60° North) are concerned about low bone-mineral density in the forearms of adolescent girls, linked to the girls' low serum 25(OH)D in winter. (Blood serum levels of 25-hydroxyvitamin D are accepted as the best indicator of a person's D status.)

But what could be the reason(s) for "the prevalence of osteomalacia and rickets" in tropical and subtropical places, where UVB is king? In the August 2000 *Am J Clinical Nutrition,* medical researchers R. Goswami et al. studied sun exposure, calcium intake, serum 25(OH)D, and other parameters in "healthy" groups of people: soldiers, doctors & nurses, pregnant women, etc. in New Delhi, India (latitude 28° N).

Avoiding the Sun

The only ones who didn't have shockingly low--I mean, almost off-the chart--25(OH)D, were the soldiers, who spent hours training outdoors. But even the soldiers, like the others, exposed only face and hands (about 10% of body surface) and had barely adequate serum levels of D. The others avoided sun except for about 25 minutes a day in summer.

Pickles/Brian Crane

But darker persons need more time than fair-skinned ones -- as much as 4 to 8 times more -- to make the same amount of D from the sun's UVB rays.

Result? Their "subnormal" 25(OH)D was coupled with *elevated parathyroid hormone* (PTH). The latter appears when blood calcium levels are too low -- the trigger for pulling calcium out of bones to get blood levels up.

The authors comment: "Such chronic and insidious PTH-dependent bone resorption is known to be relevant in the pathogenesis of osteoporosis."

So, "healthy persons in Delhi remain vitamin D deficient because of skin pigmentation and inadequate direct sunlight exposure. When exposed to factors that adversely affect vitamin D and bone mineral metabolic status, an imbalance in bone mineral metabolic homeostasis results."

The pregnant women in the study not only had unbelievably low vitamin D levels, but consumed only a third as much calcium as the others! Sadly, the poor ladies developed actual softening of their bones -- *osteomalacia.*

That's another name for rickets, an 'old' ailment that's cropping up in little black kids in sunny Texas -- who probably stay indoors too much watching tv, and also don't get enough calcium (and *magnesium.* Both minerals are needed for D to work right.)

Another Plus for Saturated Fats

Long, long ago, when prehistoric tribes migrated away from the equator, their dark skins got lighter to absorb the scarcer UVB rays. Way up north, the lighter their skin the better they survived so the trait got passed along. But people needed more D than skimpy rays provide in northern latitudes. *So they ate shellfish, herring and salmon, and they hunted mammoths and seals and ate all the vitamin D-rich flesh, fat, and organs.*

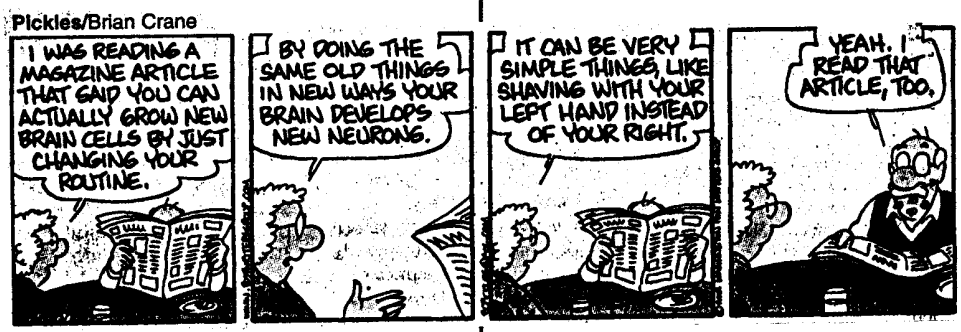
Krispin Sullivan writes:[9]

"Fish make vitamin D from the precursor of vitamin D found in algae. In the higher mammals, vitamin D is made from precursors in lichen and green grass. Reindeer fat, for example, is a good source of vitamin D because reindeer feed on lichen. Vitamin D will be found in the butterfat of ruminant animals that feed on green grass, and in pigs that spend time in the sunlight.

"...So-called primitive peoples instinctively chose vitamin-D-rich foods including the intestines, organ meats, skin and fat from certain land animals, as well as shellfish, oily fish and insects."

9. Krispin Sullivan, CN. The miracle of vitamin D. *Wise Traditions,* Fall 2000, pp 11-20.

Pickles/Brian Crane



Until 60-70 years ago, foods like the above used to be everyday fare in the U.S. [well, maybe not mammoths, seals, reindeer, and insects], and we weren't scared to eat fat and organs from animals and fowl that ate grass, spent time in the sun, and provided us with vitamin D.

Nowadays, thanks to the long reign of the 'lipid hypothesis,' we're low in D, and wallowing in poly's.

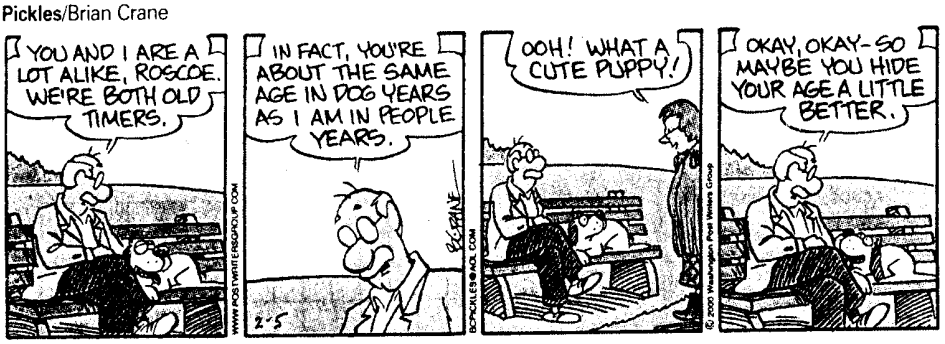
That's progress?

Repairing Bods, Young & Old

But cheer up, here's good news. I've been filling my D-reservoirs for about 5 months* and my on/off arthritic knee is healed, my torso and back are springier and my hair's thicker. (Not bad for a nonspring chicken, right?)

*As vitamin-D consciousness grows, people may start supplementing grandly without getting tested or making sure their calcium and magnesium intake is optimal. ***This is neither wise nor safe.*** An inexpensive blood test for 25-hydroxyvitamin D became available recently from a reputable laboratory, LabOne, in Kansas. They have collection facilities in clinics around the country, so call them (800-646-7788) to learn if there's one close to you; if so, ask your health professional to write you a prescription for **LabOne Test #3287 for 25-hydroxyvitamin D**. (LabOne will fax results to your MD, etc. if the fax number is on the prescription.)

Pickles/Brian Crane



The thread linking these sundries is that both too much *and* too little vitamin D encourage the dumping of calcium into our soft tissues, where it doesn't belong. Mischief happens that's hard to pinpoint or diagnose.

But *optimal vitamin D apparently facilitates the clearance of unwanted calcium from soft tissues.*

Krispin Sullivan[10] describes a remarkable Scottish study (A. MacPherson et al., *Analyst,* March 1995; 120, pp 871-5) in which researchers analyzed the calcium content both in men's *arteries* and in their *beard hair.* The more plaque (calcium) they found in arteries, *the less calcium there was in beard hair.* Ninety percent of men who'd had a heart attack (myocardial infarction) had low beard calcium.

Vitamin D raised beard calcium and this rise continued as long as the vitamin was consumed. Soon after stopping the D, beard calcium content fell to presupplement levels.

[Question: Was it getting dumped back in the poor guys' arteries???]

Not only is this further evidence that unwanted calcium in arteries is associated with heart attacks, but that low levels of D play a big part in the whole nasty displacement.

10. Krispin Sullivan, CN. *Practical Nutrition for the 21st Century.* Also, her *Preliminary Report on the Importance of Sunlight and Vitamin D.* Either can be ordered from this sterling educator at http://www.sunlightandvitamind.com or by phone: 415-488-9636. Her vitamin D book will be out next year, hurrah!

But, of course, calcium in the right places helps *bones, hair, and nails* to be strong. That's why my hair is thicker, Krispin explained. (A friend tells me his nails have gotten almost too tough to clip since he tanked up on D.)

D'you suppose the (conjectured) extra calcium in my hair could have come from muscles and joints that used to ache??? ❐

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

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