MAMA EARTH & PAPA SUN

Now that it's winter, all you folks living at latitudes higher than 30° north of the equator can wave bye-bye to a goodly portion of the sun's ultraviolet B rays—the only ones with the right length and energy to goose your skin cells into making vitamin D. (Of course, if you live upside-down at latitudes greater than 30° below the equator, my winter may be your summer and all bets are off.)

Most stories in the popular press tell you to stay out of the sun to avoid skin cancer; but I'm seeing more and more alarms in medical journals about widespread vitamin D-deficiency.

In this October's Am. J of Clinical Nutrition [Vol 74, No. 4] researchers doing a random sampling in Beijing of 1248 Chinese girls 12 – 14 years old, found too many with poor vitamin D status, the deficiency's effects magnified by their abysmal calcium intakes: ~600 mg/day. The Chinese RDA is 1200 mg.

Beijing at 40° latitude North has scarce UVB rays in winter. Not surprisingly, in winter far more than summer, many girls get muscle spasms in legs or toes along with very low plasma 25-hydroxy-vitamin D and calcium. If a girl doesn't load up on sunshine and calcium in the summer, the researchers say her outlook for the winter will not be good. ...Not so good for her future, either, since adolescence is a critical time for building dense bones.

While active rickets (osteomalacia, or softening of bones) wasn't seen, many girls were knock-kneed and some had "pigeon chest"—abnormally protruding chest caused by a deformed breast bone—signs of rickets when the girls were very young. The authors recommend at least 600 mg calcium and 200 IU vitamin D daily during the winter for teenage girls.

Skinny, in my book. But that's also the current DRI [Dietary Reference Intake] in the U.S., half the previous RDA. That's progress?!! At least the DRI for folks 70 and older was pushed up from 200 to 600 IU in hopes of reversing the current (costly!) epidemic of osteoporosis and fractures in the elderly.

In the Far, Far North...

Adults require 4000 IU or more to stay healthy, but that's from all sources: sunlight, food, and supplements. Which brings me to another story, this time from Finland. It begins about 35 years ago, when thousands of pregnant ladies in far north Oulu and Lapland who were expecting their babies in 1966 were enrolled in a huge "birth-cohort" study. Data were gathered from more than 10,000 before each birth. Afterwards, health charts from routine clinic exams provided information on the infants' health, including how much and how regularly each of the ~10,000 had been supplemented with vitamin D during its first year of life. (E. Hypponen et al., The Lancet, Nov. 3, 2001, Vol 358, 1500-03.)

In the 1997-98 follow-up, 81 were found to have been diagnosed with insulin-dependent Type 1 diabetes (median age at diagnosis 14 years; it's also called "juvenile diabetes"). The researchers say the disease was associated "with low intake of vitamin D and signs of rickets during the first year of life."

Type 1 diabetes is an autoimmune disease; i.e., the body's immune system "defenders" go haywire and attack insulin-making beta cells of the pancreas. "We believe vitamin D might somehow inhibit the autoimmune reaction targeted towards the [beta] cells of the pancreas," the authors write.

Although type 1 diabetes is comparatively rare, Finland "has the highest reported incidence" in the world. Moreover, it's increasing. The fact that the country lies between 60° and 70° latitude North should be noted. Above the Arctic Circle in northern Finland where the cohort babies were born, the researchers tell us there are only 2 hours of daylight every day in December. (They do get 23 hours of daylight each day in June—but UV-B rays are scarce there even in summer.)

Hence, the Finnish are forced to depend on vitamin D-rich foods and supplements from cradle to grave. That may explain the astonishing fact that for a period of time (the authors didn't say how long) until 1964 the recommended daily allowance for infants in Finland was 4000-5000 IU of vitamin D per day. I can't imagine this could be safe for babies over an extended period, but when I contacted Dr. Elina Hypponen, she replied: "To my knowledge, the reduction in the recommended dose was not done so much as a response to observed reactions, but as a safety measure due to possible toxicity. There are, however, some subjects that are hypersensitive to vitamin D."

In 1964 the RDA for infants in Finland was reduced to 2000 IU; in 1975 to 1000 IU; and finally in 1992 to 400 IU. The authors say this latest recommendation for infants was based "on the amount of vitamin D in a teaspoon of cod-liver oil, which had long been judged safe and effective in the prevention of rickets." But they're worried about the "constant increase" in type 1 diabetes and are talking about raising the infant vitamin D allowance—cautiously, of course.

Testing, Treating, Testing!

Scientists worldwide pretty well agree on the usefulness of the 25-hydroxy-vitamin D test, which is of fairly recent origin, for gauging an individual's vitamin D status. A giant step in preventive health will happen when this test is done routinely in medical exams, starting with babies.

Krispin Sullivan,* who's completing the definitive consumer's book on the vitamin, tells me a test that uses saliva instead of blood may be available soon. This year, my second 25(OH)D blood test, three months after my first, showed much
improved levels. By the end of those same three months, long-lost full mobility had come back to my bum knee...Blissful!

Depriving the Brain

Australian psychiatrist John McGrath is making waves with a new theory on why in Europe and North America there’s a mid-March peak in births of babies who later grow up and become schizophrenic. He says lack of UV light during key months of such pregnancies may offer a clue. (New Scientist, July 7, 2001.)

Somehow, vitamin D must be important to the normal developing brain, since there are a bunch of D-receptors in it. In new experiments where pregnant rats were deprived of UV light, or of vitamin D in their food, the rat pups had distinct brain anomalies, some surprisingly like those found in brains of some schizophrenic persons.

Backup for McGrath’s theory: Afro-Caribbean immigrants to England, as well as Surinamese immigrants to Holland, have 3-4 times more children who become schizophrenic than the rest of the populations there. Their darker skins are strong barriers to the skimpy UVB light in these northern latitudes, thus a pregnant woman’s vitamin D status and that of her fetus could be compromised, unless, of course, food and supplement sources were well-supplied.

Provident, isn’t it, that some of the best food sources of D (salmon, sardines, herring, mackerel, shrimp, eel) also contribute my beloved (good-for-brains!) omega-3s.

I’ve a growing stack of studies/reports on ailments connected to low D that are a lot more common than schizophrenia or juvenile diabetes. Along with obvious ones like osteoporosis and arthritis, they include obesity and insulin resistance --plus grim stuff like cancers of breast, colon, and prostate.

Researchers are finding low 25(OH)D levels in patients with colon or prostate cancer. Also, there are higher rates of colon, prostate, and breast cancers in parts of the globe that get little UVB light, compared to areas where UVB rays are generous. Studies show vitamin D encourages healthy “differentiation” of human cells, which makes them resistant to cancer.

* Educator and clinical nutritionist Krispin Sullivan CN will have answers to many of our questions in her book, based on worldwide medical studies and experiences with clients in her Bay area consulting practice. It should be out in spring 2002. Her tel: 415-488-9636. Web site: http://www.sunlightandvitamin.com

Pickles/Brian Crane

I GUESS THE BEST THING
ABOUT DOGS IS THEIR
LOYALTY. A DOG WILL
STICK BY YOU TO THE
BITTER END.

WHEN THE GREEK HERO
ODYSSEUS ARRIVED
HOME (REUSING AS A
BEGGAR) AFTER BEING
SOME NINETEEN YEARS.

...THE ONLY ONE WHO
RECOGNIZED HIM WAS
VIVI GOOD OLD RODOS,
WHO SMELLED HIM, THEN WAGGED
HIS TAIL AND DIED!

He probably shouldn’t have sniffed him.

Sorting Out the Rays

If UVB sunlight is so beneficial, why are people getting more skin cancers? It turns out the specific rays of sunlight now believed to be responsible for skin cancers are those reaching the earth from sunup to sunrise, winter and summer, even at higher latitudes. These are ultraviolet A -- the so-called “tanning rays” that make up 90 to 95% of ultraviolet energy in the solar spectrum. (By comparison, UVB rays are relatively scarce.) UV-A’s less energetic but longer than UVB rays--can penetrate glass --80%, while only 5% of UVB rays can.

In the 1970s sunscreen lotions began to be widely used, but until a few years ago they blocked only UVB (the “burning rays”). In a 1993 paper, CF Garland et al. offered this theory about the steep rise of melanoma since the mid-1970s:

--“Because sunscreens prevent erythema and sunburn, and inhibit accommodation of the skin to sunlight, their use may permit excessive exposure of the skin to portions of the solar spectrum other than UVB. If melanoma and basal cell carcinoma are initiated or promoted by solar radiation other than UVB, as laboratory data suggest (my emphasis), CF, then UVB sunscreens might not be effective in preventing these cancers, and sunscreen use might increase the risk of their occurrence.” [Annals of Epidemiology, 1993 Jan. Vol. 3(1), pp103-110]

Exploring better ways to safeguard skin is a hot topic now. Some scientists push for increased oral intakes of antioxidants substances such as beta-carotene, vitamin E, and ascorbic acid. (I say this can’t hurt!) Others are using “new chemopreventive agents” topically (SP Stratton et al., Eur J of Cancer, 2000 June; 36(10)). Among substances being studied by this and other groups are ascorbic acid, silymarin, vitamin E, and green tea extract. (Wow! Maybe I’ll go stir up a trial batch in my kitchen.)

A Modern-Day Vulnerability

Here’s a different take on why we’ve been getting more skin cancers these past 30 years. (Thanks, Krispin Sullivan and researcher friends Paul and Barbara Stitt, for alerting me!)

Omega-6 POLY-GLUT may be doing us in, in more ways than I realized or wrote about in FLS116/117.

I used to push for low saturated and high polyunsaturated fat intakes, like most nutritionists. This was the jolly road to heart health, remember? The ultimate awakening came via Dr. Harumi Okuyama and colleagues’ great 1997 review of the stunning inroads on Japanese health from what seemed, to my western eyes, just a modest increase in omega-6 (w6) over omega-3 (w3) fatty acid intake.

Now I’ve learned too much consumption specifically of w6 polyunsaturates blocks special vitamin D-binding proteins, as well as vitamin A-binding proteins, which normally carry these nutrients through the blood to where they’re needed. A loss of A and D in the skin makes its genetic DNA material more vulnerable to damage from ultraviolet radiation, now believed to be mainly from UVB. It’s this damage that initiates skin cancers, including melanoma.

Happily, w3s and saturated fats (natural unhydrogenated ones) don’t block the vitamin-carrying proteins. But--here’s the rub--most of us are overloaded with w6s today, even if we conscientiously avoid w6 oils. When agribiz took over family farming about 80 years ago, beef cattle, hogs, and poultry began getting grains as the main feed. Cattle used to eat what’s natural to them: mostly grass--fresh or dried (hay). Along with grains, pigs ate mostly kitchen peelings and scraps, while the chickens could also scratch for bugs and favorite weeds like (high-w3) purslane. Grains induce rapid growth. That’s one of the prime functions of the high w6 fatty acid content of grains.
For agribiz, rapid growth in meats and poultry is a money-maker. For the consumer, grain-fed animals have become a hidden source of *w6 overload* because of abnormally high w6 levels in their tissues. Even as we re-balance our w6 and w3 intake to a safe ratio (say, 1-to-1 as in hunter-gatherer days, or 3-to-1 as in Japan before they began slurping w6 oils), the ‘hidden’ w6 may screw things up. Krispin says even farmed fish have abnormally high w6 levels from *corn* in their feed!

Here's why she's concerned: “The w6 promote growth. The w3 promote repair. Too much growth without adequate repair is a recipe for trouble.” We've known since the 1980s that too high w6 levels can cause inflammation and internal cancers. Now we learn they make us vulnerable to skin cancers, too.

**Armed with this knowledge, I plan to:**
- Avoid w6 oils. (I use coconut fat, butter, or olive oil.)
- Not use w6 oils on my skin.
- Buy wild fish and shellfish, until hi-w6-feed in fish farms stops.
- Buy only free-range chickens and their eggs.
- Buy lamb. (Lamb is generally grazed in pasture with the ewes until marketed at -6 months.)
- Use nuts, seeds, grains in modest amounts only.
- Increase my intake of high w3 fish oil.
- Continue daily tbsp of flaxmeal.
- Eat plenty of chocolate. (I'm not perfect.)

Here's one more: I'm taking one 10 milligram vitamin K capsule every other day - always with a meal that has some fat for proper absorption. Vitamin K is needed for normal blood clotting, but also enables special proteins to steer calcium into the right tissues. For example, without vitamin K, one of these better-known proteins, osteocalcin, can't direct calcium into bones, teeth, etc. So, even if you have ample vitamin D, without enough vitamin K, calcium can evade your bones and invade your soft tissues and arteries instead.

Think of D and K as vitamin-partners in bone-building and artery-reaming!

Kindly bacteria are supposed to make vitamin K for us in our gut, and we also consume it from green leafy vegetables, including sea vegetables (dulse, kelp, hijiki, etc.). However, high rates of osteoporosis in pre- and post-menopausal women hint at suboptimal intakes not just of D, but of vitamin K.

The DRI for vitamin K is 90 micrograms (.09 milligrams), so my daily 5 milligrams are a hefty dose, but very safe. Toxicity isn't seen even with extended daily use of 45 mg. (The only caution is for people who are on anti-platelet drugs.)

UVB rays, and vitamin D itself, may actually protect skin from cancer, just as they do other tissues and organs. The goal is to get enough UVB to make the vitamin in your skin without *burning*. UVB intensity varies widely, depending on latitude and altitude. (For every 1000-foot rise in elevation, UVB intensity increases about 10 percent.) If you live in latitudes above 40° N, you'll only get enough UVB for reasonable vitamin synthesis mid-days in summer. Check your atlas: all of Canada and most of Europe are well above 40° latitude. Also, UVB can't penetrate fog, clouds, or smog and other air pollutants.

How will you know if you're making enough D? If you don't spend time in UVB-rich light, or if you burn too easily when you do, you'll have to get your D from food and supplements. But how much?

You and your health provider(s) can use the 25(OH)D test as a guide to:
- Determine your base level.
- Supplement with fish-oil-based vitamin D3.
- Test again 3 months later.
- Adjust supplement dosage accordingly.

Voila! - no toxicity, no deficiency!

**SICKLE CELL - GOOD TIDINGS**

I n 1993 I first began reporting about an effective nutritional therapy for the inherited blood disorder, sickle cell anemia (FL 71). The waves of interest and optimism I dreamed it would generate in the U.S. medical community didn't happen, but now a benign *tsunami* effect seems to be taking place in the *Nigerian* one. Who knows, with luck, it may even ripple back to the U.S.!

The wave-makers are Oklahoma biochemist Oji Agbai, ND, PhD, and his wife Chinere, a medical technologist and Christian minister. In case after case since the 1980s, the gentle measures they espouse have helped to alleviate the need for invasive, expensive ones. The disease, beginning in childhood, causes great suffering and often shortens lives in spite of conscientious medical care. That's the
situation for homozygous 'sickers' (people with sickle-cell genes from both parents) in the U.S.

But when Dr. Agbai was growing up in a rural community in West Africa, he tells me "even though the Ibo language had plenty of words for arthritis, rheumatism, and other ailments, it had no words for sickle cell disease," an indication of its rarity. (The characteristic severe anemia, infections, and pain in young sufferers could not have been overlooked.) He remembers that people there still ate traditional foods that had protective elements—the same ones forming the basis for the nutritional remedy he developed in the '80s and patented in 1990, "Dioscovite."

He was a chemistry professor at the U. of Arkansas in the late 1980s when he began speaking "at health conferences, community groups, churches, etc.… 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." [FLS 105-106, 1999]

(A National Institutes of Health study published in 1974 found thiocyanate to be the best anti-sickler of all likely ones tested in vitro—its effects were "profound." They never followed up.)

I described in 105-106 an unprecedented event at a big national sickle cell meeting in '99. One person after another rose uninvited from the audience, took the mike, and told how they or their children finally had gotten well, by eating daily certain recommended foods, and using Dioscovite, to promote high blood levels of thiocyanate. (Not all the medical officers were pleased.)

Agbai’s concepts consistently have gotten short shrift in U.S. sickle cell research circles where high-tech stuff like gene manipulation and bone marrow transplants get the limelight. Fate seems to have played a hand in the encouraging new developments. Unexpectedly, through a mutual friend at a Christian university in Oklahoma, the Agbai’s met the governor of Ebonyi State in Nigeria, who was in the U.S. on a 10-day economic tour. Mrs. Agbai tells me their discussions during dinner were long and lively! The governor not only got fired up by what to him was a totally new, practical approach to sickle cell, but was excited that someone from his own country had developed the program and written the definitive book on it.*

So the governor invited Dr. Agbai to Nigeria for a lecture tour, and went home with a supply of Dioscovite for one of his commissioners whose 5-year-old child has sickle cell. For 30 days in July and August, Agbai spoke in his homeland to political groups, university and medical people, and journalists. He tells me he was stunned that specialists in sickle cell knew nothing about the anti-sickling properties of their native foods. Their training was in western medicine; his concepts were as foreign to them as they are to doctors here!

At a federal medical center the doctors got very excited when he showed them transparencies of scanning electron micrographs from his book. High magnification (4000x) allowed them to observe that when blood from sicklers is added to a saline ‘control’ solution, strong sickling events produce visibly distorted ‘sickle-shaped’ red blood cells. But when an optimal (30 millimolar) concentration of potassium thiocyanate is added to another saline solution of sickler blood, no sickling whatsoever takes place, each rbc maintaining its normal plump, discoid shape. Significantly, a weaker (10mM) thiocyanate solution did not protect rbc’s from sickling

**Down to Basics**

Agbai had to start from scratch in his talks, explaining why thiocyanate, a naturally occurring molecule in human plasma, inhibits sickling of hemoglobin not just in test tubes, but in the blood of living beings! Traditionally, Nigerian folk wisdom taught people which specific plant foods would let them bypass severe anemia and other illnesses. We know now that the high content of cyanogenic glycosides and/or thiocyanate of such foods (African yam, sorghum, millet, manioc, etc.) enables people who consume them regularly to maintain optimal plasma levels of thiocyanate. This allows children and adults with homozygous sickling genes to avoid serious anemia and to live normal lives. (They also are resistant to severe malaria -- but that’s another story)**

The future for licensing or registering Dioscovite in Nigeria looks pretty good at this writing. I guess the governor’s sponsorship of the lecture tour helped, along with enthusiastic responses Dr. Agbai got from fellow-scientists. Some expressed interest in doing their own Dioscovite studies, including the director of a federal medical outfit who, years ago, had been Agbai’s fellow-student! Six million Nigerians may be homozygous for sickle cell, Dr. Agbai told me.** I hope they’re rediscovering the foods that kept their forebears alive ‘n kicking—Dioscovite may not be able to do it alone!

*For information about Dioscovite and Dr. Agbai’s anti-sickling nutrition handbook, Sickle Cell Anemia: A Solution At Last, contact Natural Health Research Institute, 6390 E. 31st Street, Suite E, Tulsa, Oklahoma 74135. Tel. & FAX: 918-627-7997.

**While this story is about Africans, sickle cell anemia occurs in most races and ethnic groups including Greeks, Sicilians, Arabs, Turks, and Asian Indians. Millions more have just a single sickling gene from one parent (‘sickle cell trait’) and don’t have typical anemia.

Illustrations are 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, & impecuniously by Clara Felix since 1981, supported by subscription. Descriptive list of available back issues, plus sample issue, $1. Subscriptions USA & Canada: $12 for six issues (approx. 1 year); $22 for 12 issues (approx. 2 years). U.S. funds only.

2001 No. 118
All rights reserved.