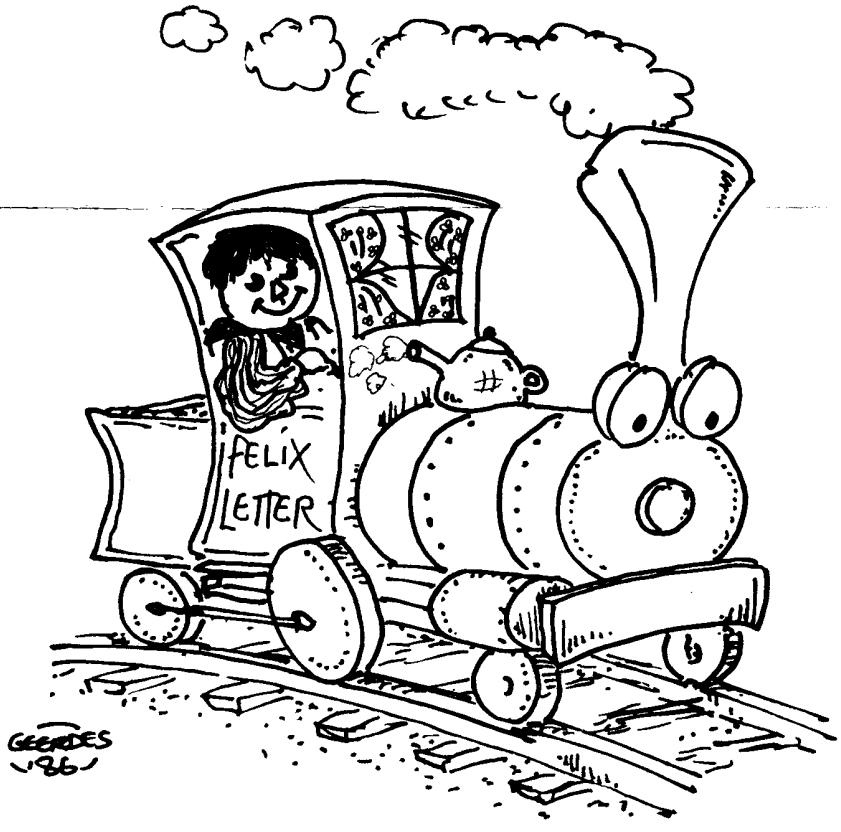
the FELIX Letter
A COMMENTARY ON NUTRITION
NOS. 33 & 34 1987

EASY WAYS TO FIGHT CANCER

I'm surprised and pleased, because conservative physicians by the dozens, who wouldn't have been caught dead championing the therapeutic virtues of any nutrient besides "polyunsaturated vegetable oils," are climbing on the "omega-3 fish-oil" bandwagon. Never have I seen the establishment take a nutrient to its bosom so rapidly! Despite solemn warnings from some quarters against dosing with omega-3-rich fish oils until more hard data accumulate, the reports in the medical literature are lit up with a rare optimism, no matter how cautiously expressed!

The optimism extends far beyond the well-accepted sterling effects on heart disease. A recent symposium in New York sponsored by Harvard Medical School described some of the cardiovascular benefits produced by EPA and DHA, the main omega-3 fats in fish oils: lowered blood pressure, less clumping of blood platelets and fewer blood clots, inhibition of inflammatory processes in arteries, lowering of plasma cholesterol, and so on. But the researchers said these same events *not only slow down atherosclerosis and protect the heart, but may also provide the people who take the fish oils a fundamental protection against generalized inflammatory reactions, as well as against immune disorders.* They suggest that sufferers from migraine headaches or rheumatoid arthritis, for example, may benefit from the omega-3 fats in fish oils.

The pleasantest news of all, reported by Rashida Karmali, Ph.D. in the January 1987 *Am.J. of Clinical Nutrition*, is that *diets high in omega-3 fatty acids decrease the incidence and growth of a variety of tumors in laboratory animals, particularly breast and prostate cancers.* Dr. Karmali, a small, comely woman with expressive dark eyes, gave a further update of her work at a February conference which I attended at U.C. Berkeley on "Oxygen Radicals & Antioxidants in Cancer & Aging." Her group at Rutgers U. and Memorial Sloan-Kettering Cancer Center in N.Y has a new study just "off the rack" (results still unpublished), showing that fish oils in the diet protected the animals from cancers induced by powerful carcinogens. The breast cancers they did get were smaller than the ones that appeared in the animals that didn't get fish oils. Even more important, *the fish oils were exceptionally effective in helping to keep the cancer from spreading to the lungs, i.e., metastasize.* Since death in breast cancer comes mainly from the spread of cancer to vital organs, this is welcome news.



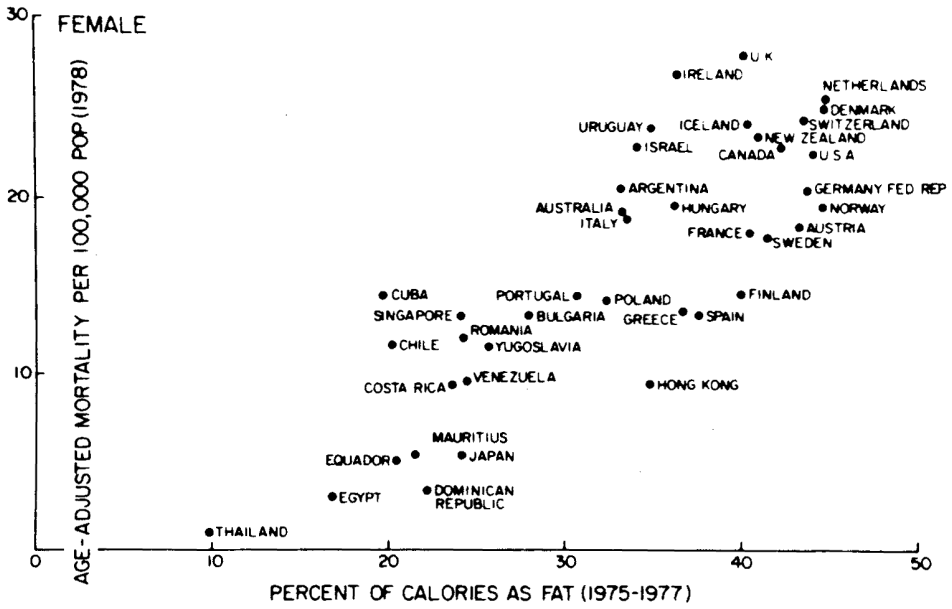
Good Tidings!

We know that animal studies can be useful guideposts, but what about direct application to breast cancer in women? Here, Dr. Karmali gave us still more reason to celebrate. A new prevention trial currently is being conducted by her group at Memorial Sloan-Kettering. She described the problems in trying to discern in a comparatively short time whether fish oils will inhibit the cancer in humans, whose life span is so much longer than that of mice. What her group needed most was a "biomarker"—an easy-to-measure metabolite in the blood that can be correlated with breast cancer. This biomarker was found just recently. When the female hormone, estrogen, is made and used by the body, it metabolizes into inert, harmless products the body easily eliminates. However, estrogen can also be transformed into active molecules that can't be gotten out of the system so readily. *Highly exaggerated levels of this active estrogen, 16-hydroxyestrone, have recently been identified in women with breast cancer,* while cancer-free women produce only small amounts.

It so happens, Dr. Karmali added, that certain strains of laboratory mice, which have a very high incidence of spontaneous mammary cancers, also have the same magnified levels of 16-hydroxyestrone! So the studies with this new biomarker are being conducted on a human *and* an animal level. She said the early results are quite encouraging. In both women and mice at high risk for cancer, fish oils added to the diet produce a very big drop in 16-hydroxyestrone levels! They appear to be protecting both species against breast cancer.

High Fats, High Cancer

So far, the strongest link between diet and cancer is the very high rate of breast, prostate, and colon cancers in countries *where the consumption of fats is very high* (e.g., U.K., Netherlands, Denmark, U.S.); coupled with very low cancer rates *where people consume very little fat* (e.g., Philippines, Thailand, Japan). For a time, hard fats as in meat and dairy were assumed to be the culprits, while vegetable oils were assumed to be the good guys. No such luck. As the information from animal studies mounts, polyunsaturated fatty acids ("PUFAs") are seen to cause cancers to INCREASE! As the amount of PUFAs goes up so do the cancers, especially breast and prostate. *The oils implicated belong specifically to the omega-6 family of PUFAs.* That's why the new reports on the *anti*-cancer properties of the *omega-3* oils in fish have the experts spinning like tops! They had been ignoring this "little known" PUFA group for 25 years.



Percent of caloric intake as fat plotted against age-adjusted mortality from breast cancer

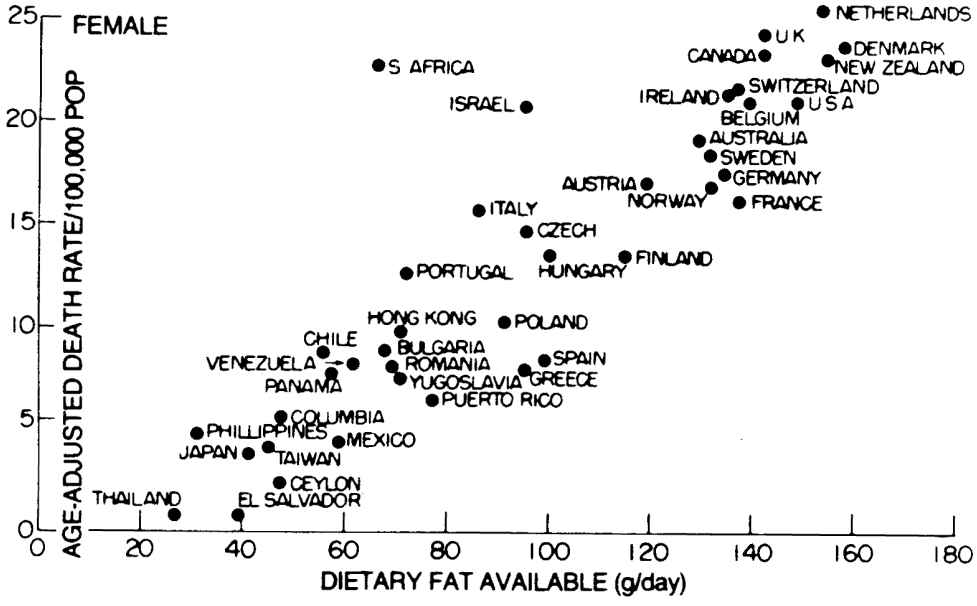
The Calorie Contribution

Researchers tried another tack. Maybe it was *calories*, not just fat. As a group, obese people (and rats) eat more and also have more cancer than the leaner folks (and rats). They *did* find in some experiments with rats and mice that cutting way, way down on the chow also reduced the number of cancers. (Maybe the saying should go, "Feed a cold, starve a cancer.") The same tactic, by the way, is the only proven strategy for extending the life span of mice, as reported by Roy Walford, M.D. at the Berkeley meeting.

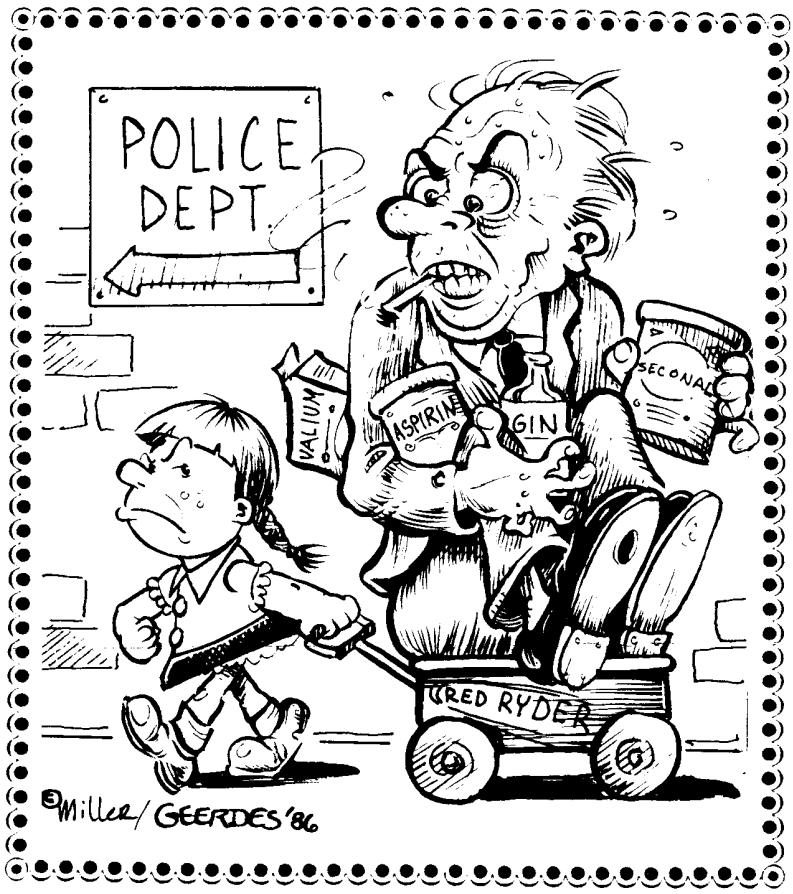
Let's face it, though. Even to stave off cancer and/or old age, how many of us would willingly go hungry for the rest of our lives! I should add that the two types of successful experiments with mice—to decrease cancer and to increase longevity—require that all essential nutrients be provided at reasonable levels; only calories are cut. To get really good effects, experimenters in one study had to reduce calories for their rats by 40%. Sure, tumor incidence was way down, but I'll bet that was one grumpy little bunch of animals! While high-caloric intake as a factor in cancer is still being investigated, the connection isn't as strong as the high PUFA one. Besides, I don't have to be a sage to predict that permanent starvation as a way to prevent or treat cancer is bound to run into some mighty big roadblocks!

On the other hand, the prospect of knocking out cancer by juggling the fats in our diet has a lot going for it. We can make our own saturated and monounsaturated fats. [I'm referring to the stuff that does such a great job rounding out our corners.] However, we can't make the basic omega-6 or omega-3 fatty acids. We're obliged to get them from foods, because they have vitaminlike properties, i.e., they are "essential" nutrients.

If they both are essential, *why do omega-6 PUFAs promote cancers, and omega-3 PUFAs inhibit them?* The answer appears to lie in a group of substances our body makes from omega-6 PUFAs. After we consume linoleic acid, the main omega-6 in food, it can be transformed into *arachidonic acid* by special enzymes in our cells. (Arachidonic also comes 'ready-made' mainly from meats, eggs, and poultry.) Arachidonic acid can behave either like a friend or foe, depending on circumstances. It's the principal source of *omega-6 prostaglandins—the molecules that act as hormone-like coordinators in our cells.* Ordinarily, a certain amount of prostaglandins ("PG") are made, most likely in response to signals calling for normal defense measures. The PG do their job, then are quickly put out of action. No problems!



Total dietary fat available plotted against age-adjusted mortality from breast cancer in various countries.



A Plethora of Peroxides

Trouble starts only when too much arachidonic is available and too many PG are made. If they're not stopped, *PG from arachidonic amplify the natural peroxide levels in our tissues.* Neutrophils and other white blood cells migrate to the area as a defense maneuver. They release peroxide which, in turn, signals the cell to make more PG—which then attract more neutrophils, and so on. *A deluge of peroxides sets the stage for inflammatory chain reactions by oxygen radicals.* If these over-defensive measures go on, depending on which tissues are targeted some of the effects can be:

- inflammatory effects in joints and muscles (e.g., arthritis)
- spasms in the digestive tract (e.g., irritable bowel syndrome)
- spasms in bronchial tubes (i.e., asthma)
- spasms in blood vessels beneath the scalp (i.e., migraine headache)
- spasms in uterine muscles (i.e., menstrual cramps)
- spasms in arteries
- clumping of blood platelets with clot formation in arteries. (In an artery already narrowed by plaque, a combination of clot and spasm can mean big trouble!)
- tissue damage eventually.

How Omega-3's Fight Cancer

The omega-3 PUFAs halt these events by putting a stop to runaway PG production. They do this by commandeering the enzymes in the cell that are needed for PG output.

How does this relate to cancer? Referring to the high peroxide levels created by PG from arachidonic, William E. Lands, Ph.D. told fellow-researchers at a conference on cancer (Jan. *Am.J.Clin.Nutr.*):

> ***Whether the peroxides then work by damaging DNA, or whether they cause more inflammatory responses to facilitate metastatic*** [cancer-infiltrating] ***processes is uncertain, but both possibilities point in the same direction. A group like this one needs to explore the roles of prostaglandins... in metastatic processes, the factors that lead to tumor cell detachment, penetration into capillaries, circulation through the blood stream, invasion of... distant tissues, and formation of secondary tumors which then are fatal. The studies presented have clearly shown a role for prostaglandins in facilitating primary tumor development.***

Another connection to cancer is *thromboxane*, one of the arachidonic PG that is known to be critical to the spread of cancer. When thromboxane levels drop, metastasis slows down. *The omega-3 PUFAs fight cancer by keeping the amounts of arachidonic PG, including thromboxane, down to reasonable levels.* No cascades of peroxides and peroxide-induced free radicals, no DNA damage, no inflammation, spasm, pain, etc.!

Good vs. Bad Prostaglandins

Moreover, *omega-3's make their own PG that work to neutralize the actions of those from arachidonic. For example, they dilate arteries, relax involuntary muscles, and slow down inflammatory events.* They don't amplify natural peroxide levels, either. Consequently, they don't encourage a barrage of oxidative free radicals. Free radicals are troublemakers; cancers can arise from the damage they do to DNA, the genetic code repository. (The February symposium at U.C. Berkeley brought together leading researchers just to share their findings in this field.)

Some medical workers are experimenting with drugs that fight cancer by slowing down arachidonic PG production. Side effects, in part, may be due to the fact that the drugs also inhibit output of the *good* kinds of PG. Drs. Karmali and Lands, among a growing group of scientists, are enthusiastic about a similar use for fish oil, *because it inhibits arachidonic PG with great safety and no side effects. Also, it doesn't destroy the good PG.*

Vegetarians, among others, will be happy to learn that linseed oil, a traditional omega-3 oil rich in alpha-linolenic acid (ALA), also pushes arachidonic PG down to normal, trouble-free levels. Donald O. Rudin, M.D., researched the concept a few years ago of using linseed oil (food-grade, not the paint store variety) to create a therapeutic omega-3/omega-6 ratio in the body. In his two-year 44-patient trial, he observed a number of spectacular reversals of chronic ailments, even in some mental disorders. His book which I coauthored should be out in early summer: THE OMEGA-3 PHENOMENON: *The Nutrition Breakthrough of the 80's*,published by Rawson Associates, NY.

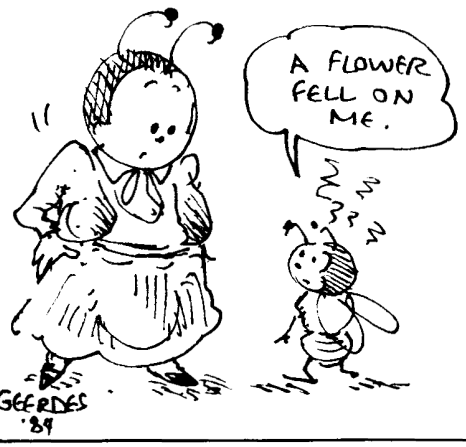
Practical Ways to Prevent the Big C

What does all this new information on omega-3 PUFAs and cancer boil down to, in practical terms for us as individuals?

For one thing, despite the enthusiastic reception in some medical quarters, somehow I can't picture the establishment, as a whole, setting aside its high-tech, high-cost regalia just to opt for the use of humble fish oils in treating society's ills! I suspect the public will be left largely to its own devices for a long, long time, on the omega-3-oil front.

A leading British researcher in the field, David Horrobin, M.D., offers some ideas for a dietary strategy that may modify the need for anti-inflammatory drugs, and possibly may offer protection against cancer. *He suggests that much of the arachidonic that turns into unruly PG may come from the 'ready-made' arachidonic in foods.* (Meat, eggs, and poultry provide the largest amounts.) By cutting down the amounts, especially of meat, that we consume, he believes we may nip a potential problem in the bud.

Another part of the strategy involves the intake of natural substances containing precursors to a group of omega-6 PUFAs, known as DGLA, *which produces 'good' PG. Like the PG from omega-3, they tend to oppose the actions of arachidonic PG, to calm down inflamed tissues, relieve pain, dilate arteries and bronchial tubes, etc.* Ingesting the precursor seems to directly increase the good PG. For example, British studies show that oil of evening primrose seeds causes more benign PG to be made, with benefits noted in ailments ranging from painful breasts to childhood hyperactivity. *The Lancet* (May 10, 1986, p. 1098) describes a study at Glasgow Univ. Medical School in which evening primrose oil reversed nerve damage in diabetic patients.



(The dried powder of a blue-green algae, Spirulina, is being studied in depth by, for one, Japanese medical researchers, because of its good effects in cancer and other ailments. Like evening primrose oil, it contains a precursor to DGLA. Healthfood stores sell Spirulina tablets or powder as a concentrated source of protein, magnesium, and other nutrients.)

A third strategy recommended by Dr. Horrobin is an increased intake of omega-3 EPA and DHA, the main PUFA in fish and fish oils. By creating a healthier omega-3/omega-6 balance in our tissues, this important dietary tactic will lull the troublesome PG and allow more of the good kind to be made.

Is 20% A Solution?

Currently, there is an avant-garde effort by a number of health workers to cut the intake of all fat to 20% or less of total calories. (See "Breast Cancer Prevention—A Controversial New Diet Program," by Susan Rennie, Ph.D. in the April 1987 *Ms.* magazine.) Since in the U.S. calories from fat are closer to 40%, dropping to 20% would require radical modifications in the eating habits of most individuals. I do think it's got some merit, but I have reservations. Aside from the practical problems in getting accustomed to eating this way, I suspect it may create a climate of fear, where people will think: "Oh-oh! I've overshot my fat quota for the day . . . I may be starting a cancer this very minute!" Fear itself can do a lot of harm. The "20% program" is, after all, only one of a number of theories on preventing cancer.

My main objection is that it puts the "em-PHA'-sis on the wrong syl-LAB'-le." Instead of a rigid obsession with counting fat calories, the diet would serve its purpose better if it were to focus on setting up a ratio of omega-3 to omega-6 PUFAs that clamps down on cancer-promoting PG.

The Wisdom of Ancestors

Rather than doggedly adding up grams of fat, why not think in terms of broader baselines—the kind that worked for our ancestors, who didn't have much cancer or heart disease? The big stumbling blocks to achieving ripe old age were the infectious diseases that decimated the young; once a person got past that slippery period, their life span was only a few years shorter than ours. People didn't eat lots of oil because before the age of technology, it was hard to extract from plant seeds. Beef was a luxury. People lived near lakes, seas, and rivers because these arteries were more important to transportation than roads, so, naturally, fish and shellfish high in EPA and DHA were basic staples. The backbone of the diet were grains, beans, potatoes, vegetables and fruits. They were low in fat and high in fiber, which helped to process the fat safely in the gut, just as the PUFAs regulated it in the blood.

Grouse, hare, and other game were commonly eaten; their fat had a lot of omega-3 and omega-6 PUFA. The fat rendered from the local pigs and poultry had a good balance of both groups of PUFAs, too. Only a few wealthy folks could afford to use it lavishly. In general, fats and oils were used frugally, compared with today.

- *So, absolutely, let's keep the calories from fats and oils and fatty foods down. It makes good sense, in the light of everything we know. But let't not do it at the expense of omega-3 fats!*

- *It also may be prudent to cut down on the ready-made source of arachidonic—i.e., meat.* Except for the rich, our cancer-free forefathers had to use it mainly as a condiment to jazz up their grains and beans.

- *And while we're taking a leaf from their book, why not make fish and shellfish staples in our diet?* They not only have far less pre-formed arachidonic than meat, they have lots of EPA and DHA to keep it in check!

The following one-day's menu is reasonably high in protective nutrients, as well as fiber. For extra safety benefits, I take and recommend vitamin-mineral supplements (see Issues 9, 10, 10, 24 and 28). Based on Dr. Rudin's program, I take a "fiber cocktail" before most meals, consisting of a spoon or two mixed of powdered psyllium seed (Metamucil or similar product) and bran, stirred with a few spoons of yogurt, and followed by a glass of water.

The day's menu is based on 2000 calories. Twenty percent would come to 400 fat calories. A gram of fat has 9 calories. By dividing 9 into 400, we get a fat allowance of 44 grams per day. That's probably even less fat than our ancestors in pre-industrial times ate. My day's menu provides more than 44 grams (or 20%), but it offers protective amounts of omega-3 and omega-6 PUFAs. For those who want to stay at 20%, however, I've provided one optional item (see *); by skipping it, you can achieve that goal and still get your good ratio of PUFAs!

CALORIES: 2000 per day

TOTAL FAT: 54 grams per day
Percent of day's calories as fat: 24%

Calculations: 54g x 9 cal/g = 486 calories

$$\frac{486}{2000} \text{ X } 100 = 24\%$$

LINOLEIC, an omega-6 PUFA: 14 grams per day
Percent of day's calories: 6%

OMEGA-3 (ALA, EPA & DHA): 9.7 grams per day
Percent of day's calories: 4%

Diet for a Day: Lo-Fat, Hi Fiber, Hi-Nutrient, Hi-Omega-3's

FOOD ITEM (GRAMS) 1 OZ. = 28 g	CALORIES	TOTAL FAT (GRAMS)	ESSENTIAL PUFA: LINOLEIC ACID OMEGA-6 (GRAMS)	ESSENTIAL PUFA: OMEGA-3 ALA (GRAMS)	OMEGA-3 EPA (GRAMS)	OMEGA-3 DHA (GRAMS)
Breakfast: 2 corn tortillas, 60 g	130	2	1	–	–	–
• 1 cup mashed beans, 180 g	225	2.8	0.6	1.1	–	–
* • 1 oz. grated cheese, 28 g	114	9	0.6	0.1	–	–
• Decaf, postum, etc. with						
1/8 cup skim milk, 30 g	10	tr.	–	–	–	–
• Fruit—e.g. 1 cup blackberries	80	0.3	–	–	–	–
Lunch: Salmon sandwich:						
• 2 slices whole grain bread, 56 g	140	1	0.6	.04	–	–
• Canned salmon—3 oz., 85 g *(Be sure to eat the bones, too!)*	120	5.7	0.1	0.3	0.5	0.85
• Tomato, sliced, 120 g	25	–	–	–	–	–
• Alfalfa sprouts, pickle relish	20	–	–	–	–	–
• 8 oz. low-fat yogurt, 227 g	145	4	0.1	–	–	–
• Walnuts—2 TBSP, 20 g	120	12.6	7	1.4	–	–
Dinner: Rice or Barley Casserole:						
• 1 cup cooked rice or barley, 195 g	230	1 g	0.5	.06	–	–
• 3½ oz. Tofu (from soybeans), 100 g	72	4.2	2	0.4	–	–
• ½ cup cooked frozen peas, 80 g	62	0.2	.02	tr.	–	–
• 1 cup cooked string beans, 125 g	45	.18	.06	.08	–	–
• ½ cup sliced mushrooms, 80 g	20	0.4	.05	0.2	–	–
• 2 cups spinach salad, 100 g	20	0.4	.05	0.25	–	–
• 2 teaspoons linseed oil, 10 g	90	10	1.8	4.5	–	–
(as salad dressing w/ garlic, herbs & vinegar)	–	–	–	–	–	–
Calorie subtotal	**1668**					
•Nonfat snacks, e.g. fruit,						
dessert, beverages, totaling	332					
TOTAL	**2000**	**54 g**	**14 g**	**8.4 g**	**0.5 g**	**0.85 g**

If you're determined to stay at 20% fat calories, by giving up the * item (1 oz. grated cheese, 114 calories) you total fat intake drops by 9 grams, to a total of 45 grams. That calculates out nicely to 20%. (You can fill in the available 114 calories with rice, vegetables, fruit, etc.)

For individuals taking concentrated fish oils, six one-gram capsules a day will provide approximately:

54 calories (9 per cap)
6 grams total fat (1g/cap)
36 milligrams cholesterol (6 mg/cap)
1 gram EPA (0.18g/cap)
0.7 gram DHA (0.12g/cap)

The 54 calories from fat will increase total fat calories in the above diet to 27%—still well under the 30% most health workers consider to be safe.

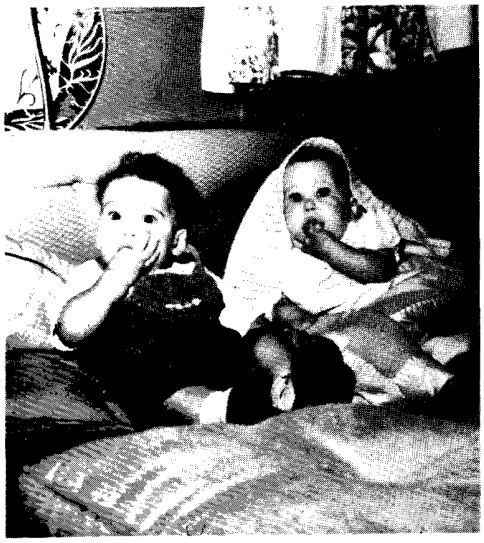
Balancing the PUFAs

Dr. Rudin, using studies on known omega-3 deficiencies, and comparing the deficient levels with those in healthy populations, estimates *the standard omega-3 intake compatible with good health to be at least 2%of daily calories.*

Omega-6 intake probably is safe at 4 to 10% of daily calories, as long as ample omega-3 are also eaten, but much remains to be determined about how much omega-6 PUFA is okay.

Dr. Rashida Karmali said: "An optimal ratio of omega-3 to omega-6 exists at which the protective effects against mammary tumor development are observed. In our [animal] studies we have found this to be approximately 1." (*J.Am.Clin.Nutr.*, Jan.)

This is a rather stunning observation. While a 1-to-1 ratio may not be applicable to humans, it's something to think about! Dr. Rudin estimates that modern diets provide *about 25 times more omega-6 than omega-3 PUFA,* with omega-6 at 10% of calories, and omega-3 at 0.4%. He says that this terribly inadequate omega-3 intake—compounded by other dietary wrongs—is responsible for much of today's illness, physical and mental. Clearly, some drastic changes are in order. Most health revolutions begin with individual experimentation, so I guess it's up to each of us!



A NOTE OF CAUTION RE WEIGHT LOSS: As I see it, the danger for those who strive for low-calorie weight-loss diets while hewing to a 20% fat calorie level, is that omega-3 sources will be squeezed out of the picture. Dr. Rudin says they are especially needed *to normalize appetite-controlling and body-heating physiology.* In the weight-loss chapters of THE OMEGA-3 PHENOMENON, menus and recipes are designed to provide generous omega-3's on a 1200-calorie, low-fat regimen. Omega-3's happen to be the *cold-climate PUFA.* They play a big role in keeping the body warm *by burning off calories to provide heat!* Eat 'em and sizzle! ■

NATURAL PROTECTORS

At the U.C. Berkeley meeting in February, scientists from Finland, Italy, Switzerland, Netherlands, Japan, U.K., France, Germany, and the U.S. shared their ongoing research on the ways in which oxygen radicals can initiate cancers. Much of the discussion focused on using the enemies of oxygen radicals—the natural antioxidants—to fight cancer.

Our body has a love-hate relationship with oxygen. We can't live without it, but our system has to put up a continual battle because of the privilege of living with it! (Sounds like some marriages, doesn't it?) Peroxides, superoxides, and other highly reactive forms are such inevitable products of everyday cellular metabolism, that our cells have developed specific enzymes to tame them.

Two of the staunchest defenders against oxygen radicals (also called "free radicals" and "oxyradicals") are vitamin E and an enzyme, glutathione peroxidase, that requires the trace mineral selenium for its actions. Vitamin E is the chief scavenger of free radicals, while glutathione peroxidase keeps peroxide levels in check. Their actions complement one another. A lack of selenium in the diet means vitamin E has much more work to do. Vitamin A, pro-vitamin A (beta-carotene), and vitamin C are also warriors in the body's war against the dark side of oxygen. Some nice "cohort" studies are emerging to show the natural antioxidants also protect us against *cancer.* "Cohort" or "prospective" studies are coming into favor because they tell a useful story but don't require impossible amounts of personnel and funds.

For example, plasma levels of antioxidants were measured in a group of about 3,000 men in Switzerland, who were tallied for state of health, age, smoking habits, etc. In the follow-up 7 years later, a pattern clearly emerged. *The men who died of cancer were more apt originally to have had lower plasma levels of the antioxidant nutrients.* For example, low levels of beta-carotene were associated with subsequent cancers in the stomach and lungs; low vitamins C and E with stomach and colon cancers, and so on. A Finnish cohort study showed that development of cancer was related to lower *selenium* values at the time of the original blood sample. In this group, the risk of fatal cancer was more than 11 times higher for those whose original blood sample showed both low selenium and low vitamin E!

Oxygen: A Mafia Hit Man?

How does oxygen become a cancer-maker? One of the known ways is through damage done by oxygen radicals to the body's genetic material—DNA. Dr. Bruce N. Ames of U.C. Berkeley reported that there are likely to be "several thousand oxidative DNA hits per cell per day" in man! (I didn't ask him how many there are on a bad day!) Apparently, we repair these breaks routinely all the time. A mutant cancer cell can arise if the number of oxidative DNA hits becomes overwhelming and the repair system falters. But then our marvelous immune defense system goes into action to identify and destroy the aberrant cell.

The antioxidants work not only to keep the DNA hits down to manageable proportions, but also help maintain the immune system in top form. Vitamins E, A, and C are indispensable in this regard. (So are the essential omega-6 and omega-3 fatty acids.)

A number of reports at the meeting emphasized how protective vitamin E was against known carcinogens, for example ozone. The interwoven, cooperative role of nutrients was illustrated by experiments showing that after vitamin E has "soaked up" free radicals and exhausted its antioxidant capacity, our good friend vitamin C helps to regenerate vitamin E and restore its effectiveness again!

Modern Oils Make It Worse

The polyunsaturated fatty acids (PUFAs) in cellular membranes are prime targets for oxygen radicals. The more PUFAs we eat, the higher our tissue PUFA content becomes. (This is equally true of man and beast. For example, when hogs are given high-PUFA feeds, their flesh quickly reflects an altered ratio of PUFA to solid fat.) Our tissues need more anitoxidant protection as a result. One possible tie-in between modern high omega-6 PUFA consumption and cancer is that practically all of the PUFA is being supplied by oils and margarines, instead of by natural seeds, grains, and nuts, which would have been the traditional sources. Oils and margarines provide some vitamin E, but unlike the whole foods, they don't have any selenium, vitamin C, beta-carotene, methionine, B-vitamins, etc.—i.e., natural antioxidants and other nutrients to protect the new PUFAs formed in our tissues.

As a matter of fact, adding high amounts of "naked" oils to the diet makes the requirements for antioxidants go up! The vitamin E supplied by the oils may barely be enough for antioxidant protection of the new PUFA "body-parts," let alone any to spare for the rest of the body!

Several experiments demonstrated that even "ample" amounts of vitamin E (2 to 3 times the RDA) did not permit normal plasma levels of vitamin E to be maintained over the period of time WHEN OILS WERE THE ONLY SOURCE OF VITAMIN E IN THE EXPERIMENTAL DIET.

Does a hefty intake of "naked" oils and margarines, lacking the protective antioxidant nutrients to offset it, set the stage for free-radical damage and increased cancer? It's something to think about.

Protective Supplements

The dietary strategy of keeping our intake of fats and oils down to modest levels makes more sense all the time. In support of another good strategy, study after study of antioxidant nutrients (there are at least 200 published each year on vitamin E alone) shows convincingly that the free-radical fighters safeguard human and animal tissues against cancer. Environmental pollutants of the sort our ancestors never had to deal with are adding to the burden of oxygen radicals normally produced by our own bodies. Even if an individual is not "supplement"-oriented, they should consider taking protective amounts of vitamins E and C.

The omega-3 fatty acids in fish oil and linseed oil are highly polyunsaturated and hence vulnerable to breakdown by free-radical oxidation. While the amounts consumed even on a therapeutic regimen are not very large, nevertheless Dr. Rudin and other health professionals recommend supplements of vitamins E, C, beta-carotene, and the essential mineral selenium in generous enough quantities to protect the new "body-parts" formed from the omega-3 PUFA, as well as the rest of the system.

The good news is that, once incorporated into our tissues, the omega-3 PUFA can then go about their business of preventing an explosion of peroxides, superoxides, and other nasty oxygen species from being set off by omega-6 prostaglandins from arachidonic acid. This may well prove to be the most fundamental anti-cancer protection of all. ■

TAKE IT EASY, SUPERMAN!

Nowadays, people seem to fall into two camps: those who pursue fitness with steely-eyed zeal; and the rest of us shleppers. Couch potatoes of the world, rejoice! You may be saving your bodies a heap of wear and tear. The February workshop at U.C. Berkeley on oxygen radicals produced some studies to hearten the more languid among us. True, the subjects were rats and we mustn't jump to conclusions. Still, in a perverse way, I found the reports comforting. A group of the animals were given endurance training on a tiny treadmill. Running time was increased gradually so that by eight weeks, the trained ones could run a full six times longer than their untrained buddies before they keeled over from exhaustion.

Virtue Is Its Own Reward

And what do you suppose they got for their trouble? No accolades, no medals—just little bodies showing signs of heavy-duty battering, identified by the scientists as "oxidative damage"! The researchers suggest it was caused by vast numbers of oxygen radicals generated as a result of the accelerated metabolic activity, accompanied, of course, by extra oxygen consumption. It seems that in animals and humans, endurance training leads to greatly increased numbers of mitochondria in muscle tissue. Mitochondria are the energy-producing 'factories' in body cells.

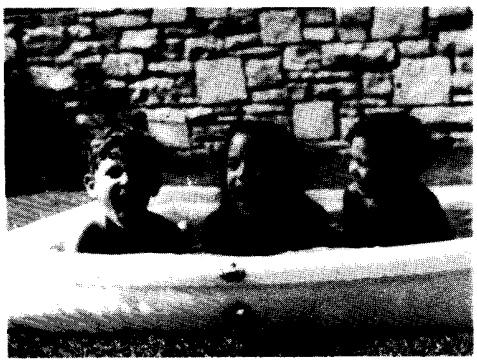
When extra power and endurance are called for, muscle cells in mice and men respond by building more 'factories.' In the mitochondria, a series of enzyme-controlled steps employ oxygen in the process of creating the desperately needed 'energy packets,' known as ATP. The same process, however, *happens to be the body's major initiator of free oxygen radicals.* (Again, our love-hate relationship with oxygen!)

Oh, No! Not Free Radicals Again!

The extra mitochondria made by the rats to deal with the demands of endurance training were not just supplying *energy* for the treadmill grind, they were also sending out a barrage of *free radicals.* As we know, these supercharged oxygen molecules can cause a heap of damage in the body, ranging from signs of early aging all the way up to fractured DNA.

The normal defense against free radicals are the antioxidants in tissues. Vitamins E, C, and beta-carotene, and the selenium-containing enzyme glutathione peroxidase all had to work overtime in the endurance-trained animals. The researchers noted that, in fact, levels of all major anti-oxygenic enzymes *did* increase after the workouts. At the same time, levels of vitamin E dropped—a sure sign that the vitamin was depleted in the struggle to defuse the oxygen radicals.

The downside effects of endurance training, e.g., oxidative damage and loss of vitamin E, were seen not just in muscles but throughout the whole system. One group of rats who were deprived of vitamin E had half as much endurance in the running trials as animals who got enough vitamin E. Besides going down in disgrace, the poor little critters suffered a lot more oxidative injury to their tissues!



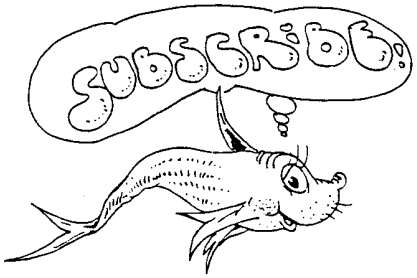
While rats (or mice) and men are not the same, the implications are pretty clear. There is indeed a very good chance that prolonged strenuous exercise, *even in endurance-trained athletes,* will take a toll in tissue damage. If the tissues are low in the essential nutrients required for antioxidant protection, all the consequences will be magnified. Vitamin E happens to be the major antioxidant. If an athlete or would-be athlete starts out with a deficiency, and loses even more vitamin E during heavy workouts, the chances of letting loose a meteor shower of free radicals to bombard the body become that much greater.

Where Do We Go From Here?

In order to shield our bodies from oxidative attack, must we then confine our major workouts to pushing buttons on the TV's remote control? I hardly think so. The benefits of at least moderate, regular exercise are too well-documented to allow such a cowardly retreat! Lusty physical activity puts a sparkle in the eyes, sweetens the disposition, and increases protective HDL-cholesterol carriers in the blood. Besides, our lymphatic system, which carts nutrients to our cells and waste materials from them, is designed to be activated solely by muscular movements, which squeeze and stimulate the lymph vessels to do their invaluable chores.

The question seems to boil down to: How much exercise can human bodies handle without unleashing a flood of dangerous free radicals to undo the good effects? I don't think this question has any easy answers, if only because of the tremendous difference in individuals. Much more research is needed. In the meantime, whether we choose to exercise frivolously or passionately, it would seem logical to take the sensible precaution of obtaining—via diet, and supplementation where necessary—a generous supply of the nutrients that build a powerful antioxidant defense system.

Lester Packer, Ph.D., one of the organizers of the conference and a leading researcher in antioxidants, was my physiology professor at U.C. Berkeley when I was a re-entry student in the nutrition department more than ten years ago. I still remember the large-scale classroom model he used to show how molecules of vitamin E snuggle in between the phospholipid molecules (fatty acids plus a phosphorus group) that make up the membranes of our cells; they were there, he told us, specifically to protect the unsaturated fatty acids in membranes from oxidative attack. I remember asking him then if he himself took supplements of vitamin E. He said he did. He was buzzing around in his usual frisky fashion at the conference in February. After the workshops and panels were over, there was a dinner cruise on San Francisco Bay for participants and guests. We watched the sun set behind the Golden Gate bridge. And then, wonder of wonders, because the bay was exceptionally calm, the captain of the Admiral Hornblower sailed his little yacht right under the bridge! A great bluegrass band was playing lively hoedown music, and Dr. Packer, Dr. Ames, and a bunch of other worthies were stomping fit to kill. I'll bet he still takes vitamin E. ■



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