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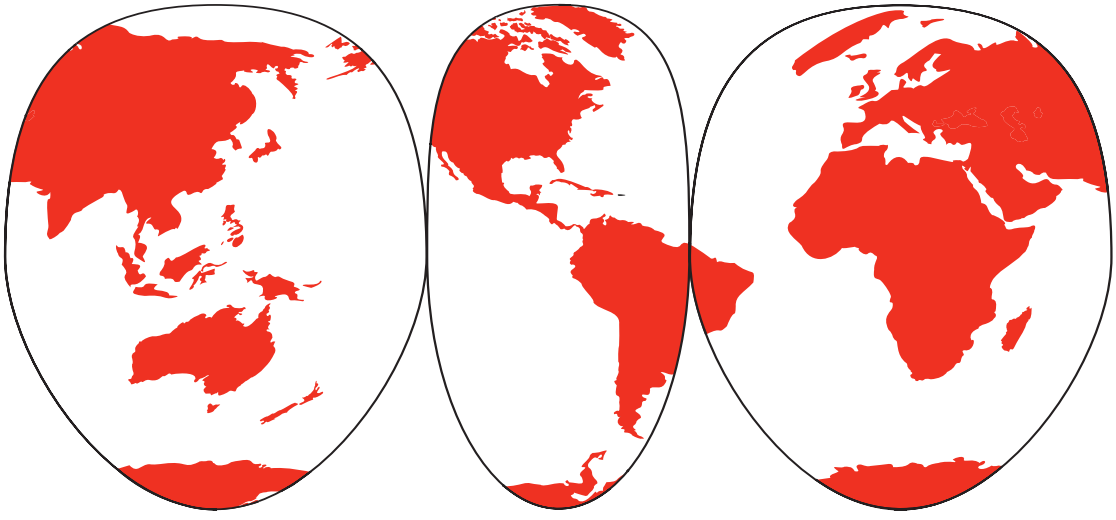
Heart Disease Issue

Heart Disease Issue



HEART DISEASE ISSUE

What Causes Heart Disease? A New Theory of Heart Disease
The Many Roles of Cholesterol The Benefits of High Cholesterol
The Dangers of Statin Drugs CoEnzyme Q10 for Healthy Hearts
Cholesterol and Stroke The Oiling of America

A PUBLICATION OF
THE WESTON A. PRICE FOUNDATION®

Education • Research • Activism

TECHNOLOGY AS SERVANT SCIENCE AS COUNSELOR KNOWLEDGE AS GUIDE

WiseTraditions

IN FOOD, FARMING AND THE HEALING ARTS

Heart Disease Issue

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THE WESTON A. PRICE FOUNDATION®

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The Weston A. Price Foundation is a nonprofit, tax-exempt charity founded in 1999 to disseminate the research of nutrition pioneer Weston A. Price, DDS, whose studies of isolated nonindustrialized peoples established the parameters of human health and determined the optimum characteristics of human diets. Dr. Price's research demonstrated that men and women achieve perfect physical form and perfect health, generation after generation, only when they consume nutrient-dense whole foods and the vital fat-soluble activators found exclusively in animal fats.

The Foundation is dedicated to restoring nutrient-dense foods to the American diet through education, research and activism and supports a number of movements that contribute to this objective, including accurate nutrition instruction, organic and biodynamic farming, pasture-feeding of livestock, community supported farms, honest and informative labeling, prepared parenting and nurturing therapies. Specific goals include establishment of universal access to clean, certified raw milk and a ban on the use of soy-based infant formula.

The Foundation seeks to establish a laboratory to test nutrient content of foods, particularly butter produced under various conditions; to conduct research into the "X" Factor, discovered by Dr. Price; and to determine the effects of traditional preparation methods on nutrient content and availability in whole foods.

The board and membership of the Weston A. Price Foundation stand united in the belief that modern technology should be harnessed as a servant to the wise and nurturing traditions of our ancestors rather than used as a force destructive to the environment and human health; and that science and knowledge can validate those traditions.

The Weston A. Price Foundation is supported by membership dues and private donations and receives no funding from the meat or dairy industries.

Wise Traditions

IN FOOD, FARMING AND THE HEALING ARTS

Heart Disease Issue

A PUBLICATION OF

THE WESTON A. PRICE FOUNDATION®

Heart Disease Issue

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President's Message

This special reprint makes available articles on heart disease, cholesterol and cholesterol-lowering diets and drugs that have been published in *Wise Traditions*, the quarterly journal of the Weston A. Price Foundation, since the Spring, 2001 issue.

The recent publication of results from the ENHANCE trial, which found no benefit from a drug combination that significantly lowered LDL-cholesterol but did not reduce plaque formation in the arteries nor confer a projected reduction in mortality, has received widespread attention in the media, including an article "Do Cholesterol Drugs Do Any Good?" in the January 17, 2008 issue of *Business Week*.

According to the article, many researchers now question the wisdom of prescribing cholesterol-lowering statin drugs to the general population—drugs the pharmaceutical industry believes should be taken by 40 million Americans. Growing doubt among the ranks of medical professionals has emerged with the accumulation of reports on serious side effects from cholesterol-lowering measures: muscle weakness, neuropathy, heart failure, memory loss, depression, fatigue, digestive disorders and cancer.

Results of the ENHANCE trial have led to the startling revelation that the studies on which the FDA based its approval of statin drugs looked only at surrogate outcomes, namely the lowering of LDL-cholesterol and raising of HDL-cholesterol, as a substitute for a clinically meaningful endpoint, namely the prevention of heart attacks. Up to this point, drugmakers have not had to show that statins actually save or extend the lives of patients.

What drugmakers have done for the past 30 years is create the impression that they do, often by exaggerating the benefits of their drugs using the parameter called "relative risk." For example, a widely published advertisement for the statin drug Lipitor proclaims, "Lipitor reduces the risk of heart attack by 36 percent in patients with multiple risk factors for heart disease." In the fine print, the reader learns that 3 percent of patients taking a placebo had a heart attack versus 2 percent of patients taking a Lipitor. The exaggerated figure of 36 percent is obtained by comparing the two numbers without reference to the sample size. You will read about other statistical tricks in this issue.

Researchers are also re-examining the promotion of soul-numbing lowfat diets. "Dietary fat recommendations. . . may have led to significant and harmful unintended consequences," wrote the authors of a January 22, 2008 article in the *American Journal of Preventive Medicine*. Official government guidelines have indeed misled Americans into abandoning nutritious whole foods such as butter, eggs and organ meats, foods universally recognized by traditional peoples as necessary for good health and optimal development of children.

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STATIN PAYMENTS

You may have read that doctors receive payment or bonuses for prescribing statins, the cholesterol-lowering drugs. I'm a chapter leader in Kauai, and a family physician, so I'm in a good position to fill in some details about how doctors actually get paid more for writing more statin prescriptions. The mechanism is a little cumbersome to describe clearly, but I'll take a stab at it.

We have a series of "quality measures" that are tracked by the insurance company. One quality measure is the number of mammograms we do on our patients between ages 40 and 69, another is that we send our diabetic patients to the eye doctor once a year for retinal exams. For our patients who carry a diagnosis of "coronary artery disease," we have to write them a prescription for a cholesterol-lowering drug. If any one doctor doesn't follow any one of these imperatives, he loses points toward a cash bonus, and the entire group is similarly penalized. As you can imagine, there is lots of peer pressure to prescribe!

Actually, we don't get our bonus unless the patient goes and buys the drug or gets the test or sees the eye doctor and so on, so it's not enough just to write the prescription, we have to talk up the drug enough to get them to go out and buy it. Currently, there are only a few means by which a person can be labeled as a patient with coronary artery disease. Having a heart attack is one, and having abnormal results on heart tests (like angiograms) is another. Diabetes is now considered a "coronary artery disease equivalent" and so, in the near future, doctors may be required to get all our patients who have type one or type two diabetes to take their statins, or lose more money.

These HMOs are insurance companies like Blue Cross, which offer their clients (employers and patients) HMO programs. The HMO plan we have is offered by HMSA (Hawaii Medical Something Something). For whatever reason, HMSA wants to offer an HMO program for people, and doctors who participate as providers must comply with the rules of the program and accept payments according to the rules. There are clear benefits to pharmaceutical companies in this structure but no obvious reason why HMSA would want to encourage people to buy expensive drugs that HMSA must pay for. One might speculate that there are some quid-pro-quo relationships between the insurance companies and the pharmaceutical companies, but I have no idea what they are. However the ties are structured, I feel, as do many other scientists, that these kinds of business relationships lead to behaviors that pose real threats to patient care, and to human health in general. Because industrial connections like this fund most research, they distort the scientific process and are far more insidious, invisible, and totalitarianistic than expensive dinners and trips to Hawaii, which are what the media would have us believe is the sum total of the problem.

By the way, the bonus is actually not a bonus at all. This is where it gets Orwellian. We give up a certain percentage of the payment for accepting HMO patients, and we get it all back, in theory, if we meet all of our quality measures. We never do because of computer glitches which continually fail to track our prescribing, testing, and referring patterns accurately. Nobody can explain why we've agreed to accept HMO insurance plans, but we seem to feel we have no choice. And we will have less choice before long; Medicare is planning to begin similar programs. Each of these programs takes more money away from the doctors and gives it to middle managers, ensures that drug companies get more money, and that expensive tests of limited value are done more often.

These are some reasons why savvy business people are going into "alternative" medicine where they benefit from cash payments and total autonomy. Several here on Kauai are making millions.

Catherine Shanahan MD
Kalaheo, Hawaii

SHOCK IN HOLLAND

Holland has been shocked by a tragedy in which a police officer shot his wife, three sons and then himself, apparently without any reason. These things have happened before, but I can't help but speculate.

A few months ago I interviewed a statin victim, also a police officer, who told me, that "when they changed me from Zocor to Lipitor, the muscle pain only got worse. And the world turned even blacker than it had been. It got so bad that I took a nail and connected myself to the grid. But I did not succeed. Then the doctor put me on Seroxat as

Letters

well, for the depression. Man, you cannot guess what happened to my head on that combination. I had this scary urge to take my gun and just shoot everybody."

This officer told me that policemen in Holland get regular checkups and are talked into a statin as soon as their cholesterol is a little above 200. "Half the service is on a statin," he told me. "When they get the statin, they start functioning lousy. Then they go on Seroxat and feel dumb."

Melchior Meijer
Zoutkamp, The Netherlands

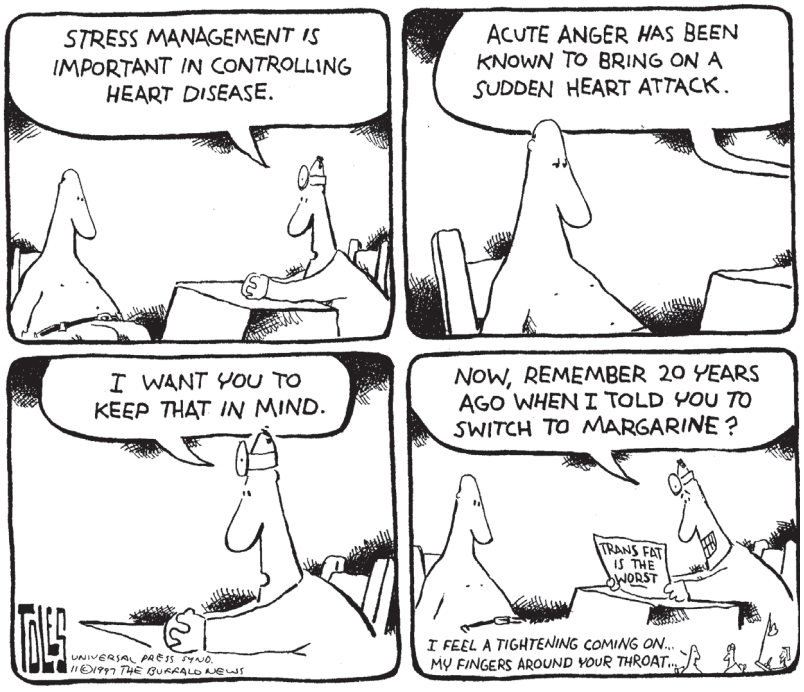
A FABRICATION

The so-called "Mediterranean Diet" is an American fabrication for the simple reason that in Italy—let alone in the entire Mediterranean area—people eat in different ways. Yet there was historically such a thing as an Italian diet. Here is the story: at the end of the 19th century, Italy had just been unified into a brand new nation. At that time Pellegrino Artusi wrote a book of recipes entitled *The Science in the Kitchen* and the Art of Eating Well. It was a collection of traditional recipes from Tuscany and Emilia-Romagna (concerning food, Emilia is for Italy what Bourgogne is for France) and it became the second bestselling book in Italy (the first being the Bible). It is actually mentioned in a high school text book, *History of Italian Literature*, and for good reason as it was responsible for the spread of a common language in the middle class of the new nation. It remained the bible of Italian food for the middle class until the 70s-80s, when the lowfat craze kicked in. I remember that as the time when we really started eating pasta and bread.

The Artusi book is the antithesis of what today is called the Mediterranean diet: for instance one recipe for breakfast calls for eggs, butter, anchovies, capers and tuna. Artusi emphasizes the use of animal fat and meat; in fact, the book is a feast of animal food. The book actually starts with a rating of the nutritive power of different kinds of meat, with beef at the top of the list. There is a section about pasta in which Artusi warns children, elderly and pregnant or lactating women from consuming pasta "because it would distract from the consumption of more nutrient-rich foods, as meat or fish. . . " and cautions, "people with tendency toward obesity" to refrain from consuming it "because every doctor knows that flour has no nutritive power and immediately turns into body fat."

The most famous Italian products are animal-based: 400 kinds of traditional cheese (most of which are required by state-enforced purity laws to be made from raw milk, like Parmigiano Reggiano) and hundreds of cold cuts (prosciutto crudo, prosciutto cotto, salame, coppa, pancetta, mortadella, to name a few).

During the 1950s (when Ancel Keys visited Italy and initiated the Mediterranean diet myths) a lot of people had a hard time affording meat, especially in the south. But that was certainly not considered something good. In fact most families that could not afford meat would still buy little pieces of it, at least once a week, to feed the kids. My grandpa, who fought in WWII, would tell me sometimes: "Quit complaining about food. You can have meat twice a day, you don't know how lucky you are. At your age I knew



Letters

what famine was like." Elders who went through fascism, war, German occupation, and then saw their towns destroyed by Anglo-American bombings would commonly speak that way to the new generation.

Finally, in a local newspaper from the northern Italian town I'm from, there is a historical page—sort of "the way we were." A few months ago it published the following documents from its archive: at the beginning of the 1920s, the price of food was increasing. A group of "middle-class housewives" wrote to the authorities asking for the creation of a committee to control the prices. They also wrote down a list of the essential goods whose price should be kept controlled, in order of importance. The most important was "first choice butter." Then came the "second choice butter." Then lard. Then olive oil. Then a list of meats and cold cuts. There is no mention of bread or pasta in the list. Very different from the so-called "Mediterranean diet."

Cristiano Nisoli
University Park, Pennsylvania

NO BENEFICIAL ROLE

I want you to know how much I'm enjoying my first issue of the WAPF newsletter. I was happy to see, in one of the letters you published, that I'm not the only RD in the organization.

I thought you might be interested in something I read the other day. I am a member of the American Dietetic Association and was doing some catchup reading when the following paragraph caught my eye (from an article on the new Dietary Reference Intakes in the Fall '02 "Dietetics in Practice," a quarterly ADA publication):

"According to the report [from the Institute of Medicine's Food and Nutrition Board], saturated fat and cholesterol provide no know [sic] beneficial role in preventing chronic diseases and so are not required at any level in the diet. Since completely eliminating saturated fat and cholesterol from the typical American diet would make it difficult to meet other nutritional guidelines, the panel recommended keeping intake as low as possible while maintaining a nutritionally adequate diet."

Hmmmm. . . I'd like them to take a look at the lipid profile of human milk and tell me again that saturated fat and cholesterol are not required at any level! Or do they believe our nutritional requirements change that dramatically when we wean?

Thanks again for all you do. I can't tell you how glad I am that I found the WAPF!

Amy Crown
Tucson, Arizona

STATIN MADNESS

This statin craziness gets worse. I've a friend with a cholesterol level of 157 who was put on the statin drugs with the intention of getting it down to 120. She immediately got sick and stopped the drugs. Another friend's 89-year-old mother in reasonable mental health was put on a statin drug a year ago and now has advanced senile dementia. Age or drug? No proof either way, but I've got my suspicions.

Glenda Glass
Ione, California

MEMORY LOSS

Thank you for a great article on cholesterol on your website. We are currently dealing with memory loss with my mother-in-law, age 70. She has been on lovastatin (mevacor) for several years and her memory has gotten worse and worse. She went for a battery of tests and the doctor said it was not dementia or Alzheimer's, but couldn't relate it to anything else.

I suspected statin drugs from different articles I had stumbled upon. Lo and behold we discovered she was indeed taking lovastatin. We took her off for a few weeks and she seemed better, less in a fog.

Then the doctor treating her memory loss told her to go back on the statins. "If your primary doctor prescribed them, you must need them," he said. We were fit to be tied. If anyone knew about the tie-in to memory loss and these drugs, it should have been him!

After doing a little research on this doctor we discovered that he had recently received a nice big grant from the pharmaceutical industry to do research on using statins to improve memory in Alzheimer's patients!

When we visited her primary care doctor, we were able to convince her to take my mother-in-law off statins for two months at least. She did acknowledge the possibility of a tie-in to the memory issues. But then she immediately said, "If that is the case, then we will really be stuck for a new med to lower her cholesterol. It is important to do that."

Well, after reading numerous articles about how damaging statins are and how little they really do, I must ask,

Letters

isn't quality of life better than (so-called) quantity of life, assuming the claims of increased lifespan are true? Is it better for my mother-in-law to go around confused and in a fog than to risk the potential heart issues that these drugs are "supposed" to prevent? All the research I see shows no positive proof to back up those claims, yet this drug is the number one selling drug (and quite expensive, too) in this country. Not to mention the dangers of lowering cholesterol levels too low and damaging other key functions and components of our bodies?

How do lowly consumers go about getting their voices heard to help prevent millions more from being damaged by these awful drugs? When did doctors in this country become legal drug pushers, turning healthy adults into pill-popping patients? When a patient identifies a side effect to a drug, why isn't it reported? I find it hard to believe that all the people who claim memory loss and amnesia, as well as muscle and other problems, with these drugs are just making it up.

Jean Golden
Sacramento, California

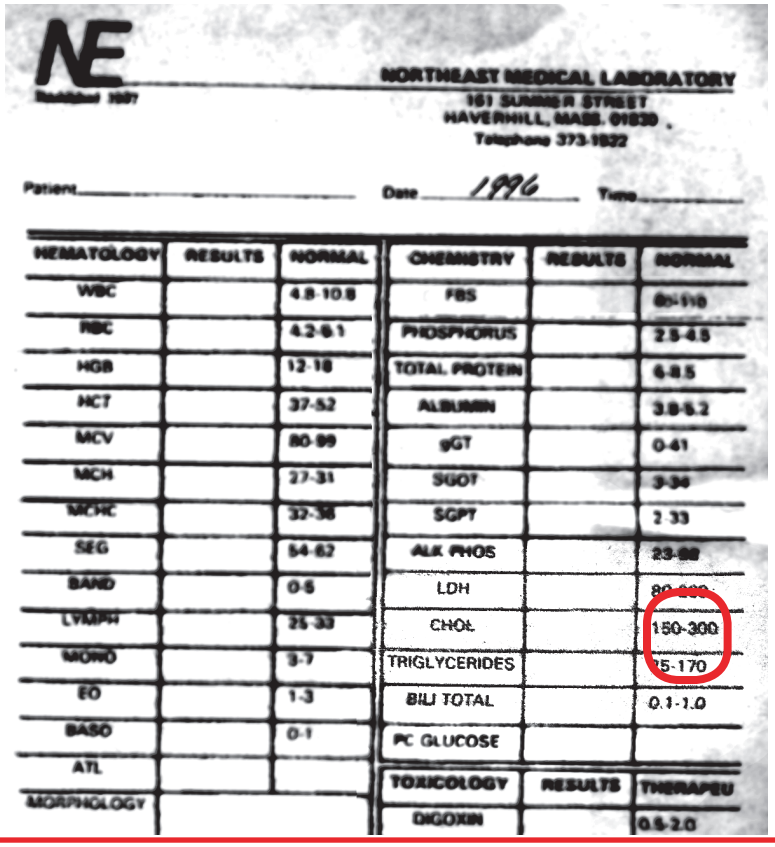
The best way for consumers to avoid being damaged by statin drugs is to "just say no." Only when large numbers of patients begin voting with their feet will the power of the pharmaceutical industry start to wane. Our role at the Weston A. Price Foundation is to provide the information patients need to find the courage to refuse statin drugs. For proof that "high cholesterol" is a new and invented disease, read on.

NORMAL CHOLESTEROL

I have in front of me a copy of my mother's cholesterol report taken in 1996. The established parameter for normal cholesterol is listed at 150-300. She typically runs around 270 and had experienced a lot of pressure to go on statins; she fortunately has enough confidence to "just say no."

She had been on a very lowfat diet for years and was never able to lower her cholesterol levels very much. She now suffers from lung problems. Fortunately she has become an avid consumer of raw milk, raw milk kefir, eggs and other nutrient-dense foods. Thank you for your research and all your posts on this subject.

Rhonda Mullis
Deltona, Florida



NE

NORTHEAST MEDICAL LABORATORY
161 SUMMER STREET
HAVERHILL, MASS. 01830
Telephone 373-1822

Patient _____ Date 1996 Time _____

HEMATOLOGY	RESULTS	NORMAL
WBC		4.8-10.8
RBC		4.2-6.1
HGB		12-18
HCT		37-52
MCV		80-99
MCH		27-31
MCHC		32-36
SEG		54-62
BAND		0-6
LYMPH		25-33
MONO		3-7
EO		1-3
BASO		0-1
ATL		
MORPHOLOGY		

CHEMISTRY	RESULTS	NORMAL
FBS		65-110
PHOSPHORUS		2.5-4.5
TOTAL PROTEIN		6-8.5
ALBUMIN		3.8-5.2
gGT		0-41
SGOT		3-34
SGPT		2-33
ALK PHOS		23-92
LDH		80-225
CHOL		150-300
TRIGLYCERIDES		35-170
BILI TOTAL		0.1-1.0
PC GLUCOSE		

TOXICOLOGY	RESULTS	THERAPEU
DIGOXIN		0.5-2.0

A CANDIDATE FOR STATINS?

I've been a WAPF adherent for about a year. A few weeks ago I started having frequent heart palpitations. Naturally, I was frightened. Although I wanted to find out what was wrong, I was also afraid to visit my doctor for fear they'd want to check my cholesterol, and heaven forbid anything be out of line or they'd try to put me on medication. But with my mother's recent heart attack looming over me, and since the palpitations weren't going away, I made an appointment. My doctor did as thorough an in-office exam as possible, checking for all sorts of things. He also ordered the dreaded lipids panel. After getting the EKG results, he told me that he could detect nothing wrong with my heart function, although if the palpitations continued we could consult a specialist. I felt enormously relieved to know I wasn't going to keel over from heart failure anytime soon, and

Letters

hoped that my blood chemistries would also be reassuring. Wouldn't you know, right away my palpitations decreased in frequency, and over the next few days almost disappeared! I gave the matter some thought and realized that I had been under quite a bit of stress, with many demands on my time.

Then I got the call from the doctor's office. My labs looked fine, except for my LDL-cholesterol, which was "sky high" at 188, and could I come in soon to discuss treatment? I spent a week's worth of my son's naptimes on the computer, looking for information that could assuage my fear.

Today I returned to my doctor's; I was so anxious that on the way there I gave myself a running pep talk. I thought perhaps I was just paranoid, and that surely he wouldn't prescribe statins to an otherwise healthy 34-year-old woman. Guess what? Apparently no one is a bad candidate for drugs nowadays, because I came away with a prescription for simvastin! (Don't worry, there's no way I'm going to take the stuff.) It seems that even though my HDL is outstanding at 84, my triglycerides are fine at 99, my risk ratio is a perfectly acceptable 3.5, my other labs were totally normal, and I have absolutely no signs of heart disease (other than the now-resolved palpitations), I simply cannot continue with such high LDL. He admitted that my cholesterol had nothing to do with the palpitations, and even that my risk ratio and HDL are good, but kept saying that my LDL had to come down, preferably under 100.

But what really blew my mind was when I told him that I was very concerned about starting meds right now, since my husband and I are currently trying for another child. I thought he'd surely agree to postpone any treatment, but instead he said to go ahead and start the meds, and then quit once I became pregnant! He then told me that not taking medication during the pregnancy and breastfeeding period wouldn't be a big deal, since it wasn't like I was going to have a heart attack right now. So if it's no big deal, then why risk my taking them during early pregnancy??

Hollie Regalo
Murfreesboro, Tennessee

VANISHED TINS OF FAT

Now in my 80s, I have eaten butter all my life, not just a little smear, but I have plastered that so-called yellow poison on my bread, my toast, mashed potatoes, you name it, I have always applied it with a heavy hand. And I will not buy meat unless it has fat on it. My blood pressure is normal and I am as active and mentally alert as someone in their 50s.

Back in the 1940s, the doctors told my great grandmother not to eat fat—they were doing it even then. "But I like fat," she said, as she sliced through rolled roast of beef. She ignored them and lived to an advanced age.

When I was young, fat was never wasted. Drippings from the roast were kept in one tin and fat from the bacon in another. They were later used for frying, smeared on bread and for making suet puddings and many other delicious edibles.We did not use oils, except for cod liver oil. We drank full fat milk and in my farming days, we would not consider butchering and dressing a thin animal. And we spread additional fat across the roast before it went in the oven.

A motto to live by: listen to your body, not the dietitians. Enjoy your food and don't be afraid of fat. It's not going to kill you. And keep those tins of fat on hand!

Geoffrey C. Morell
Washington, DC

SATURATED FAT MIRACLE

I saw your website and I want to briefly tell you about my wife. She has had autistic symptoms all her life, and for the last two years, Addison symptoms so serious she was a semi-invalid and very depressed. She is now cured (since last August). She is happy, works hard, no symptoms, no medication, all her lifelong autism symptoms are gone as well! It was truly a miracle. By accident and out of desperation she tried a diet extremely high in saturated fat. That was all. In three days she was a new person! I think someone should get the word out about this.

Doc Scantlin
Huntingtown, Maryland

Caustic Commentary

Sally Fallon and Mary Enig take on the Diet Dictocrats

SUMMER 2001

THE SCHEME TO INTERVENE

The Federal government has issued draconian new guidelines for cholesterol-lowering to prevent heart disease. The guidelines are aimed at all Americans age 20 and over, with waistlines greater than 40 inches and whose LDL cholesterol levels are over 100. "Many more people are eligible for treatment under the new guidelines," said one commentator, "because the population has gotten more overweight." Recommendations include cutting intake of saturated fats to less than 7 percent of total calories and cholesterol intake to less than 200 mg per day. "At risk" individuals are encouraged to consume cholesterol-lowering margarines and salad dressings and to eat lots of grains, beans, fruits and vegetables—the kinds of foods that make many people gain weight. Most importantly, the government recommendations will make 36 million people candidates for cholesterol-lowering drugs—three times the number currently taking them. Drugs for cholesterol-lowering already constitute a huge market. Sales of Lipitor, for example, bring in more than $5 billion per year for Pfizer. With the new recommendations, pharmaceutical stock prices naturally have shot up. The more stringent guidelines and harsh tone of the report are said to be necessary because Americans are not taking prevention of heart disease seriously enough—which means that sales of cholesterol-lowering drugs and lowfat, cholesterol-lowering imitation foods are not increasing fast enough to please the multinational corporations that sell them. The guidelines are also well timed to stem the fallout from the publication of "The Soft Science of Dietary Fat," an explosive exposé in the March 30 issue of *Science*. Author Gary Taubes points out that 50 years of mainstream nutritional research and hundreds of millions of research dollars have not proved that eating a lowfat diet will help you live longer. Taubes notes that the principal political supporter of the lowfat agenda was Senator George McGovern, who had spent some time on the severely lowfat Pritikin diet. . . before dropping out of the program. The McGovern Committee's "Dietary Goals for the United States," which almost single-handedly changed nutritional policy in the US, was written by a vegetarian, Nick Mottern, a former labor reporter with no background in nutrition. Thus have government, science and industry put their curse on healthy traditional foods and ushered millions of perfectly healthy Americans into the jaws of the medical care system.

SENIOR MOMENTS

The drugs that so many Americans now take to lower their cholesterol are called statins. They work by blocking an important enzyme the body uses to make cholesterol. Researchers say that statins are completely safe, even though many studies show a correlation of statin use with increased risk of cancer, intestinal diseases, stroke, depression, accidents and suicide. In May, a retired physician participated in an interview on *The People's Pharmacy*, a national radio show, to describe another side effect—memory loss. Dr. Duane Graveline said he experienced bouts of total amnesia while taking the drug. Spokesmen for Pfizer, the makers of the statin-drug Lipitor, say that there has never been a single case of amnesia reported in any of the clinical trials on the drug. Nevertheless, a warning of potential problems with memory, insomnia or depression is listed on the product label. Dr. Graveline says he would never take another statin drug and is concerned that doctors may attribute cognitive problems in their patients to aging or Alzheimers rather than entertain the premise that statin drugs might be the cause. (The May 28 issue of *US News & World Report* follows its article on the new guidelines with an article on Alzheimers, oblivious of the irony.) Even worse, he said, is the possibility that doctors may prescribe statins to people whose memory loss might be disastrous, such as airline pilots or school bus drivers.

ET TU, DIABETES

The new guidelines do not spare diabetics, whose condition is now included as a risk factor that must be treated with lowfat diets high in grains and other carbohydrates. This comes in the wake of a study by Dr. James Hayes, an endocrinologist and director of the Limestone Medical Center in Wilmington, Delaware. Whereas most type-II diabetics are encouraged to get at least 60 percent of their calories from carbohydrates, he put his diabetic patients on an 1800-calorie diet with 50 percent of caloric intake from fat and just 20 percent from carbohydrates. Ninety percent of the fat content in the diets was saturated fat. The patients showed an impressive weight loss and normalization of blood parameters without ketosis.

Caustic Commentary

FALL 2001

CHOLESTEROL AND THE ELDERLY

Damage control experts are dealing with yet another study that disproves the theory that high cholesterol levels are a bad thing. Researchers participating in the Honolulu Heart Program measured cholesterol levels in 3572 Japanese American men (aged 71-93) and compared changes in cholesterol levels over 20 years with all-cause mortality. In general, cholesterol levels fell with increasing age, but the researchers were astounded to find that the earlier patients start to have lower cholesterol concentrations, the greater the risk of death. Furthermore, those with higher levels of cholesterol had better hemoglobin status and hand grip strength. In other words, when cholesterol levels go down in the elderly, so does physical function and they become frail. "We have been unable to explain our results," said the investigators. They urged "a more conservative approach in this age group." What that means is that it is not a good idea to put the elderly on lowfat diets and cholesterol-lowering drugs, but don't expect to see this finding translated into medical policy anytime soon (*The Lancet* 8/4/01 358:351-355).

I'm from the cholesterol police, and I'd like a word with you.

WINTER 2001

AMERICA ON PARADE

What America Eats is the subject of a special issue of *Parade Magazine* (November 11, 2001). In it we learn that the average amount of time spent preparing the family dinner is 33 minutes; that one-third of Americans buy more convenience foods than they did just two years ago; that pizza is America's favorite food; and that 66 percent of Americans eat breakfast at home—usually cold cereal. Sixty-eight percent of Americans eat cold cereal as a snack and 27 percent admit to having cold cereal for dinner. Americans are eating more chicken, fish and veggie burgers. Still, 82 percent of Americans eat cold cuts. Nutrition advice includes eating more fish, more tea and more monounsaturated fats like olive oil and canola oil. Since Americans are eating less meat and fewer eggs, foodmakers are fortifying "healthier" foods with choline, a nutrient needed for brain development, which we used to get from meat and eggs. A Dr. Isadore Rosenfeld advises Americans to eat a "good" breakfast of orange juice, skim milk (or soy milk) and cereal, but to avoid bacon, ham and sausages. "Such a breakfast can only lead to diabetes, hypertension, obesity and hardening of the arteries, and is . . . worse than no breakfast at all," he says. "Experts" providing food advice include the CEOs of Nestlé, ConAgra, Kraft and Campbell Soup, who predict that next year Americans will use more processed foods. Interspersed with this ageless wisdom are advertisements for drugs to treat menopause, heartburn and osteoarthritis, and mattress pads for fibromyalgia sufferers.

SPRING 2002

MORE CHOLESTEROL MADNESS

In spite of widespread cholesterol-lowering measures, heart disease remains the top killer in the US, according to a new report (*Washington Post*, January 1, 2002). Almost one million Americans per year die of heart disease, twice as many as die from cancer. The American Heart Association's insistence that we be more diligent in following a lowfat diet represents the triumph of hope over experience. Ever since the mid-1930s, when Americans began to consume supposedly lower-fat processed foods based on vegetable oils, the rate of heart disease has continued to climb. And, naturally, the report is being used to promote greater use of drugs to lower cholesterol. In fact, according to an article in the *Wall Street Journal* by Thom

Burton, many insurers now grade doctors' performances and dole out monetary bonuses and penalties based on measuring and "improving" patients' cholesterol levels. And the fastest and easiest way for doctors to lower cholesterol is to prescribe a powerful statin like Pfizer's Lipitor. The new government guidelines are structured in such a way as to transform virtually every American into a candidate for cholesterol-lowering drugs, and Pfizer's profits are climbing. Income for the huge pharmaceutical company rose 38 percent in the last quarter of 2001 to $1.93 billion. Karen Katen, president of Pfizer's human pharmaceuticals group, said Lipitor "still has enormous room to grow" because of "widespread under-diagnosis of high cholesterol" (*Wall Street Journal*, January 24, 2002). Enormous creativity has been shown in increasing the market for these expensive and toxic drugs, including drug-discount cards for poor Medicare beneficiaries, American Heart Association literature aimed at Blacks and Hispanics promoting use of vegetable oils and egg substitutes, and smiling football coaches in full page ads promoting statin drugs. Meanwhile, yet another study has linked low cholesterol levels with depression (*Psychosomatic Medicine* 2000, 62), creating new customers for antidepressants. It's a crazy system based on fear and a misplaced respect for what passes as medical science.

SUMMER 2002

NEW GUIDELINES, MORE PATIENTS

The "New Cholesterol Guidelines" have turned tens of thousands more healthy people into patients, "eligible" for cholesterol-lowering statin drugs. When a correspondent asked the National Heart, Lung and Blood Institute (NHLBI) why there were no open meetings required for the development of the new standards, and why the New Guidelines were not published in the Federal Register, he received the following amazing reply: ". . . the guidelines for cholesterol management released on May 15, 2001 were developed by a panel of experts—the Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III [ATP III])—convened by the National Cholesterol Education Program, an educational program coordinated by the National Heart, Lung and Blood Institute. The ATP III panel is not an advisory committee to the NHLBI but rather a group of recognized experts providing their scientific judgement about cholesterol management to clinicians. The panel's recommendations for clinicians are based on a thorough review of the scientific evidence by the panel. The guidelines developed by the ATP III are not regulations and health professionals are not required to follow them." The "recognized experts" include Drs. Grundy, Hunninghake, McBride, Pasternak, Stone and Schwartz, all of whom have received consultant fees from the producers of statin drugs.

NEW CHOLESTEROL TEST, MORE PATIENTS

Atherotech, Inc., a leading cardiodiagnostic company, has announced the completion of a private offering of $11.5 million in financing to be used to further the "rapid widespread adoption of the company's VAPTM (Vertical Auto Profile) cholesterol test as the new standard of care in cholesterol risk assessment." The test "detects 50 percent more people at risk for heart disease than the traditional cholesterol panel." According to a company press release, "The VAP Test is available in 43 states, and we expect another stellar year in 2002 as physicians convert to the VAP Test to comply with the recently released NCEP ATP III guidelines."

MORE GRUMPY PATIENTS

Scientists have identified low testosterone as the cause of "Irritable Male Syndrome," the grumpy, noncommunicative, moody male that makes life miserable for his wife and family (*Examiner*, May 26, 2002). With the New Cholesterol Guidelines and the new VAP cholesterol test, families can expect more exasperating behavior in their menfolk—because testosterone is made out of cholesterol. When you lower cholesterol with lowfat diets and statin drugs, the results can be tragic for all involved, as chronic low cholesterol levels lead to depression and irrational anger. The whole cholesterol story adds up to an incredible phenomenon—drug companies

FOR SCIENTISTS AND LAYMEN

Please note that the mission of the Weston A. Price Foundation is to provide important information about diet and health to both scientists and laymen. For this reason, some of the articles in *Wise Traditions* are necessarily technical. It is very important for us to present the science that supports the legitimacy of our dietary principles.

In articles aimed at scientists and practitioners, we provide a summary of the main points and also put the most technical information in sidebars. These articles are balanced by other pieces that explain our principles in simpler terms and provide practical advice to our lay readers.

promoting a dangerous drug as though it were government policy, new guidelines and new tests to convince the majority of US adults that they need to lower their cholesterol, and then the tragic consequences—black moods, sudden anger, hell on earth. . .

MEAT STUDIES

The press has been quick to publicize a new study claiming "Teen Vegetarians Healthier Than Meat-Eaters." What did the researchers deem "healthier?" The vegetarian teens had lower intakes of fat, including saturated fat, and ate more vegetables. (Never mind that the vegetarians "drank more diet soda and caffeine," reflecting the desire of most of the teenagers to keep weight off.) There was no front page coverage for a study showing that animal protein consumption is associated with greater bone density in the elderly (*Am J Epidemiol* 2002;155:636-644), nor for a study showing that blood homocysteine levels are higher in vegetarians than in meat eaters (*J Nutr* 2002 Feb;132(2):152-8), implying that vegetarians are more at risk for heart disease. And, finally, steak lovers will be pleased to learn that researchers in Lyon, France found that processed meats were linked to colon cancer but consumption of fresh (unprocessed) red meat does not raise the risk of colon cancer (www.msnbc.com/news/591170.asp).

FALL 2002

A NEW ENEMY

Now that the public has discovered that half of all heart attacks occur in individuals with "normal" or even low cholesterol levels, the American Heart Association spin doctors have found a new enemy of the cardiovascular system—it's not cholesterol after all, but inflammation (*Associated Press* 8/5/2002). Replacing "the standard theory through the modern era of cardiology," low-grade inflammation is said to cause plaque embedded in the arteries to loosen, thereby triggering fatal blood clots. "The implications of this are enormous," says Dr. Paul Ridker of Boston's Brigham and Women's Hospital. "It means we have an entirely other way of treating, targeting and preventing heart disease that was essentially missed because of our focus solely on cholesterol." The new way of treating heart disease will consist of blood tests to measure a substance called C-reactive protein, which is a marker for inflammation occurring anywhere in the body (not just in the arteries). But if you think that the new way of treating heart disease will include abandoning the widespread use of statins, those cholesterol-lowering drugs that have so many dreadful side effects, think again. "Many people ordinarily considered at low risk will probably be put on statin drugs, which lower inflammation as well as cholesterol." Thus, by declaring a new enemy, the medical profession can put just about everyone on statins, which magically not only lower cholesterol but also have a slight effect on inflammation. And because animal fats contain arachidonic acid, a substance falsely accused of causing inflammation, doctors can continue to recommend avoidance of saturated fats. Never mind that saturated animal fats provide vitamins A and D, nutrients the body uses to prevent inflammation.

HEART FAILURE

In 17 years of practice in Tyler, Texas, Dr. Peter H. Langsjoen has seen a "frightening increase in heart failure secondary to statin usage." Says Langsjoen: "Over the past five years, statins have become more potent, are being prescribed in higher doses and are being used with reckless abandon in the elderly and in patients with 'normal' cholesterol levels. We are in the midst of a CHF epidemic in the US with a dramatic increase over the past decade. Are we causing this epidemic through our zealous use of statins? In large part I think the answer is yes." Langsjoen has compiled a review of studies showing that statins interfere with Co-enzyme Q_{10} (CoQ_{10}), which is essential for muscle function—and the heart is a muscle. This phenomenon is well known to the drug companies because Merck & Co has two unused 1990 patents combining CoQ_{10} with statins to prevent CoQ_{10} depletion and its attendant side effects. Statins have created a life-threatening nutrient deficiency in millions of otherwise healthy people while the drug companies have sat back "with arrogance and horrific irresponsibility and watched to see what happens. As I see two to three new statin cardiomyopathies per week in my practice, I cannot help but view my once great profession with a mixture of sorrow and contempt." Langsjoen has piloted a citizen petition to the FDA calling for a black box warning on the statin package insert information. Langsjoen and colleagues "do not expect any response from the FDA, but ten years from now when the full extent of statin toxicity becomes painfully evident, at least we can, in good conscience, know that we tried and who knows, sometimes small sparks may spread in dry grass (www.redflagsweekly.com/features/2002_july08.html)."

Caustic Commentary

WINTER 2002

COPPER AND HEART DISEASE

While public health officials continue to promote lowfat diets and cholesterol-lowering statin drugs as the solution to heart disease, many other good theories go ignored. One of these theories has to do with copper deficiency. A researcher named Leslie M. Klevay has shown that copper deficiency leads to atherosclerosis in many animals (*J Am Coll Nutr* 1998;17(4):322-326). Copper is needed for a number of important biochemical reactions. The polymerizing enzyme lysyl oxidase (LOX) is copper dependent. This enzyme helps form the internal elastic lamina (IEL), a thin elastic layer in the arteries which is separated by only one endothelial cell layer from the blood. Without adequate copper, the lamina is not sufficiently elastic and intimal thickening results—and a recent theory of heart disease has to do with abnormal thickening of the arteries, followed by inflammation and the release of blood clots. Copper is also necessary for the formation of thyroid hormones and the production of heme iron in blood cells. Both milk and meat are deficient in copper, and the small amount of copper in most plant foods is difficult to absorb. (Legumes and whole grains that have not been properly prepared can actually block the absorption of copper.) The only reliable source of copper is liver, especially that of lamb and other ruminants. There was thus a very good reason for people to eat liver once a week, something our government now tells us not to do. . . in order to avoid heart disease.

TRANS FATS AND INFLAMMATION

The latest establishment theory on heart disease posits low-grade inflammation in the arteries as a cause, leading to the release of blood clots followed by heart attack. A new study directly fingers *trans* fats from stick margarine as a cause of inflammation. A recent study showed that consumption of stick margarine in human subjects provokes an increase in the production of inflammatory prostaglandins associated with atherosclerosis. Neither liquid soy oil nor butter had the inflammatory effect (*J Lipid Res* 2002 Mar;43(3):445-52). This research was carried out at the USDA Human Nutrition Research Center on Aging at Tufts University. Jean Mayer and Alice Lichtenstein of Tufts have been major spokespersons for avoiding foods containing those "evil" saturated fats. We expect that reports on this study will include the suggestion to consume toxic liquid vegetable oils and low-*trans* soft spreads instead of stick margarine, without any suggestion that we should go back to butter.

FALL 2003

AND IF IT TASTES GOOD, YOU MUSN'T EAT IT!

A new reason for not eating delicious, satisfying foods like cheese, meat and chocolate, says soy-promoting Neal Barnard, MD, of the Physicians Committee for Responsible Medicine, is that these foods create opiates in the brain and make you feel good. "There's a reason why people call these things 'comfort foods,'" says Barnard. "They're getting an opiate when they eat them." New research indicates that many traditional high-fat foods stimulate the production of dopamine, a brain chemical associated with intense good feelings. Naturally, the food puritans are not pleased. Surely Mother Nature did not mean for us to enjoy our food! Someone must be punished for foisting comfort foods on the public and since we can't sue Mother Nature, Barnard suggests we sue the fast food chains who've gotten the public "suckered into high-fat meals—like cheeseburgers and shakes. . ." (*Washington Times*, June 15, 2003). The food chains need to be sued, all right, not for the natural foods they serve, but for using imitation foods, particularly partially hydrogenated vegetable oil, which doesn't tickle our pleasure centers in quite the same way and makes us eat and eat and eat in a desperate attempt to get into the comfort zone.

THE FEAR FACTOR

When the guilt trip doesn't work, the food industry turns to the other potent weapon in their arsenal: fear. A good example can be found in the recent headline, "Women Who Eat High-Fat Foods Could Be Doubling Their Risk of Breast Cancer, Scientists Say." This and similar pronouncements heralded a new study published in International Journal of Cancer: "Eating high-fat red meats and dairy products such as cream [one of those comfort-zone foods] may increase the risk of breast cancer in premenopausal women," says nutrition researcher Eunyoung Cho of the new study. "I would not recommend that [Atkins] diet for premenopausal women unless they replace red meat with poultry and fish.. . . Breast cancer risk increases 58 percent by eating animal fat." What the study really showed was that if your diet contains 14 percent of calories as animal fat, your chances of getting breast cancer are 0.68 percent; if your diet contains 18-21 percent of calories as animal fat,

your chances of getting breast cancer increase to 0.88 percent; and if your diet contains more than 21 percent animal fat, your chances of getting breast cancer actually go down to 0.73 percent. Spokesmen for the study used every trick in the book to make these trivial results seem scary. In addition to the incredible hype over minor differences, they divided the subjects into unequal quintiles (the highest quintile of 21-46 percent had the greatest range); determined fat percentages by dietary recall that was surveyed only two times during the study; neglected to mention the fact that there were twice as many smokers in the group with highest animal-fat consumption compared to lowest; and failed to report on many studies showing that animal fats have no effect on breast cancer rates (*Int J Cancer* 2003 Mar;104(2):221-7).

NO DIFFERENCE

A new TV ad in Canada advises viewers to "Ask your doctor about the Heart Protection Study from Oxford University." This was a large study which showed a small but statistically significant relationship between treatment with statin drugs and lowered rates of heart disease—as one commentator put it, take a massive group and follow them long enough and something statistically significant will come out. But what the ad doesn't tell you is that there are two recent studies, both of large groups, where treatment with expensive statin drugs made no difference in outcome. In the ALLHAT study, deaths in the second largest cholesterol-lowering trial ever were equal in both the treatment and control groups. In the ASCOT study, just published in *The Lancet*, those taking Lipitor fared only slightly better than those taking a dummy pill. Neither study made mention of the side effects experienced by those on cholesterol-lowering drugs, including neuropathy, muscle wasting leading to crippling back pain, heart failure, liver failure, cancer, weakness, fatigue, depression and memory loss. Instead, the industry is claiming that statin drugs can help patients reduce anxiety, depression and feelings of hostility! (*The Record* 8/11/2003). Now throw in "the promise" that statins will protect against Alzheimer's, multiple sclerosis and osteoporosis (*Newsweek* August 14, 2003) and you've come up with a scheme aimed at putting the entire population on expensive drugs that have subtle but serious side effects. Fortunately, not all of the people are fooled all of the time and statin sales have not lived up to expectations. A recent article in the *Wall Street Journal* carried the title: "The Statin Dilemma: How Sluggish Sales Hurt Merck" (August 25, 2003).

MAGIC BULLET

We've heard fantastic claims about various nostrums, but the hype surrounding a new remedy called the "polypill" takes the cake. Proposed not by crackpots but by two distinguished scientists, Nicholas Wald, Professor and Head of Wolfson Institute of Preventive Medicine, and Malcolm Law, a Professor at the University of London and University of Auckland in New Zealand, and promoted by none other than the prestigious *British Medical Journal* (and also hyped in the tabloids), the polypill will contain six different ingredients: a statin to lower LDL-cholesterol, three (yes three) blood pressure drugs (a beta blocker, a diuretic and an ACE inhibitor), aspirin to reduce clotting tendencies and folic acid thrown in to prevent high homocysteine levels. Richard Smith, editor of the *British Medical Journal*, claims that the issue introducing the polypill is possibly the most important issue of the journal in the last 50 years. He urges readers to save their copy since it would likely become a collector's item because of the Wald and Law contributions. Wald and Law claim that the polypill will have "a greater impact on the prevention of disease in the western world than any other known intervention." There have been absolutely no studies on the proposed panacea but the inventors insist that they can prevent almost nine out of ten heart attacks and four out of five strokes in anyone with cardiovascular disease and everyone age 55 or older. Claims for the efficacy and safety of the polypill are based solely on meta-analyses and statistical analyses of clinical trials. This magic bullet will have very few side effects, say the promoters, because lower-than-normal dosages will be used. For those who believe all this, we have a bridge for you.

WINTER 2003

TARGETING CHILDREN

Here's a trend we all could have predicted—children and teenagers are now the targets of cholesterol-lowering diets and drugs. Worried parents are taking their healthy children to pediatric cardiologists who duly put them on diets that deny them eggs, butter, whole milk and meat, while prescribing cholesterol-lowering margarines like Take Control and Benecol, soy foods and "high-fiber" foods like oat bran, oatmeal, beans, barley and fruit. If dietary changes don't bring cholesterol levels down, the children get medications—resin powders for

children and statins, including Lipitor, for adolescents. Some doctors have objected, but Peter Kwiterovich, director of the lipid center at Johns Hopkins Children's Center in Baltimore says ignoring high cholesterol in children is taking a chance with their hearts later in life. "I think every child should have their cholesterol measured and be assessed for obesity, at a minimum, and then appropriate interventions come into play." Marc Jacobson, head of the center for atherosclerosis prevention at Schneider Children's Hospital in New York and a member of the American Academy of Pediatric's nutrition committee agrees. Children, he says "definitely should be screened, and they definitely should be treated if found to be at high risk" (*Washington Post*, December 2, 2003). No one has described the results of cholesterol-screening in children better than Dr. Uffe Ravnskov: "At best, emphasis on lowering cholesterol in children will create families of unhappy hypochondriacs, obsessed with their diet and blood chemistry. At worst, it will have profound and unfortunate effects on the growth of children . . ."

CHOLESTEROL AND MEMORY

Cholesterol-lowering measures may also have profound and unfortunate effects on their minds. A recent study found that increasing levels of LDL- and total cholesterol are associated with beneficial effects on memory in middle-aged women (*J Neurol Neurosurg Psychiatry* 2003;74:1530-1535). The researchers warned: "Possible cognitive effects of cholesterol reduction should be considered in future studies of lipid-lowering agents." But the prescription-happy pediatric cardiologists aren't exercising the same caution, blithely ratcheting down cholesterol levels in children with no thought as to how such measures will affect their neurological development or their ability to reach adulthood with all their mental facilities intact.

SUMMER 2004

ANOTHER PARADOX

Good science requires that theories conform to the evidence; if evidence that contradicts a theory emerges—even a single piece of evidence—then scientists are obliged to come up with a different theory. But in the case of the lipid hypothesis for heart disease, contradictory evidence is given the status of "paradox." The theory is never abandoned, just promoted with more vigor. The latest "paradox" emerges from a study carried out in Japan. Researchers followed 3731 Japanese men and women aged 35 to 89 years from 1984 to 2001. Food intakes were determined from a 24-hour food diary at the beginning of the study. During the following 15 years, 60 deaths from cerebral infarction (stroke) occurred. A high intake of animal fat and cholesterol was significantly associated with a reduced risk of death from stroke (*Stroke* 2004;10:1161-01).

STATINS FOR MS?

With a view to expanding the market for one of the world's most profitable classes of drugs—statin drugs for cholesterol lowering—scientists are now promoting them as a treatment for multiple sclerosis. In a recent clinical trial, MRI tests showed a decrease in the number and volume of new lesions in MS patients treated with statins (*Lancet Neurol* 2004 Jun;3(6):369-71). However, a member of the THINCS group (The International Network of Cholesterol Skeptics) reports that a colleague involved in multiple sclerosis research found little correlation between the severity of lesions as measured by MRI scans and neurological function in MS. The Lancet report makes no mention of this fact.

FALL 2004

STATIN MADNESS. . .

In 2003, sales of statins totaled almost $14 billion, up 10.9 percent from 2002. Growth of this magnitude can only be achieved by rapidly expanding the customer base. First proposed for men deemed "at risk" for heart disease by virtue of "high" cholesterol levels, doctors now recommend statins for both men and women of all ages, especially targeting diabetics and sufferers of rheumatoid arthritis. The literature even promotes statin use as a cancer prevention measure. The cholesterol juggernaut is not daunted by cautionary studies, such as a review appearing in the May 12, 2004 issue of the *Journal of the American Medical Association*. The authors looked at studies going back almost 30 years and concluded that statin drugs do not provide any benefit to women who do not have already existing heart disease. More healthy Americans joined the ranks of patients in July with new recommendations to lower LDL-cholesterol (the so-called "bad" cholesterol) to less than 100, 30 points lower than previously recommended. The authors of the recommendations, which were published in the journal *Circulation* and endorsed by the National Heart Lung and Blood Institute, the American Heart

Association and the American College of Cardiology, have made a living promoting pharmaceuticals, with most receiving honoraria from all the major drug producers, including Merck, Pfizer, Parke-Davis, AstraZeneca, Abbott, Dupont, Sankyo, Bayer and Bristol-Myers Squibb. The challenge for the statin makers is to convince everyone "qualified" to actually take the drugs—only about half of them do. One proposal calls for making statins available as an over-the-counter drug (already an option in the UK). Another, presented at a UK medical meeting by Dr. John Reckless (this is his real name!), calls for adding statins to tap water—like fluoride. (Actually some of the bestselling statins—Lipitor, Baycol, Crestor and Lescol—contain a fluoride compound.) "It would be a great way of protecting people from heart disease before it even starts," says Dr. Reckless. What Reckless fails to mention is that statins pose a massive risk of severe, horrible birth defects if taken by pregnant women, defects more horrible than those caused by thalidomide. The list of statin-induced defects includes holoprosencephaly (defective septum separating lateral cerebral ventricles with cerebral dysfunction), atrial septal defect, aortic hypoplasia, neural-tube defects, duplication of spinal cord, spina bifida, left renal dysplasia, disorganized lumbosacral vertebra and deformities in the limbs. "We seem to be sleepwalking into what could be a major medical disaster," writes Dr. Malcolm Kendrick. "Most people, and most doctors, are unaware—or don't seem to care—that statins should never ever be taken by women of childbearing age. . . . Yet, when statins are available OTC it is absolutely certain that women of childbearing age will take them, knowing nothing of this risk. It is equally certain that a number of these women will become pregnant, and many of these pregnancies will result in horribly deformed children" (*redflagsdaily.com*, 6/18/2004).

. . . AND THE DIET TO GO WITH IT

Not content to make you depressed, weak, achy and forgetful with statins, the medical profession recommends a lowfat diet of processed foods so you'll feel even worse. A WAPF member recently diagnosed with "high" cholesterol shared with us the handouts his doctor gave him and it's the same old, same old—margarine instead of butter, skim milk, nondairy creamer, lowfat milk and cheese, lean meat, skinless chicken breasts, egg substitutes, liquid vegetables oils and lowfat baked goods. No bacon, liver, sausage, cream, full-fat cheeses or coconut but high-sugar items like sherbert, angel food cake, lowfat jelly beans and hard candy are OK. In an editorial, Dean Ornish, dean of the ultra-lowfat diet, even argues that Medicare should reimburse dieticians who counsel heart patients on how to follow this spartan regime (*Washington Post*, August 8, 2004). Invoking "powerful benefits" including "sustained weight loss, improved sexual function, increased energy, decreased blood pressure, dramatic reductions in angina and better control of diabetes," Ornish promises that a diet of ersatz, tasteless food will increase your "joy of living," providing far more motivation than the "fear of dying." Here's what we'd like to know: Even if such a diet were effective (which it is not), how many measley days would such soul-numbing measures add to the human carcass?

WINTER 2004

ANOTHER PARADOX

We have often described how proponents of the cholesterol theory of heart disease deal with contradictory evidence—not by chucking the whole thing in the garbage where it belongs but by assigning it to the category of paradox. An "American paradox" has emerged from a study published in the *American Journal of Clinical Nutrition* (2004;80:1175-84). Using coronary angiography, researchers looked at the progression of buildup in the arteries in 235 postmenopausal women with established coronary heart disease. They found that a greater saturated fat intake was associated with *less* progression of coronary atherosclerosis, whereas carbohydrate intake was associated with *more* progression. But don't look for these startling findings to be reflected in government dietary policy anytime soon; an editorial in the same issue explains away the politically incorrect test results with all sorts of statistical mumbo jumbo.

MORE STATIN PROBLEMS

A new look at the effects of statin drugs on cognitive function should give pause to anyone thinking of taking them. Researchers tested the learning ability of patients taking a low dose of simvastatin compared to controls (*Am J Med* 2004 Dec 1;117(11):823-9). Using tests called Elithorn Mazes, researchers looked at the level of improvement when patients take the test several times. The time for solving a puzzle of some kind was improved by 16 percent in the control group whereas the statin group showed no improvement. The difference, together

with the differences in a few other tests, was highly significant, meaning that a considerable number of statin-takers were unable to learn anything from the first test. The researchers tried to explain away the results by stating that "The observed treatment effects were quantitatively small and were primarily manifest not as an absolute decline in performance but as a failure to improve upon repeat posttreatment testing." In a THINCS group report, Dr. Uffe Ravnskov describes the results somewhat differently: "Consider that this result was achieved in a study comparing only 189 statin-treated patients with 94 controls after only six months and on the lowest simvastatin dose used in clinical practise. Translated to the US population, it means that millions of people may have become unable to learn from previous experiences due to their cholesterol-lowering treatment."

SPRING 2005

OUR FRIEND CHOLESTEROL

If your doctor is pressuring you to take drugs or stop eating butter in order to lower your cholesterol, be sure to tell him or her about the study that appeared in the February 2005 *Journal of the American Geriatrics Society* (Vol 53, pages 219-226). Researchers evaluated 2277 senior Americans, aged 65 to 98, 21 percent of whom were taking cholesterol-lowering drugs. Over a period of three years, lower total cholesterol and lower LDL-cholesterol (the so-called "bad" cholesterol) were associated with a greater risk of dying. Use of cholesterol-lowering drugs seemed to lower this association, but did not abolish the elevated risk of death. This study confirms a similar finding from the Honolulu Heart Program, where those who had low levels of cholesterol over 20 years had a higher risk of dying from all causes (*Lancet* 2001;358:351-55). And if your pediatrician is pressuring you to lower your child's cholesterol by denying traditional foods like eggs, butter and whole milk, be sure to tell him or her about a study published April 1, 2005, in the *American Journal of Epidemiology* (Vol 161, No 1, pages 691-699). Investigators looked at cholesterol levels and psychosocial development in 4,852 children, ages 6 to 16 years. Non-African-American children with low cholesterol (less than 145 mg/dl) were almost three times more likely to have been suspended or expelled from schools than those who had higher cholesterol levels. The authors concluded that low total cholesterol "may be a risk factor for aggression."

DEATH BY MARGARINE

In Holland, people with "high" cholesterol or one of the 588 other risk factors for heart disease get a prescription for a cholesterol-lowering drug and advice to buy Unilever's Becel Pro Aktiv, a margarine containing cholesterol-lowering plant sterols. Nurses offer cholesterol tests in the supermarket next to the margarine shelves while the Dutch heart association promotes Becel with scaremongering TV commercials. This is a fairytale deal between Uniliver, the Dutch heart association and Dutch health insurers (who pay for the margarine!), one that could well happen in the US. In a letter to Dr. Uffe Ravnskov's THINCS group, W. M. Nimal Ratnayake, PhD, of Health Canada explains just why plant sterols are so dangerous. Stroke-prone rats fed sterols hyperabsorb these compounds leading to increased rigidity of red blood cells and drastically reduced life span. Humans prone to hemorrhagic stroke have similar abnormalities in the red blood cells (*Clin Exp Hypertension* 1980;2:1009-1021). Furthermore, hemorrhagic stroke occurs at higher rates in persons with low levels of cholesterol (Irbarren, *JAMA*, 1995).

NOVEL ROLE

Scientists have discovered a novel role for cholesterol, one that explains why low cholesterol is linked to cancer and many other diseases. Cholesterol in cell membranes appears to anchor a signaling pathway linked to cell division and cancer. "Cell signals have to be tightly controlled," says Dr. Richard GW Anderson, chairman of cell biology at UT Southwestern Medical Center and head of the study. "If the signaling machines do not work, which can happen when the cell doesn't have enough cholesterol, the cell gets the wrong information, and disease results." Every cell in our body is surrounded by a membrane composed of fatty acids and containing cholesterol. The cholesterol-containing regions of the cell membrane are more rigid than the other areas and play a critical role in organizing signaling machinery at the cell surface. The correct arrangement of signaling modules in these domains is vital for communication inside the cell and is dependent on proper levels of cholesterol (*Science*, March 4, 2005).

HYPED RESULTS

In yet another example of hyped results, researchers have announced that "intensive lipid lowering [with a statin drug] beyond currently recommended levels provides significant

additional clinical benefits in patients with coronary heart disease." Citing the results of the Treating to New Targets (TNT) trial, Dr. John LaRosa, a tireless proponent of getting everybody's cholesterol as low as possible, made the announcement at the American Cardiology's annual scientific session, held in Orlando, Florida, March 2005. Dr. Eric Topol, who runs theheart.org, which is funded by AstraZeneca, a maker of cholesterol-lowering drugs, was even more emphatic: "There isn't any question left at this point that we should be more aggressive." However, a cold look at the study results reveals nothing to crow about. Researchers followed 10,000 patients who were given either a low or high dose of the popular cholesterol-lowering drug Lipitor. Total mortality was identical in the two groups—5.6 percent in the low-dose group and 5.7 percent in the high-dose group. The high-dose group had slightly lower mortality from coronary heart disease but higher mortality from other causes (*N Engl J Med* 2004;350:1495-504). LaRosa dismissed the higher levels of noncardiovascular mortality in the high-dose group—as well as several cases of reported side effects—as an artifact due to chance and suggested altering the current cholesterol recommendation to one that calls for even more aggressive lipid lowering. One independent commentator has suggested that TNT refers to Twisting Natural Truths!

SUMMER 2005

DEMENTED THEORY

Researchers are scratching their collective heads over recent findings that cast doubt on the widespread use of cholesterol-lowering drugs. The first was published in the May 24 issue of the journal *Neurology*. Scientists in Sweden analyzed data from 392 men and women in Goteborg, Sweden over an 18-year period. They found that high total cholesterol at ages 70, 75 and 79 was associated with a reduced risk of dementia between ages 79 and 88. What this means is that we need to keep our cholesterol levels high if we want to have keen minds well into old age. But scientists wedded to the cholesterol theory dare not make so bold a statement. Instead, they weasel-word. "These findings raise more questions than they give answers," says Michelle M. Mielke of the Center on Aging and Health at Johns Hopkins Bloomberg School of Public Health and one of the study authors. "Therefore, we strongly urge that consumers not make changes in their diet or medication without consulting with their doctors first." Rachel Whitmer, a research scientist specializing in cognitive aging at Kaiser Permanente Northern California also specializes in saying nothing with a lot of words: "Lingering questions were not put to rest, but new exciting ones are raised. . . . This study is another example of the importance of timing in terms of when one measures a risk factor, and the need to consider risk factors for dementia over the entire life course." A second study, which was a follow-up of the Framingham Heart Study and published in *Psychosomatic Medicine* (2005;67:24-30), found that lower naturally occurring total cholesterol levels are associated with poorer performance on cognitive measures such as abstract reasoning, attention/concentration, word fluency and executive functioning. Once again, double talk was necessary: ". . . competing risks must always be taken into consideration," said the researchers. "Lower cholesterol values may have modestly detrimental effects on cognitive function for the individual but, depending on the patient's risk profile, may have beneficial effects with respect to cardiovascular morbidity and mortality." Rather than risk dementia in the elderly (and not so elderly) by force-feeding statin drugs, the medical profession needs to admit that the whole theory is demented.

FALL 2006

RECIPE FOR DISASTER

The American Heart Association and the American Academy of Pediatrics have ganged up to target children with a starvation diet guaranteed to saddle them with health and behavioral problems as they enter adulthood (Reuters Health 9/28/2005). Clothed in platitudes—"breast feed through the first year," "skip calorie-packed, low-nutrient foods, "delay introducing juice until at least 6 months of age"—the new guidelines dictate withholding foods that growing children need most, namely animal fats and salt. Parents are advised to feed them lean meats, skinless chicken, "low-mercury" fish and fat-free milk. In this scheme, children don't even get the small amount of fat in lowfat milk—it must be fat free! And they don't get butter either, but vegetable oils and soft margarine. Plenty of whole grains (including extruded whole grain breakfast cereals) mean lots of stress on the developing intestinal tract and salt restriction guarantees suboptimal intellectual development. The phrase that comes to mind as one contemplates the consequences of this appalling advice is "wailing and gnashing of teeth."

Caustic Commentary

CLASS ACTION

Consumers have filed the first-of-its-kind, nationwide class action lawsuit against Pfizer, maker of the popular cholesterol-lowering, statin drug Lipitor. The lawsuit alleges that Pfizer engaged in a massive campaign to convince both doctors and patients that Lipitor is a beneficial treatment for nearly everyone with elevated cholesterol, even though no studies have shown it to be effective for those over 65, and for women at any age who do not already have heart disease or diabetes. In fact, the ASCOT study, the largest clinical trial on the effectiveness of statin therapy in women, found that women at increased risk of developing heart disease who took Lipitor developed 10 percent more heart attacks than the women who took the placebo. The proposed class action seeks to represent women who have taken Lipitor and who have no history of heart disease or diabetes; people aged 65 and over who have taken Lipitor and who have no history of heart disease or diabetes; and third-party payers such as insurance companies, union health and welfare funds, self-insured employers and others who paid for Lipitor for patients in either of these two groups. The law suit was filed in US District Court in Boston by Steve Berman, managing partner of Hagens Berman Sobol Shapiro on behalf of several individuals, Health Care of All and the Teamsters. For further information see www.hbsslaw.com.

WINTER 2005-SPRING 2006

CRAZY LOGIC

The slide into madness that started with the anti-saturated-fat agenda reached its lowest point in December when the Illinois State Board of Education proposed rules that would ban whole milk from school lunches (*Associated Press*, December 10, 2005). Under the new rules, cartons of whole milk, which have a high fat content, would be considered junk food, but baked Cheetos and one-ounce bags of baked potato chips would not. Whole milk flunks three of the major guidelines now used to assess whether a food is healthy or not: calories from fat exceeding 35 percent (except for nuts and seeds), calories from saturated fat exceeding 10 percent and total calories exceeding 200 for an individual package. Of course, whole milk could be packaged in tiny cartons, like the baked potato chips, but the crazy logic that allows junk food in small packages does not seem to apply to real foods like whole milk. Besides, the dairy industry makes more profit on skim milk (because they can sell the butterfat separately in high-value foods like ice cream) and sugar-laden chocolate milk—which school children are now consuming by the gallon.

NOT IDEAL

Now that the cholesterol-lowering drugs called statins have become the treatment of choice for heart disease, scientists are looking at just how much they can lower levels of LDL-cholesterol (the so-called "bad" cholesterol) without actually killing the patient with the treatment. The Treating to New Targets (TNT) Study, published in 2004, found that high doses of statins improved cardiovascular disease outcomes slightly but resulted in higher numbers of deaths from other causes. In the Incremental Decrease in End Points Through Aggressive Lipid Lowering (IDEAL) study, published in the November 16, 2005 issue of the *Journal of the American Medical Association* (JAMA), researchers did not even find a benefit for cardiovascular disease from aggressive cholesterol lowering, although they were able to tease "reduced risk when certain secondary outcomes (composite end points of any coronary heart disease event)" from the data, In an editorial on the IDEAL trial, published in the same issue of *JAMA*, Dr. Christopher Cannon repeats the dogma that for LDL-cholesterol, "lower is better for preventing MI stroke, need for cardiac procedures and death," but hints at problems with the study when he calls for careful monitoring of "adverse effects" and even pursuit of "new avenues of treatment." That's because total mortality was higher in the high-dose statin group and, of even more concern, almost all of the participants reported some kind of side effect from the treatment—with almost half of the participants in each group suffering a serious adverse effect. Dr. Uffe Ravnskov points out other flaws in the study: only 20 percent of the study group was female in order to conceal the bad effects of statins on women; fully 79 percent of the subjects took aspirin, a serious confounder; the authors used the criteria of relative risk to exaggerate any marginal benefits; and, finally, the authors did not address current research indicating that low LDL-cholesterol is actually a risk factor for heart disease and that LDL lowering can be detrimental. It all points to the fact that cholesterol lowering as a treatment for heart disease is less than ideal.

Caustic Commentary

SUMMER 2006

LIPITOR LOWER

Pfizer will have trouble meeting its marketing objectives this year as sales of its popular cholesterol-lowering drug Lipitor have fallen "significantly short of expectation." Pfizer had hoped to increase Lipitor sales by 7 percent in 2006; instead sales declined 3 percent in the first quarter. Financial analysts blame competition from other cholesterol-lowering drugs and generic versions of these statins now coming on the market. According to Hank McKinnel, chairman and chief executive of Pfizer, the company is counting on "powerful clinical data and new educational campaigns on [Lipitor's] health benefits" (*Financial Times*, April 20, 2006). In other words, expect to see more phony science and heavy advertising to promote this dangerous and unnecessary drug, even to groups for whom clinical data has shown no benefit whatsoever—women, the elderly. . . and children.

STARTING WITH THE YOUNG

Yes, children are now a target of lipid-lowering campaigns. A study published in the *New England Journal of Medicine* (March 23, 2006), which found fewer "coronary events" in young blacks genetically predisposed to have lower LDL-cholesterol levels, has led to renewed calls for cholesterol lowering in young people. "The new findings suggest the need to redouble our efforts to reduce LDL-cholesterol levels in younger persons by promoting healthy diets and reducing obesity," wrote Alan R. Tall of Columbia University Medical Center. "Even small successes will probably be leveraged for later gains in lowering the risk of cardiovascular disease." Dr. Scott Grundy, an unabashed apologist for the lipid hypothesis, went further: In addition to restricting cholesterol and saturated fat, he argues that "[i]n some people it may be necessary to add drugs to reduce cholesterol levels." These lowfat and statin proponents seem oblivious to research showing the downside of low cholesterol levels in young people. For example, a study published in the *American Journal of Epidemiology* (161(7):691-99, 2005) found that non-African-American children with cholesterol concentrations below the 25th percentile were nearly three times as likely to have been suspended or expelled from school as those with total cholesterol levels at or above the 25th percentile. Among many roles in the body chemistry, cholesterol is necessary for neurological development, for the proper function of serotonin and other "feel-good" chemicals, and for the production of sex and stress hormones.

NIGHTMARES

Falling sales may be giving Pfizer executives nightmares because cholesterol-lowering drugs are giving nightmares to the people taking them. A recent report published in the *British Journal of Medicine* (April 21, 2006) describes a 72-year-old woman who experienced extreme nightmares after beginning "treatment" for "hypercholesterolemia" with Lipitor. When she discontinued the drug the nightmares ceased, and when she agreed to a rechallenge with Lipitor, the nightmares occurred again. The problem was solved by going off Lipitor for good. The author of the report speculates that the nightmares could be a direct effect of the statin on the central nervous system and notes previous reports of nightmares associated with other cholesterol-lowering drugs.

HEART FAILURE

More bad news for Pfizer includes a doubling of heart failure rates since statins were introduced (*Circulation*, February 6, 2006). A new study of older men and women shows that higher LDL-cholesterol levels are associated with decreasing mortality risk in women. For both men and women, the risk of fatal heart failure decreases with higher LDL-cholesterol levels (*Journal of the American Geriatric Society*, December 2005).

FALL 2006

STATIN WOES

Go to http://www.askapatient.com and click on ratings and then Lipitor, where you will find almost 700 comments on the cholesterol-lowering drug. What is interesting is the very high number of patients reporting side effects, including severe fatigue, joint pain, digestive problems, craving for fatty foods, difficulty breathing, thinning hair, depression, lack of concentration, memory lapses, thoughts of suicide, nightmares, peripheral neuropathy, paralysis, dizziness, painful charley horses, weight gain, blurred vision, headaches, insomnia, difficulty walking, rashes, blisters, slurred speech, eczema and "itching all over." Yet most of the ratings are positive, with patients expressing satisfaction at bringing their cholesterol levels down, and persevering in spite of the debilitating side effects. Such is the level of cholesterol anxiety engendered by

the phony lipid hypothesis. Perhaps "complete decline in the power of reason" should be added to the list of side effects from cholesterol-lowering drugs.

WINTER 2006

LACK OF EVIDENCE

Recent national recommendations suggest that physicians should use drugs to achieve LDL-cholesterol levels of less than 70 for patients at "very high cardiovascular risk" and less than 100 for patients at "high cardiovascular risk." In a recent review of all controlled trials, cohort studies and case-control studies that examined the independent relationship between LDL-cholesterol and major cardiovascular outcomes in patients with LDL levels less than 130, researchers were surprised to find no evidence to suggest "that the degree to which LDL-cholesterol responds to a statin independently predicts the degree of cardiovascular risk reduction" (*Annals of Internal Medicine*, October 3, 2006). In other words, using statin drugs to get your LDL-cholesterol as low as possible does not reduce your risk of heart disease. But rather than question the whole business of cholesterol-lowering for lack of evidence, the research team concluded that "there are no intrinsic barriers to producing such evidence." The strategy of lowering LDL-cholesterol by drugs is not a bad one, they say, only the studies that fail to support such a strategy are bad. Studies that eliminate conflicting variables and research bias might provide "valid evidence," they claim, but in the meantime, treatment with statins should continue.

MORE LACK OF EVIDENCE

Another report published this year describes a Finnish study in which researchers enrolled 400 home-dwelling people between the ages of 75 and 90 years who suffered from cardiovascular disease. The patients were randomly assigned to receive either usual care from their primary care physician or specialized care based on "current evidence-based European guidelines for chronic CVD." Over an average of 3.4 years, the group receiving "specialized care" had significantly higher use of drugs to lower blood pressure and cholesterol levels. However, the incidence of heart attack, heart failure, stroke and cardiovascular death were similar between the two groups, and deaths due to any cause also occurred at similar rates (18 percent versus 17 percent). Nor did the time until a first cardiovascular event differ between the two groups (*American Heart Journal* 2006;152:585-592). So why bother with the expense and aggravation of "specialized" care? The evidence for the aggressive use of drugs in the elderly is just not there—yet the elderly remain prime targets for the pharmaceutical industry.

SPRING 2007

SPARCL FIZZLES

Researchers presented the results of the Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) with a lot of fanfare at the 15th European Stroke Conference in Brussels, Belgium, in May, 2006 (theheart.org). The study enrolled 4731 patients who had suffered a recent stroke and assigned them to receive a strong cholesterol-lowering statin drug or a placebo. LDL-cholesterol fell by 38 percent in the statin group compared to 7 percent in the placebo group. Those treated with statins showed reductions in fatal and ischemic stroke, but experienced a significant *increase* in hemorrhagic stroke. When it came to overall deaths, the SPARCL Trial really fizzled—216 deaths among those taking statins versus 210 in the placebo group. So taking statins after a stroke increases your chances of dying by 3 percent. . . after several years of suffering from the effects of drastic cholesterol lowering. But the study report makes no mention of side effects. Apparently the researchers didn't ask the participants how they felt. And then there are the costs to consider. Even defenders of using statins for stroke prevention note that based on SPARCL data, statin therapy costs $203,000 to prevent one stroke in five years (*Stroke*, online publication February 1, 2007).

SUMMER 2007

DEMENTIA AND CHOLESTEROL

Manufacturers of statins and their cohorts in the media are blithely promoting these cholesterol-lowering drugs to ward off Alzheimer's disease and dementia. A good example is a February 8, 2007 article appearing in the British paper *The Daily Mail*. The authors of "Diet high in cholesterol can trigger onset of Alzheimer's" warn about studies showing that "eating lots of foods containing saturated fats, such as butter and red meat, can boost levels of proteins in the brain linked to dementia," and that "large amounts of harmful cholesterol are found in foods high in saturated fats such as red meat, butter, cheese and offal such as liver and kidneys." These dire warnings are not based on studies of humans eating red

meat and butter—an online search for red meat or butter plus Alzheimer's yields nothing—but are based on research in which rats are given large amounts of purified cholesterol. The article cites "...growing evidence that taking cholesterol-lowering statins makes people less likely to develop Alzheimer's later in life." No reference is provided for this remarkable statement, remarkable given the many published reports of statin-induced cognitive decline. More sobering news comes from the Honolulu-Asia Aging study. Researchers followed over 1000 Japanese-American men over a 40-year period, starting in 1965. They found that cholesterol levels in men with dementia and, in particular, those with Alzheimer's, had declined at least 15 years before the diagnosis and remained lower than cholesterol levels in men without dementia throughout that period. Their conclusion: "A decline in serum total cholesterol levels may be associated with early stages in the development of dementia" (*Arch Neurol* 64:103, 2007).

CHINKS IN THE STATIN DIKE

"Trends in mortality from coronary heart disease have not effectively changed since statins were approved in the United States. . ." This damning statement appeared in the *International Journal of Cardiology*, February 21, 2007. The authors state categorically that the "beatification" of statins as miracle drugs is not justified. "Changes in lifestyle should be considered the cornerstone of cardiovascular prevention. . . Adherence to healthful lifestyle has been shown to be associated with reductions in the rates of coronary disease, diabetes in women and mortality in elderly. Patients with major lifestyle problems enrolled in recent statin trials were given only drugs, and no statin has ever been compared with a non-atherogenic lifestyle and shown to be superior or additive." The authors also note that studies on statin drugs minimize and under-report the side effects. Meanwhile in the Netherlands, a talk program called Radar has caused a furor in medical circles. The program zeroed in on statin side effects and included interviews with author Dr. Uffe Ravnskov and colleagues from The International Network of Cholesterol Skeptics (THINCS), who, on prime time TV, challenged the dogma that high cholesterol causes heart disease. According to the Dutch Cardiology Society, the program's assertions have caused "great unrest among patients." Wybren Jaarsma, chairman of the Society, writes that many colleagues have faced questions from patients over whether they should "continue care that has been scientifically shown to be effective and necessary. . . . You must continue taking prescribed cholesterol medication," he declares. Establishment physicians have refused invitations to debate the subject on air. Instead, Dutch doctors are calling for restrictions on television programs that they claim "deliberately use matters of patient safety to boost viewing figures." Such calls for censorship are a sure sign of a sinking ship.

FALL 2007

THE STATIN SHUFFLE

While the pill-pushers continue to promote cholesterol-lowering with a vengeance—a recent article published in the *American Heart Journal* (2006:785-92) announced that clinicians are "under-prescribing" statin drugs—evidence accumulates that the little pill taken by 12 million Americans (a number the pharmaceutical industry would like to triple) may be bad news for a lot of people in a lot of ways. One recent study found that statin treatment caused a deterioration of blood sugar control in diabetics (*Atheroscler Thromb* 2006 Apr;13(2):95-100). Another reports that statin-induced cholesterol lowering causes muscular damage even when the patient has no symptoms of pain or weakness (*J Pathol* 2006 210(1):94-102). Another found elevated risk of lymphoid malignancy with statin use among Japanese patients (*Cancer Sci* 2006;97:133-138). Yet another presents evidence that statins interfere with selenium pathways *(Lancet* 363:892-94, 2004). Very low cholesterol is associated with poor survival in heart failure patents (*American Journal of Cardiology*, September 2006), a finding the study author called "counter intuitive." Most serious is accumulating evidence that cholesterol-lowering is bad for our brains. One new study indicates that a decline in total cholesterol levels precedes the diagnosis of dementia by at least fifteen years (*Archives of Neurology* 2007;64:103-107). Evidence that low levels of LDL-cholesterol are associated with Parkinson's disease have become so strong that a team at the University of North Carolina is planning to explore the link with clinical trials involving thousands of subjects (*Reuters*, January 15, 2007). Cholesterol circulating in the bloodstream is unavailable to the brain—both LDL and HDL are too large to pass the blood-brain barrier, so cholesterol needed by the brain must be manufactured in the brain. Statins, however, do pass the barrier and enter the brain where they can interfere with cholesterol production and set the scene for cognitive decline.

What Causes Heart Disease?

By Sally Fallon and Mary G. Enig, PhD

For almost forty years, the lipid hypothesis or diet-heart idea has dominated medical thinking about heart disease. In broad outlines, this theory proposes that when we eat foods rich in saturated fat and cholesterol, cholesterol is then deposited in our arteries in the form of plaque or atheromas that cause blockages. If the blockages become severe, or if a clot forms that cannot get past the plaque, the heart is starved of blood and a heart attack occurs.

Many distinguished scientists have pointed to serious flaws in this theory, beginning with the fact that heart disease in America has increased during the period when consumption of saturated fat has declined. "The diet-heart idea," said the distinguished George Mann, "is the greatest scam in the history of medicine."And the chorus of dissidents continues to grow, even as this increasingly untenable theory has been applied to the whole population, starting with lowfat diets for growing children and mass medication with cholesterol-lowering drugs for adults.

But if it ain't cholesterol, what causes heart disease? We don't know enough to say for sure but we do have many clues; and although these clues present a complicated picture, it is not beyond the abilities of dedicated scientists to unravel them. Nor is the picture so complex that the consumer cannot make reasonable life-style adjustments to improve his chances.

WHAT IS HEART DISEASE?

Coronary Heart Disease (CHD) is not a single disease, but a complex of diseases of varied etiology. Some of the recognized causes of heart disease include damage to the heart muscle or valves due to a congenital defect; or to inflammation and damage associated with various viral, bacterial, fungal, rickettsial or parasitic diseases. Rheumatic fever or syphilis can lead to heart disease, as can genetic or autoimmune disorders in which cellular proteins in the heart muscle are deranged or which disrupt enzymes affecting cardiac function.

These factors probably contributed to most cases of heart disease recorded in the early part of the century, when rates of infectious diseases were much higher and antibiotics were not in use. Nevertheless, heart disease was relatively rare in 1900, accounting for approximately 8 percent of all deaths in the US.

But by 1950, CHD was the leading cause of mortality in the US, causing more than 30 percent of all deaths, and the figure has been climbing ever since. Today CHD accounts for about 45 percent of all deaths. The incidence rose most precipitously between 1920 and 1960. Since that time, *mortality* rates from CHD have declined somewhat. This means that victims of heart disease are living longer, due most likely to improved surgical techniques, the advent of angioplasty and the use of anti-clotting drugs given to heart attack victims. But the *morbidity* rates—the incidence of heart disease—continue to rise, although at a lower rate than before. Of greatest concern is the high rate of heart disease in American men between the ages of 45 to 65—during the period of greatest family and career responsibilities.

The interesting thing is that most cases of heart disease in the twentieth century are of a form that is new, namely heart attack or myocardial infarction—a massive blood clot leading to obstruction of a coronary artery and consequent death to the heart muscle. Myocardial infarction (MI) was almost nonexistent in 1910 and caused no more than 3,000 deaths per year in 1930. Dr. Dudley White, inventor of the electrocardiograph machine, stated the following during a 1956 American Heart Association televised fundraiser: "I began my practice as a cardiologist in 1921 and I never saw an MI patient until 1928." By 1960, there were at least 500,000 MI deaths per year in the US. Rates of stroke have also increased and the cause is similar—blockage in the large arteries supplying the brain with blood.

According to current theory, the factors that initiate a heart attack (or a stroke) are twofold. One is the pathological buildup of abnormal plaque, or atheromas, in the arteries, plaque that gradually hardens through calcification. Blockage most often occurs in the large arteries feeding the heart or the brain. This abnormal plaque or atherosclerosis should not be confused with the fatty streaks and thickening that is found in the arteries of both primitive and industrialized peoples throughout the world. This thickening is a protective mechanism that occurs in areas where the arteries branch or make a turn and therefore incur the greatest levels of pressure from the blood. Without this natural thickening, our arteries would weaken in these areas as we age, leading to aneurysms and ruptures. With normal thickening, the blood vessel usually widens to accommodate the change. But with atherosclerosis the vessel ultimately becomes more narrow so that even small blood clots may cause an obstruction.

The other half of the MI equation is the blood clot or thrombus that blocks blood flow to the heart or brain. Thus, any search for the causes of heart disease must consider complex factors in the blood that promote clotting at inappropriate times, that is, other than in response to bleeding from a rupture or wound. In fact, while a great deal of attention has been focused on the cause and solution to atherosclerosis, the role played by clotting factors in the blood has been relatively neglected. Yet a heart attack due to a clot can occur even in the absence of arterial blockages.

Inflammation may also cause blockages. In fact, a new view considers coronary artery disease to be an inflammatory process, characterized by cycles of irritation, injury, healing and reinjury inside the blood vessels. The inflammatory response is actually a defense mechanism that helps the body heal but when the inflammatory process goes awry, plaques may rupture, provoking clots that lead to heart attacks.

The health and integrity of the blood vessel walls is another factor that must be considered.

Heart disease was relatively rare in 1900, accounting for approximately 8 percent of all deaths in the US.

Aneurysms, the dilation and rupture of blood vessels due to weakness in the vessel walls, will naturally provoke a clotting response, not to mention the more immediate danger of rapid blood loss. In addition, biochemical imbalances in the smooth muscle cells may result in spasms that can be just as effective as a blood clot in cutting off blood flow to the heart.

Finally, arrythmias—abnormalities in the rhythm of the heart's pumping mechanism—can lead to interrupted blood flow, oxygen starvation of the heart muscle or complete shut down of the heart—the so-called cardiac arrest. Regulation of the nervous impulses that govern the heart depends on a large number of factors—from mineral status to the integrity of the myelin sheath.

KNOWN RISK FACTORS

There are dozens of risk factors for heart disease. Those cited most often by medical orthodoxy include high blood cholesterol, smoking, lack of exercise, stress and overweight. (Others rarely mentioned include baldness, short stature and hairy earlobes!) A high level of cholesterol in the blood is a mild risk factor for individuals with familial hyper-cholesterolemia (cholesterol levels chronically above 350 mg/dl) but for most of us, there is no greater risk of heart disease between cholesterol levels that are "high" (over 300 mg/dl) and those that are "low" (under 200 mg/dl).[1]

One factor of apparent importance is smoking, which has been associated in many studies with an increased risk of coronary mortality, even after correction for other risk factors. It is easy to speculate on the mechanism by which smoking causes heart disease. Exposure to fumes containing free radicals may promote the growth of atherosclerotic plaques. Perhaps chronic carbon

CHOLESTEROL - YOUR BODY'S BEST FRIEND

Cholesterol is the body's repair substance. Scar tissue contains high levels of cholesterol. When your arteries develop irritations or tears, cholesterol is there to do its job of patching up the damage.

Along with saturated fats, cholesterol in the cell membrane gives our cells necessary stiffness and stability. When the diet contains an excess of polyunsaturated fatty acids, these replace saturated fatty acids in the cell membrane so that the cell walls actually become flabby. When this happens, cholesterol from the blood is "driven" into the tissues to give them structural integrity. This is why serum cholesterol levels may go down temporarily when we replace saturated fats with polyunsaturated oils in the diet, even though the body's overall cholesterol levels actually go up.

Cholesterol acts as a precursor to vital corticosteroids, which regulate mineral metabolism and blood sugar levels. Corticosteroid hormones also help us deal with stress and protect the body against heart disease and cancer, Furthermore, the sex hormones, such as androgen, testosterone, estrogen and progesterone, are made from cholesterol. Cholesterol is also a precursor to vitamin D and to the bile salts. Bile is vital for digestion and assimilation of fats in the diet.

Recent research shows that cholesterol acts as an antioxidant. This is the likely explanation for the fact that cholesterol levels go up with age. As an antioxidant, cholesterol protects us against free radical damage that leads to heart disease and cancer.

Cholesterol is needed for proper function of serotonin receptors in the brain. Serotonin is the body's natural "feel-good" chemical. Low cholesterol levels have been linked to aggressive and violent behavior, depression and suicide.

Mother's milk is especially rich in cholesterol and contains a special enzyme that helps the baby utilize this nutrient. Babies and children need cholesterol-rich foods throughout their growing years to ensure proper development of the brain and nervous system.

Dietary cholesterol plays an important role in maintaining the health of the intestinal wall. This is why low-cholesterol vegetarian diets can lead to leaky gut syndrome and other intestinal disorders.

Men who have cholesterol levels over 300 mg/dl are at slightly greater risk for heart disease. For women, there is no greater risk for heart disease, even at levels as high as 1000 mg/dl. In fact, mortality is higher for women with low cholesterol than for women with high cholesterol.

Cholesterol readings are highly inaccurate. They vary with the time of day, time of the patient's last meal, levels of stress and the type of test used. Tests for HDL and LDL are especially subject to inaccuracies.

monoxide intoxication limits the heart's utilization of oxygen.

But smoking as a risk factor is more complex than simple cause and effect. In a multi-year British study involving several thousand men, half were asked to reduce saturated fat and cholesterol in their diets, to stop smoking and to increase the amounts of unsaturated oils such as margarine and vegetable oils. After one year, those on the "good" diet had 100 percent more deaths than those on the "bad" diet, in spite of the fact that those men on the "bad" diet continued to smoke.[2] In a study of Indians from Bombay and Punjab, researchers found that those from Punjab had one-fifth the number of heart attacks even though they smoked eight times more cigarettes.[3] And while smoking was widespread at the turn of the century, myocardial infarction was not. This suggests that there may be factors in traditional diets that protect against the negative effects of smoking. It also raises the question of whether additives now used in cigarette paper and filters and changes in the curing process itself have exacerbated the harmful effects of cigarette use.

Perhaps the association between smoking and heart disease is really an association with some other factor—stress, biochemical imbalances, nutrient deficiencies—that creates the desire or the need to smoke. Often when people quit smoking they become nervous and overweight, which may seem a bad bargain of one risk factor in exchange for two more.

Regular physical activity is one of the few risk factors that has proved consistent. In all studies, regular physical activity is inversely associated with mortality from CHD, and physical activity is the only factor that has shown dose-response in the trials. Common sense tells us why exercise may be beneficial. When we exercise, our heart beats more rapidly, the arteries widen to provide more oxygen and arterial blood flow improves.

Lack of exercise may also be a risk factor because it is a marker for something else that is the true cause. People who are overweight, for example, are less inclined to exercise. Prosperous people who have leisure time are more likely to exercise than those who must work long hours to make ends meet—and we know that heart disease in westernized nations is more prevalent among the poor.[4] Dietary factors may make people less inclined to exercise. An interesting finding in the Framingham study was that those who ate the most saturated fat, the most calories and the most cholesterol were the most physically active.[5] They also weighed the least and had the lowest levels of serum cholesterol!

Common sense also tells us why overweight may be a risk factor. People who are overweight are less inclined to exercise. They probably eat large quantities of refined foods that provide lots of calories but little nourishment. They may have biochemical imbalances that contribute not only to overweight but also to some of the many aspects of heart disease, such as the tendency to form blood clots.

Many doctors have noticed that heart attack often strikes in the months just after severe emotional trauma—loss of a spouse or close friend, bankruptcy, layoff or disappointment. We know that grief changes many aspects of the body chemistry, making us more vulnerable to all sorts of diseases—not just heart disease but also cancer, allergies, tuberculosis and depression. But mankind has always suffered loss and grief. The question is why these traumas cause heart attacks today but not in 1900.

Although the known risk factors may not be the underlying causes, it makes sense to exercise regularly, to avoid smoking, to maintain an appropriate body weight and to minimize stress. Unfortunately, avoidance of these risk factors is no guarantee. We all know of slim, nonsmoking, active, successful individuals who have developed heart disease—including athletes who have keeled over while jogging. And stress cannot always be avoided. All of us face loss and challenge. The question is, how do we fortify ourselves to deal with stress in a way that minimizes its impact on the physical body?

THE ABCs OF NUTRIENT DEFICIENCIES

In 1930, Dr. Weston Price published an interesting paper in the *Journal of the American Dental Society*.[6] For years, Dr. Price had been analyzing the amount of fat-soluble vitamins in butterfat—vitamins A, D and a third nutrient he referred to as Activator X. He noted that these nutrients were most plentiful in the spring and fall, when cows had access to rapidly growing green grass. During the winter and the dry summer months, levels of these vitamins in butterfat declined or disappeared completely.

Dr. Price also tabulated the number of deaths from heart attacks in local hospitals. When he plotted these two variables against time on the same graph he found that deaths from heart disease were inversely proportional to the vitamin content in the butter. In other words, when nutrient levels were high, deaths from heart disease were low; and when nutrient levels were low in the winter and summer, deaths from heart disease were high. He found this pattern in many different localities, even in areas in the far north where there was only one vitamin peak, in midsummer, due to the short growing season.

Heart disease researchers have largely ignored the possible role of vitamin A and D in protecting the heart, probably because these fat-soluble vitamins are found only in the foods they have demonized—animal fats. Yet both nutrients play numerous important roles in the body chemistry, principally as catalysts for protein and mineral assimilation.[7] Both nutrients

support endocrine function and protect against inflammation. Vitamin A is needed for the conversion of cholesterol into steroid hormones and, in fact, is rapidly depleted by stress. Cholesterol-lowering drugs increase the body's need for vitamin A.

Vitamin D helps prevent high blood pressure and protects against spasms. As vitamin D is needed for calcium absorption, it contributes to a healthy nervous system and helps prevent arrythmias.

Only recently has Activator X been identified as vitamin K_2, the animal form of vitamin K.[8] This vitamin prevents calcification of the cardiovascular system and appears to protect against the inflammation and accumulation of lipids and white blood cells which characterize atherosclerosois. In fact, research is rapidly redefining heart disease largely as a deficiency of this vitamin.

In the 1960s, a pair of Canadian doctors named Wilfred and Evan Shute claimed to prevent recurrence of problems in CHD patients with the administration of vitamin E.[9] They pointed out that lack of vitamin E in the American diet is partially due to the milling process which eliminates the highly perishable wheat germ, a significant source of vitamin E. High levels of rancid polyunsaturated fatty acids from commercial vegetable oils can actually raise the body's requirements for vitamin E. Vitamin E is an antioxidant that can prevent free radicals from causing damage at the cellular level and it plays an essential role in cellular respiration, particularly in the cardiac muscles. Vitamin E makes it possible for these muscles and their nerves to function with less oxygen. It promotes dilation of the blood vessels and inhibits coagulation of the blood by preventing clots from forming.

Dr. Linus Pauling, famous for his work on vitamin C, proposed vitamin C deficiency as a possible cause of CHD.[10] A six-year Finnish study linked low blood levels of vitamin C to increased risk of heart attack during subsequent years.[11] As an antioxidant, vitamin C protects against free radical damage. It has the effect of making oxygen metabolism more effective and may also help prevent clot formation. Vitamin C is essential for the production of collagen and therefore protects the integrity of the artery walls. Vitamin C is used up very quickly during periods of stress.

Researcher Kilmer McCully has found a positive relationship between deficiencies in folic acid, B_6 and B_{12} and severity of hardening or stiffness of the arteries, as well as the buildup of pathogenic plaque.[12] Vitamin B_6 and vitamin B_{12} are found almost exclusively in animal products—the foods that proponents of the lipid hypothesis advise us to avoid.

Another nutrient found exclusively in animal products, particularly in red meat and organ meats, is coenzyme Q_{10}, which serves as an antioxidant and as fuel for the mitochondria in the cells. In the body, coenzyme Q_{10} is most concentrated in the heart muscle cells. It seems to be helpful in reducing inflammation and has been used successfully in the treatment of heart disease.[13] Cholesterol-lowering drugs greatly increase the body's need for coenzyme Q_{10}.

Deficiencies of certain minerals have also been proposed as possible causes of heart disease. According to Dr. Roger Williams, an inadequate supply of magnesium may result in the formation of clots and contribute to calcium deposits in the blood vessels.[14] Heart attack patients improve their survival chances from 50 to 82 percent when given intravenous magnesium in the first 24 hours following myocardial infarction although this simple treatment is rarely given at hospitals.[15]

Many other minerals play a role in cardiovascular health. Copper and zinc, for example, are contained in enzymes that the body uses to defuse free radicals and that help create healthy collagen. These minerals are most easily assimilated from animal foods.

Deficiency of selenium has been linked to CHD[16] and is associated with Keshan disease, characterized by fibrotic lesions in the heart.[17] In conjunction with vitamin E, selenium has been used successfully to reduce or eliminate angina attacks. Soils in most of Finland are deficient in selenium, which may account in part for the fact that heart disease in that country is high. A national program to add selenium to the soil, initiated in 1985, may offer partial explanation for the decline in heart disease in Finland (although the decline began before the selenium enrichment program was instituted).

It is easy to make the case that, in spite of our prosperity, the actual nutrient content of our foods has declined during the last 70 years. A number of researchers have cataloged the decline in soil minerals, due to intensive farming practices.[18] Most milk in the US today comes from cows housed in confinement dairies. They are fed dry feed and never see the green grass their bodies need to make large quantities of vitamin A nor the sunlight they need to make vitamin D. Isolated isomers of vitamin D are added to milk in an attempt to rectify this situation. Processed food, usually based on sugar, white flour and vegetable oils, has replaced many nutrient-dense foods that were eaten routinely in the past. Few Americans eat liver on a weekly basis any more or take cod liver oil as our ancestors did.

Nor do they use lard, which is another rich source of vitamin D. Like humans, pigs can get sunburned and, like humans, they make vitamin D through the action of sunlight on their skin and store the nutrient in their fat. Pigs raised in confinement will die if not exposed to UV-B light, the wave length needed for vitamin-D production. Fifty years ago, lard contributed important nutrients to the American diet but few people use it today.

ELUSIVE ANSWERS

The problem is that it is difficult to turn these clues and theories into solid scientific research. As vitamins and minerals work in synergy, it is impossible to accurately assess their effects as separate entities. For example, vitamin A and vitamin D are needed for magnesium and calcium absorption; vitamin C works with vitamin E and vitamin E works with selenium.

And whether nutrients are absorbed is also dependent on many factors. Phytic acid and oxalic acid in plant foods like soy and certain raw vegetables, for example, can block absorption of many minerals. Endocrine insufficiencies and lack of beneficial intestinal flora may inhibit nutrient absorption, even though the nutrients are plentiful in the food consumed.

Added to this is the fact that vitamin and mineral content of our foods varies enormously. Researchers cannot rely on nutrient tables to determine the quantities of vitamins and minerals their patients are consuming. They must analyze all the foods eaten to get accurate numbers—an expensive undertaking.

Health officials have attempted to get around this problem by giving synthetic vitamins in pill form, but this practice presents problems as well. Synthetic, vegetarian-sourced vitamin D_2 added to milk actually has the opposite effect of animal-sourced vitamin D_3, causing decalcification of the hard tissues and calcification of the soft tissues, including the soft tissues of the arteries.[19] For this reason, the dairy industry has quietly replaced D_2 added to milk with D_3, but D_2 is still added to increasingly popular imitation milks made from soy, rice, almonds and oats. Synthetic vitamin E has had disappointing results in trials[20]—the Shute brothers actually used wheat germ oil, a source of natural vitamin E complex. Synthetic vitamins B_1 and B_2 can cause imbalances affecting the utilization of B_6. In general, vitamins from food work more efficiently and are needed in smaller quantities than synthetic vitamins. Animal studies indicate that minerals taken in as a part of whole foods have more beneficial effects than those given as supplements.

Vitamins and minerals can be ineffective or even toxic in large amounts. Individuals with high levels of serum vitamin C had no better long term survival rates that those with levels that were in the normal range.[21] The single negative study showing that magnesium had a detrimental effect on CHD survival (and used to justify withholding magnesium treatment after a heart attack) employed a far higher dose of magnesium than studies showing a positive effect.[22]

These complications do not mean that the effects of vitamins and minerals on cardiovascular health cannot be studied. It does mean that these

ALL ABOUT ANGIOGRAPHY

One method doctors use to determine the effectiveness of various drug and dietary treatments for heart disease is coronary angiography. It is performed by injecting iodine atoms into the blood vessel and taking an X-ray. A narrow and flexible plastic tube is inserted into the femoral artery in the groin and pushed gently upwards through the aorta, the chief artery of the human body, until it reaches the vessel to be investigated, such as the coronary vessels, those that provide the heart muscle with blood. When the tip of the catheter reaches the entrance of one of the coronary vessels, the iodine solution is slowly injected.

Let us keep in mind that a change in diameter of the coronary artery is nothing but a surrogate outcome. It is assumed that a widening of a coronary vessel on an X-ray means less atherosclerosis and thus a better chance to avoid a heart attack, but this is only an assumption. It is also important to realize that the differences observed in vessel diameter involve only very small changes, changes measured in hundreths of a millimeter

Artery walls are surrounded by smooth muscle cells. When such cells contract, the artery narrows. When they relax, it widens. Various factors may stimulate the smooth muscle cells of the coronary arteries to contract including mental stress, anxiety, exposure to cold and even a sustained hand grip. The latter effect was studied by Dr. Greg Brown who found that a hand grip sustained for a few minutes was followed by a 35 percent decrease in the vessel diameter. Since almost all heart disease patients receive drugs that relax the coronary vessels, and since the insertion of the tube into the groin and upward into the aorta is in itself a stressful experience—one that might cause the patient to clasp his hands in a sustained grip—changes observed through angiography can hardly have any value in the study of diet or drugs.

There are more uncertainties. Dr. Seymor Glagov and his colleagues from University of Chicago studied the hearts of 136 deceased individuals and found that when vessels become sclerotic, they actually widen to compensate for the narrowing brought about by the deposition of cholesterol in their walls. In fact, this widening overcompensates for the deposition until the cholesterol deposits occupy about 40 percent of the area beneath the muscle wall. Only thereafter does the vessel become steadily narrower. In other words, an increase of vessel diameter may be due to better relaxation of the vessel wall or disappearance of cholesterol in a highly sclerotic vessel; but it also could be due to a compensatory widening during the first stages of cholesterol deposition. Yet angiographic results are used to justify various cholesterol-lowering regimens, from lowfat diets to cholesterol-lowering drugs.

Excerpted from *The Cholesterol Myths* by Uffe Ravnskov, MD, PhD.

studies must be performed with great care. Experts in the biochemistry of human nutrition should be involved in the design of such studies—something that rarely occurs. Study design must also include built-in protection against bias—from both those who are antagonistic to the view that nutrition plays a role in heart disease and those who may be too eager to embrace a strategy that relies on supplements.

Many opportunities to find dietary causes of heart disease have been squandered. Dr. Price's research on butter and heart disease, for example, could not be repeated today, partly because Americans no longer consume foods grown locally and partly because most have given up eating any butter at all. Data from the 1960s cited by Ancel Keys in his Seven Countries Study found a fivefold difference in rates of heart disease between Crete and Corfu.[23] Keys and his colleagues had a unique opportunity to look at subtle dietary differences, including differences in soil composition, water content and cooking methods, because both populations consumed mostly locally grown food at the time but probably no longer do. Unfortunately, no one pursued this line of research.

ADVENTURES IN MACRONUTRIENT LAND

Macronutrients are the larger components of our food—proteins, carbohydrates and fats. Proponents of the lipid hypothesis have zeroed in on the fat component of our diet, blaming either all fats or just saturated fats for the CHD epidemic. The "prudent" diet calls for reduction of fat consumption to 30 percent of caloric intake and of saturated fat consumption to just 10 percent of caloric intake, or less than two tablespoons of saturated fatty acids in a diet of 2400 calories.

What clues can we derive from a study of lipid consumption patterns? One is that the actual amount of fat in the diet probably does not matter (except when it is so low as to result in deficiencies). The amount of fat in the American diet has held fairly steady at 35-40 percent of calories for the last 90 years, during the period when rates of heart disease were rising. The Masai, with 60 percent of their calories from fat, are free of heart disease. The traditional diet of the Eskimo and the North American Indians contained as much as 80 percent of calories as fat and there is no indication that they suffered from heart disease.

What consumption patterns do indicate, however, is that it is the type or quality of fat that matters. Ninety years ago, Americans consumed mostly animal fats—lard, butter and tallow from pasture-fed animals. These fats were stable and provided many important fat-soluble nutrients. Today most of the fats in the American diet are derived from plants—as liquid vegetable oils or oils that have been hardened through the process of partial hydrogenation. Large amounts of calories from polyunsaturated vegetable oils are new to the human diet and should certainly be explored more fully as a contributing factor.

There are several ways in which modern vegetable oils may have an adverse effect on CHD. First, because of modern processing methods, they tend to be rancid. Rancid fats contain large numbers of free radicals, molecules with unpaired electrons that are highly reactive. Free radical damage in the arteries is thought to be an important factor in the initiation of plaque. Secondly, these oils lack vitamins A and D found in animal fats and through processing are likely to be shorn of naturally occurring vitamin E and other antioxidants.

Another problem is that when polyunsaturated oils are consumed in large amounts, imbalances can occur that may predispose to heart disease. Research suggests that traditional diets contained from four to ten percent of calories as polyunsaturated fatty acids with a ratio of about twice as many omega-6 fatty acids (mostly linoleic acid) as omega-3 fatty acids (mostly a-linolenic acid).[24]

Individuals who are trying to avoid saturated fats often end up with over 20 percent of calories as polyunsaturated fatty acids. The situation is further complicated by the fact that commercial vegetable oils contain mostly omega-6 fatty acids. The body uses these types of fatty acids to make localized hormones, called prostaglandins, that initiate the process of blood clotting and of inflammation. This is an important mechanism. Without it, we would bleed to death when we cut ourselves and our wounds would not heal. The problem occurs when these clot- and inflammation-promoting prostaglandins are not balanced by prostaglandins that inhibit clotting.

Many of the anti-inflammatory and clot-inhibiting prostaglandins are made from omega-3 fatty acids, of which there are very few in commercial vegetable oils, or indeed in fruits, vegetables, fish and eggs raised by modern farming methods. Thus, when the diet contains too much omega-6 fatty acids and not enough omega-3 fatty acids, there may be a tendency to form blood clots leading to heart attacks.[25]

The research on omega-3 fatty acids is not conclusive. While some studies indicate that omega-3 fatty acids may be helpful, others showed no effect. One explanation for this may be found in the fact that saturated fats help the body store and use omega-3 fatty acids more effectively.[26] Therefore, we would expect to find a correlation with consumption of omega-3 fatty acids and low rates of heart attacks in populations that use traditional diets containing saturated animal fats. But when omega-3 fatty acids are given to individuals who are avoiding saturated fats, the outcome may not be positive. In fact, there is evidence that overconsumption of omega-3 fatty acids in a diet lacking in satu-

rated fats may actually be bad for the heart. In test animals, diets high in canola oil, which is relatively high in omega-3 fatty acids but low in saturated fats, caused fibrotic heart lesions, vitamin E deficiencies and abnormal changes to the blood platelets.[27] When saturated fats were added to the canola-based diets, these problems did not occur.

Trans fatty acids are hardened fats created from liquid vegetable oil by a process called partial hydrogenation, and much evidence supports the theory that these manufactured fats contribute to heart disease.[28] The tragedy is that those who are trying to avoid saturated fats and cholesterol will probably eat more *trans* fatty acids, because these are used in foods promoted as low in saturated fat and cholesterol.

Those who are trying to avoid eating lots of fat often replace fat calories with carbohydrate calories, usually calories in the form of refined flour and sugar. Yet several researchers have published studies linking consumption of refined carbohydrates, particularly sugar, with increased heart disease, including Yudkin in the 1950s and Lopez in the 1960s. Yudkin found that use of sugar was associated with increased adhesiveness of the blood platelets, increased blood insulin levels and increased blood corticosteroid levels (a sign of stress).[29]

In addition, sugar consumption is associated with increased incidence of diabetes, and diabetics are said to be prone to heart disease. One researcher has noted that a diet high in any type of carbohydrate, including carbohydrates from whole cereal grains, is associated with CHD.[30] Of course, many products containing white flour and sugar also contain high levels of *trans* fatty acids and improperly prepared whole grains contain phytic acid that can block the uptake of important minerals including magnesium, zinc and copper.

Protein, the third macronutrient, also plays a role in heart health. When protein intake is inadequate, the heart muscle shrinks and cannot perform effectively.[31] But supplementation with liquid protein drinks predisposes to arrhythmias. High protein diets that do not contain fats, particularly animal fats, can deplete stores of vitamin A and D and consequently interfere with mineral assimilation.[32]

OTHER THEORIES PROPOSED TO EXPLAIN THE CHD EPIDEMIC

Price	Deficiency of fat-soluble vitamins A and D
Yudkin, Ahrens	Refined carbohydrates
Kummerow, Mann	Trans fatty acids from hydrogenated fats
Hodgson	Excess omega-6 from refined vegetable oils
Addis	Oxidized cholesterol and oxidized fats (free radicals)
Shute	Vitamin E deficiency
Pauling	Vitamin C deficiency
McCully	Deficiency of folic acid, B6 and B12
Webb	Protein deficiency
Anderson	Magnesium deficiency
Huttunen	Selenium deficiency
Klevay	Copper Deficiency
Geliejnse	K2 Deficiency
Annand	Heated milk protein (pasteurization)
Oster	Homogenization
Ellis	Microbial agents (viruses, bacteria)
Benditt	Monoclonal tumor theory
Gofman	Exposure to x-rays
de Bruin	Thyroid deficiency
LaCroix	Coffee consumption
Morris	Lack of exercise
Stern	Exposure to carbon monoxide
Purdey	Exposure to pesticides
Ridker	Inflammation
Marmot	Stress
Ravnskov	Infection
de Mesquita	Acidosis of the Heart
Barker	Low Birth Weight
Smith	Changes & fashions in reporting cause of death

BAD CHOLESTEROL?

Scientists now realize that the cholesterol that our bodies make, and that we get from traditional foods, does not cause heart disease. But cholesterol, like polyunsaturated fatty acids, may become oxidized or rancid when it is processed at high temperatures. In early experiments with vegetarian rabbits, purified solutions of processed cholesterol were used, cholesterol that was rancid or oxidized. Oxidized cholesterol accumulates in the foam cells that are involved in the buildup of pathogenic plaque.[33]

Rancid or oxidized cholesterol occurs in powdered eggs and milk, both used in many processed foods. Powdered milk is added to lowfat milks to give them body.

Another type of cholesterol is Lp(a) which occurs in humans, other primates and guinea pigs, organisms that do not manufacture vitamin C. Nobel laureate Linus Pauling and his colleague Mathias Rath proposed that our bodies produce Lp(a) to compensate for low levels of vitamin C.[34] They caused atherosclerosis in guinea pigs by depleting their bodies of vitamin C. Vitamin C depletion caused Lp(a) to appear in the plaque. A high level of Lp(a) is a risk factor for heart disease.[35] That does not mean the Lp(a) is the cause. The cause may be vitamin C deficiency in association with other factors, such as low levels of vitamin B_3 (niacin), which also lowers Lp(a). Consumption of *trans* fatty acids causes levels of Lp(a) to rise while consumption of saturated fats lowers blood levels of Lp(a).[36]

INFECTION AND HEART DISEASE

A number of pathogens have been associated with the development of CHD or have been found in the atherosclerotic lesions at autopsy, including both viruses and bacteria.[37] These pathogens have been around as long as man has lived on the earth. The culprit, therefore, is not the microbes but a compromised immune system which can no longer deal with them appropriately. A healthy immune system depends on an array of nutrients, including vitamin A, vitamin C and various minerals that play an antioxidant role.

One of the most tragic aspects of the cholesterol campaign is that it has caused Americans to abandon the very fats that provide protection against infection. Not only do animal fats carry vitamin A, they also contain palmitoleic acid, a 16-carbon monounsaturated fatty acid that has strong antimicrobial properties. Butterfat and coconut oil contain fatty acids that have similar properties. They protect against viruses and pathogenic bacteria and enhance the immune system. Areas of the world where coconut is consumed have low levels of heart disease.

THYROID

Thyroid insufficiency has been identified as a risk factor for heart disease, but treatment with thyroid hormone replacement does not necessarily improve the outcome.[38] Hormones taken orally may have unexpected effects compared to those produced by the body, effects that may increase the risk of heart disease, such as the provocation of arrythmias. Thyroid health depends on iodine status, but other factors are involved. Vitamin A, for example, plays a key role in thyroid health.[39] As individuals with poor thyroid function have difficulty converting carotenes in plant foods into true vitamin A, they must obtain adequate vitamin A from animal foods. Unfortunately, patients with thyroid problems are often advised to follow a lowfat diet because they are prone to heart disease.

OTHER THEORIES

Many other theories have been proposed to account for the current epidemic in CHD: chlorine and fluoride added to water; pesticides that mimic human estrogens or that provoke free radical reactions; carbon monoxide fumes; industrial chemicals; artificial lighting; synthetic vitamins; minerals that are toxic or that are consumed in toxic amounts; pasteurization and homogenization of milk; legal and illegal drugs; consumption of coffee and other stimulants; and additives in processed foods. Most are factors unique to the twentieth century and all need further study.

But who will do this work? Even today, all but a small fraction of the research dollar still goes to further study of the lipid hypothesis, and vested interests have the power to prevent funding for studies that may prove embarrassing.

SOLUTIONS

How can we protect ourselves against heart disease? Based on what we have learned from the scientific studies, it is possible to formulate a set of guidelines for heart disease prevention, guidelines that include both avoidance of external stressors and common sense dietary advice. Not all external stressors can be avoided, not in today's fast-paced industrial age, but a good diet can provide many factors that help the body deal with environmental toxins and high levels of stress.

There are many points contained in the following guidelines that can be debated but one thing is certain: if you are still afraid of saturated fats and cholesterol, you will find yourself on the wrong dietary path. If you are avoiding foods containing saturated fat and cholesterol, you will not only deprive your body of vital nutrients, but the foods that you consume as substitutes will contain many components—polyunsaturated oils, *trans* fatty acids, refined sugar—that have been associated with increased rates of heart disease.

DR. ORNISH AND THE LIFESTYLE HEART TRIAL
from *The Cholesterol Myths* by Uffe Ravnskov, MD, PhD

Coronary heart disease is a multifactorial disease that requires multifactorial intervention. This is the view of Dr. Dean Ornish and his group at the Preventive Medicine Research Institute, Sausalito, California, a view they share with many other doctors and researchers. Dr. Ornish and his group chose to intervene with a lowfat, low-cholesterol vegetarian diet, stopping smoking, stress-management training and moderate exercise. They selected 94 patients with a diagnosis of coronary artery disease according to a previous coronary angiogram. Fifty-three were randomly assigned to the experimental group and 43 to the control group, but when told about the design of the study only 28 and 20, respectively, agreed to participate.

A new angiogram was performed after one year, but one of the angiograms disappeared; in three patients the second angiogram could not be evaluated; one patient was not studied because of unpaid bills; one died during heavy exercise; and one dropped out because of alcohol misuse. Thus, only 22 patients in the experimental group and 19 in the control group were available for analysis.

The results seemed promising. In the treatment group, total cholesterol fell by an average of 24 percent and LDL-cholesterol by 37 percent; mean body weight decreased by ten kilograms; less severe chest pains were reported; and the coronary arteries widened a little, whereas they became a little more narrow in the control group. These improvements were strongly related to the degree of adherence to the intervention program in a "dose-response" manner, as the authors wrote in their report. The vascular improvements were still there after a prolongation of the study by five years, but now the difference was calculated using less demanding statistical parameters. Unfortunately, there was no difference in frequency, duration or severity of angina between the groups, but this unexpected finding was "most likely" due to bypass operations performed in the control group. Nothing was mentioned about how many operations had been performed, however, and no comparison was made between those who had not had an operation. In addition, a further six individuals were unavailable for follow-up study.

And there were more flaws. Not only was it an unblinded study (although in the latest publication it was called blinded!), the low number of participants also resulted in a most uneven distribution of the risk factors. For instance, at the start the mean age was four years higher, mean total cholesterol 8 percent higher and mean LDL-cholesterol 10 percent higher in the control group; but mean body weight was almost 25 pounds higher in the treatment group. Such large differences between risk factors obviously complicate evaluation of the treatment effect.

But let us assume that the improvement of the treated individuals was true and a result of the intervention—and this may well be possible—which of the intervention measures had a beneficial effect? Was it a weight reduction of more than 25 pounds? Was it a difference in smoking habits? (One in the experimental group smoked and stopped; nothing was mentioned about the number of smokers in the control group.) Was it the exercise? Was it the inner sense of peace and well-being produced by the stress-management education? Or was it a combination of these factors?

That the diet had any importance is unlikely because there is no evidence that vegetarians have a lower risk of coronary disease than other people. It is also unlikely that it was the change in LDL-cholesterol levels because at the end of the study there were no significant differences between these values in the two groups. The latter also contradicts the statement that the changes of coronary atherosclerosis and the diet were strongly correlated in a "dose-response" manner. To the pertinent question "Precisely how strong were the correlations?" asked by Elaine R. Monsen, editor of *Journal of the American Dietetic Association*, Dr. Ornish answered that "the study wasn't really set up to do these kinds of analyses, so when we get beyond saying they're correlated, we're on shaky ground."

It is laudable to try prevention without drugs, and we already know it may be health-promoting to avoid being overweight, to exercise a little and to avoid smoking and mental stress, but with such weak evidence, why inflict millions of people with a diet that only rabbits find tolerable? Perhaps the results would have been better if the patients' inner sense of peace and well-being had been strengthened even more by allowing them to follow a more appealing and nutritious diet!

(Ornish D and others. Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *The Lancet* 336, 129-133, 1990; Ornish D. Reversing heart disease through diet, exercise and stress management: An interview with Dean Ornish. *Journal of the American Dietetic Association* 91, 162-165, 1991; Gould KL, Ornish D and others. Changes in myocardial perfusion abnormalities by positron emission tomography after long-term, intense risk factor modification. *Journal of the American Medical Association* 274, 894-901, 1995)

TEN COMMANDMENTS FOR AVOIDING CHD

1. Don't smoke. If you find it impossible to quit, at least try to cut back and smoke only additive-free cigarettes. Smokers should avoid polyunsaturated oils at all costs. Saturated fats and vitamins A and D are particularly protective of the lungs.
2. Exercise regularly but you needn't overdo. A brisk daily walk, ten minutes on the trampoline, swimming in non-chlorinated water and sports are all appropriate.
3. Avoid overweight. Eat nutrient-dense foods and keep sweets to a minimum, but avoid crash dieting.
4. Don't work too hard. Counteract stress by doing something that you love to do everyday. During periods of unavoidable hardship or loss, increase consumption of foods rich in protective nutrients.
5. As much as possible, avoid exposure to fumes, chemicals, pollutants and pesticides.
6. Avoid all processed foods labeled "lowfat" or that contain polyunsaturated vegetable oils, hydrogenated fats, white flour, refined sugar and additives.
7. Consume high-quality animal products including a variety of sea food and whole raw milk, butter, cheese, eggs, meat, fats and organ meats from animals raised on green pasture.
8. Consume a variety of fresh vegetables and fruits, preferably organically grown.
9. Ensure sufficient mineral intake by using whole raw dairy products; homemade bone broths; and whole grains, legumes and nuts that have been properly prepared to reduce phytic acid and other factors that block mineral absorption.[40]
10. Supplement the diet with foods rich in protective factors including small amounts of cod liver oil (vitamins A and D); wheat germ oil (vitamin E); flax oil (omega-3 fatty acids); kelp (iodine); nutritional yeast (B vitamins); desiccated liver (vitamin B_{12}); rose hip or acerola powder (vitamin C); and coconut oil (antimicrobial fatty acids).

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WHAT CAUSES HEART ATTACKS?

By Tom Cowan, MD

The kidneys nourish the heart. Traditional Chinese medical texts.

The story of how I came to understand the cause, and therefore the appropriate treatment, of acute coronary syndrome involves fascinating elements of surprise and serendipity. I thought it best, therefore, to describe how this tale unfolded for me.

Acute Coronary Syndrome (ACS) describes a constellation of illnesses that include angina (chest pain), unstable angina (basically bad chest pain) and myocardial infarction (otherwise known as heart attack or MI). These three illnesses form a continuum, with angina as the mildest symptom and heart attack—when there is actual death of the heart cells—as the most severe. The history of thought about this group of illnesses is both fascinating and controversial.

It seems that heart attacks were rare in this country until about the 1930s. The incidence of fatal MIs quickly increased from about 3,000 per year during that decade to almost half a million per year during the 1950s. In fact, mid century, this formerly rare disease had become the leading cause of death in the US. The incidence has risen continually since then until just recently, when it seems that the tide may be turning a bit and the incidence lessening, or at least leveling off. Nevertheless, after decades of reckless fiddling with the American diet as a way to prevent heart disease, almost a million Americans still die from heart disease each year.

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This theory about the cause of heart attacks is so ingrained in our culture that until recently, even a medical skeptic like myself never really questioned it.

THE CONVENTIONAL THEORY

As you can imagine, when it became clear that we were suffering from an epidemic of this disease, physicians and cardiologists developed an intense interest in the cause and possible treatment of the disease. Around the late 1940s, the medical establishment proposed a simple and plausible explanation for MI, and this explanation soon became universally accepted.

The current thinking about heart attacks focuses on the blood supply to the myocardial (heart) cells from the network of coronary arteries, that is, the arteries that supply blood to the heart itself. There are four main arteries, each supplying blood to a different region of the heart. Medical experts believe that when one or more of these arteries gets blocked with plaque, a condition called atherosclerosis, then the inside of the artery becomes narrowed, the blood flow becomes compromised and, in times of myocardial stress (such as exercise or emotional trauma), the insufficient blood flow causes damage to the particular region of the heart fed by the blocked artery. This diminished blood flow first causes pain (angina) and then, if more severe, death to the heart tissue.

from *Materia Medica and Pharmacognosy*
David Culbreth, M.D. (1927)

Here was an elegant and plausible theory. Voilà! Case closed. The only thing left to figure out was what was causing the arterial blockages. This answer was famously supplied by Dr. Ancel Keys in the 1950s. Keys fingered cholesterol as the culprit, claiming that excess cholesterol floating around in the blood built up as plaque in the arteries. For over fifty years the theory has survived without any significant changes. In fact, if someone has a heart attack today, we often call it a "coronary," referring to the presumed source of the problem, the coronary arteries.

This theory about the cause of heart attacks is so ingrained in our culture that until recently, even a medical skeptic like myself never really questioned it. My only issue with the theory centered on the material in the plaque, which research subsequently revealed to be mostly inflammatory debris, not cholesterol. But I never really gave any thought to the basic premise, namely, that blocked arteries cause heart attacks.

It should be mentioned that this theory about the cause of heart attacks has led to a massive industry devoted to its diagnosis and treatment. Angiograms (in which dye is injected into the vessels to see if they are blocked), bypasses, stents, angioplasties (like roto-rooters for blocked arteries), cholesterol-lowering drugs and lowfat, low-cholesterol diets are all based one hundred percent on the acceptance of blocked arteries as The Cause of acute coronary syndrome. The whole debate in modern cardiology, both alternative and conventional, is how to stop the buildup of plaque or—more recently—how to prevent plaque in the arteries from breaking free and forming a clot, thereby completely blocking an artery already narrowed by the buildup.

THE DIGITALIS CONNECTION

Around two years ago I received an email from the son-in-law of a recently deceased and apparently well-known Brazilian cardiologist, Quintilaino H. de Mesquita. Before he died, Dr. Mesquita had published a summary of twenty-nine years of research carried out at his cardiology hospital, data on what he called the "true cause

and effective treatment of MIs." His son-in-law and fellow researcher, Carlos Monteiro, emailed me a simple question, which was: "When you put your cancer patients on low-dose whole digitalis plant extract, does this lower their incidence of MIs?"

His question was actually a response to a series of articles describing the effectiveness of low-dose whole digitalis leaf extract in the treatment of a variety of cancers, which I had recently posted on my website, www.fourfoldhealing.com. I wrote back asking why he wanted to know this. He replied that in Dr. Mesquita's groundbreaking study on what he called the myogenic (that is, arising from the muscle) theory of heart disease, he had stumbled on an unexpected result: the digitalis they were using to treat MIs had also dramatically lowered the incidence of cancer in their heart patients, and mine was the only website they found that mentioned this association.

As I had never heard of either the myogenic theory or of the use of digitalis for heart attack, I asked what this was all about. His response was a box of articles and books all published over the last fifty years that seemed to refute the coronary blockage theory of MIs and support what he called the myogenic theory. I spent the next two months poring over these studies until I became convinced that this was perhaps the biggest medical news of the decade, maybe of the entire century.

THE MYOGENIC THEORY

Briefly, the myogenic theory of MIs states that:

1. The coronary obstruction theory does not adequately explain all the observed facts concerning MIs.
2. The major etiologic (cause and effect) factor in an MI is a destructive chemical process; specifically, in situations of stress on the myocardial (heart muscle) tissue, often as a result of small vessel disease, the myocardial tissue gets insufficient oxygen and nutrients. This leads to destructive lactic acidosis in the tissue which, if unchecked, leads to death of the myocardial cells. This process is largely unrelated to coronary artery disease.
3. The regular use of cardiotonics, primarily low-dose whole digitalis extracts or an extract of another herb called g-strophanthin,

Interestingly, in the 1940s and 1950s, when the coronary blockage theory was first proposed, the majority of cardiologists did not accept it.

WHY PLAQUE IS A PROBLEM

While plaque in the arteries leading to blockage may not be the main cause of heart disease, there is no doubt that the phenomena of athersclerosis (plaque formation) is a real problem in people, especially as we age. Certain sections of our arteries are subject to thickening and the formation of what is called fatty streaks for reasons that have to do with flow dynamics, that is, the velocity of blood flow and turbulence in that particular artery. A certain amount of thickening in places where the blood creates a lot of pressure on the arteries is normal and protective, and it therefore occurs in everyone.

But the build up of plaque is a different situation and can lead to many problems. For example, blocked arteries in the legs can cause calf cramps and pain, which we refer to as intermittent claudication (leg pain while walking). In the brain, plaque formation leads to ischemic (lack of blood flow) stroke. In the kidneys, diminished blood flow due to plaque formation is a possible contributing factor in some cases of hypertension (high blood pressure). Likewise, blocked arteries leading to the liver or spleen can result in reduced function of these organs.

The reasons for this plaque formation are unclear. Although scientists have long blamed such build up on high cholesterol levels in the blood, informed medical researchers today often cite inflammation in the vessels as the cause. Of course, this inflammation is secondary to other factors, such as stress, consumption of processed vegetable oils and nutrient deficiencies (particularly of vitamins A and C and minerals like copper).

But plaque formation is not a sufficient explanation for the whole phenomena of myocardial ischemia. The reason the heart but not the spleen or the liver has "attacks" is because the energy use of the heart is so much higher and also because the heart can never rest. Because scientists have overlooked these factors, treatment of heart disease today is far less effective than it otherwise could be.

The only other organ that might be said to suffer from an "attack" is the brain when a stroke occurs. However, strokes usually happen when a clot forms in one of the arteries feeding the brain. The process is not the same as lactic acid build up in the heart.

In fact, researchers have found that the more the coronaries narrow, the less danger there is of a heart infarct.

prevents this lethal acidosis and therefore prevents and corrects the true cause of this syndrome. The result is substantially lower morbidity and mortality from heart disease.

Let's look at some of the data supporting these three conclusions. First, does the coronary obstruction theory adequately explain the observed facts? Interestingly, in the 1940s and 1950s, when the coronary blockage theory was first proposed, the majority of cardiologists did not accept it. They pointed out that while coronary arteries are not the only arteries to have plaque, the only tissue to suffer from decreased blood flow during a heart attack is that of the heart. In other words, no one has a spleen attack or a kidney attack, yet the arteries feeding these organs also get plaque buildup.

Furthermore, the medical literature reveals some surprising findings. In a 1998 paper by Mirakami,[1] the author found that of those with an acute MI, 49 percent had a blockage, 30 percent had no coronary blockage, 14 percent had insufficient blockage to impair blood flow, and 7 percent had "another condition." In a 1972 paper,[2] a researcher named Roberts showed that in acute MIs, only 50-60 percent had evidence of sufficient blockage to impair blood flow. And a 25-year autopsy study of patients who died from an acute MI, carried out by Spain and Bradess, found that only 25 percent had sufficient blockage to account for their MI, while a total of 75 percent had only mild to moderate blockage.[3]

In a second paper,[4] these same authors reported on a surprising discovery: when a heart attack is fatal, the longer the time elapsed between the MI and death (and then subsequent autopsy), the more likely they were to find significant blockages. If death occurred one hour after onset of an MI, only 16 percent had sufficient blockages to account for their MI; if death occurred 24 hours after the onset of an MI, the number with sufficient blockages to account for the heart attack increased to 53 percent. The authors concluded that the arterial blockages are a *consequence*, not a cause, of myocardial infarction.

As I looked into this subject further, I found that some of the most prominent cardiologists in our history were skeptical about the coronary artery theory of MI. For example, in 1972, Dr. George E. Burch stated, "The cardiac patient does not die from coronary disease, he dies from myocardial disease."[5] A 1980 editorial in the prestigious journal *Circulation* states, "These data support the concept that an occlusive coronary thrombus (otherwise known as a blockage) has no primary role in the pathogenesis of a myocardial infarct."[6] Finally, as recently as 1988, Dr. Epstein of the National Institutes of Health states: "They found that in an advanced state of narrowing of the coronary arteries, the supply of blood to the heart muscles is fully assured via collaterals that enlarge naturally in response to the blockage."[7] In fact, researchers have found that the *more the coronaries narrow, the less danger there is of a heart infarct.*

These shocking studies dovetail perfectly with a different study, one that rocked the world of cardiology, published in 1988 titled "Twenty years of coronary bypass surgery."[8] Referring to two major studies, the Veterans Administration (VA) study and the NIH Coronary Artery Surgery Study (CASS), the authors made the following statement: "Neither the VA nor the CASS has detected a significant difference in long-term survival between the medical and surgical treatment groups when all patients were included." In other words, surgery to bypass blocked arteries did not improve the chances of patient survival—not the result one would expect if blocked arteries were

HOW TO PROTECT YOUR CAPILLARIES

- Avoid high blood sugar: diabetes is a serious risk factor for capillary damage. A high-fat, low-carbohydrate diet is your best defense against diabetes. If you have diabetes, follow the protocol posted at www.westonaprice.org/moderndiseases/diabetes.html.
- Don't smoke! Smoking is a risk factor for capillary damage.
- Engage in moderate outdoor exercise.
- Avoid commercial liquid vegetable oils, which are full of free radicals that can damage capillaries.
- Follow a nutrient-dense traditional diet.

the cause of heart attacks. Thus, evidence for the coronary artery theory of MI is not strong; in fact, it is actually refuted in the relevant literature.

THE THEORY FITS THE FACTS

So, if heart attacks are not the result of coronary artery disease, then what does cause all these MIs? The myogenic theory of Dr. Mesquita, in fact, fits all the current observations about this condition. The myogenic theory postulates that as a result of disease in the *small* vessels—the capillaries and small arterioles—which is a consequence of such factors as stress, diabetes, smoking and nutritional deficiencies, heart cells, which are very active metabolically, suffer from inadequate oxygen and nutrient supply. This oxygen and nutrient deficiency increases under stressful conditions. When this happens, the heart cells revert to their backup system, which is anaerobic fermentation for energy generation—very similar to what happens in your leg muscles when you run too far or too hard. The anaerobic fermentation produces lactic acid which collects in the tissues. Because the heart, unlike your leg muscles, cannot rest, the acidosis progresses if untreated, leading to actual death of the myocardial cells.

As a result of this necrotic process, inflammatory debris collects in the tissues, and it is this debris that is the actual source of the coronary artery blockages seen in death from acute MI. As you would predict, the longer the time period between the MI and death, the greater the likelihood of blockage—exactly as observed in the studies. The only conclusion one can draw from this is that the heart cells die first and only then does the artery become blocked with debris liberated at myocardial cell death, which is precisely the kind of debris that is found in these blockages.

The current practice of flushing out arterial blockages can help remove the debris and restore blood flow to the compromised arterial system, but this in no way suggests that blocked arteries represent the primary event in the sequence leading to an MI. However, the whole emphasis on the coronary artery blockage is fundamentally a dead end and doomed to failure, whether it is approached from a surgical (bypass, stents, etc.) or a medical (cholesterol-lowering drugs, restricted diets, etc.) point of view.

The whole emphasis on the coronary artery blockage is fundamentally a dead end and doomed to failure, whether it is approached from a surgical or a medical point of view.

MYOGENIC THERAPY

The myogenic theory points us to a very different kind of preventive treatment for heart disease, one that focuses on small vessel disease and the prevention of heart tissue acidosis. The theory also explains why stress, diabetes and smoking are such strong risk factors for MI,

BE KIND TO YOUR ADRENAL GLANDS

Since the adrenal glands, specifically the adrenal cortex (the outer portion of the adrenal gland), produce protective cardiotonics, an important strategy in protecting yourself against heart attack is to strengthen the ability of this important gland to work properly.

- Avoid stimulants such as caffeine and related substances in coffee, tea and chocolate. Caffeine causes the adrenal medulla (the inner part of the adrenal gland) to produce adrenaline. In response, the adrenal cortex must produce a host of corticoid hormones that bring the body back into homeostasis. Repeated jolts of caffeine can lead to adrenal burnout, a situation in which the adrenal cortex is unable to produce the myriad of protective and healing substances for the body, including the cardiotonics.
- Don't try to lower your cholesterol—the cardiotonics are made from cholesterol.
- Take cod liver oil for vitamin A. The body needs vitamin A to make all the adrenal cortex hormones from cholesterol. Vitamin A intake should be balanced with vitamin D (from cod liver oil) and vitamin K_2 (from the fats and organ meats of grass-fed animals).
- Don't consume *trans* fats. *Trans* fats (from partially hydrogenated vegetable oils) interfere with the enzyme system needed for the production of adrenal cortex hormones.
- Take care to avoid low blood sugar. When blood sugar drops too low, the adrenal glands go into overdrive to produce hormones that bring the blood sugar back up. This means avoiding sugar and not skipping meals. There is just no substitute for three good meals a day, at regular intervals, which contain adequate protein and plentiful amounts of good fat.

The amazing thing is that these compounds are exact chemical copies of hormones made by our adrenal glands. And our adrenal glands produce these cardiotonics out of . . . cholesterol!

because these factors have all been shown to primarily affect small capillaries and small blood vessels, not the large coronary arteries.

But the story gets even more interesting. It turns out that there are simple, inexpensive and very effective compounds that effectively prevent lactic acidosis in the heart tissues. These medicines have been known for centuries as cardiotonics and have been used for treating heart disease in every traditional medical system in the world. The two best known are digitalis (the common foxglove) and strophanthus, an African vine. These plants are the source of so-called cardiac glycosides: digoxin and digitoxin from digitalis, and ouabain from strophanthus.

The function of these compounds is to regulate the rhythm and power of the cardiac contraction and to prevent or reverse lactic acid buildup in the cardiac tissue. This is why these plants have been used for centuries to treat congestive heart failure, rhythm disturbances and other disorders of heart function.

The amazing thing is that these compounds are exact chemical copies of hormones made by our adrenal glands. And our adrenal glands produce these cardiotonics out of . . . cholesterol! Now we know why all the draconian dietary and pharmaceutical measures to lower cholesterol have not resulted in a decrease in the rates of MI, and why numerous studies have shown that as we age, those with the highest levels of cholesterol live the longest. When we lower cholesterol, we are depriving our bodies of the very substance they need to manufacture cardiotonics.

The myogenic theory also explains why stress can lead to heart attacks. In conditions of stress, our adrenal glands must work very hard to create numerous hormones that regulate the blood sugar and help the body heal. If the adrenal glands are weak or overloaded, production of cardiotonics goes on the back burner.

While there are few studies in the conventional literature that have considered the effectiveness of digitalis or strophanthus in the treatment of MI, Dr. Mesquita's clinical results over twenty-nine years show a dramatic lowering of the death rate, recurrent MI rate, angina rate and all symptoms in the spectrum of acute coronary syndrome with the use of oral low-dose digitalis glycosides. These results are published in *Teoria Miogenica Do Enfarte Miocardico*, available through the Infarct Combat project website, www.infarctcombat.org.

Also, a German cardiologist, Dr. Berthold Kern, used g-strophanthin in a study for the German government which showed a dramatic reduction in MIs in his practice, down from the expected 400 to 20, with the use of this medicine.[9] Furthermore, many reports are coming in from Germany in which doctors have noted a decrease of up to 81 percent in angina attacks with the use of oral g-strophanthin.[10]

In my practice, I generally use oral strophanthin in the form of the preparation known as Strodival for all my angina and MI patients, and I have uniformly recorded a decrease in angina episodes, improved exercise tolerance and, thus far, no MIs. When combined with a nourishing traditional diet, cod liver oil, high vitamin butter oil, CoQ_{10} (which helps strengthen the heart muscle) and Standard Process heart nutrients (Cardioplus, two capsules three times per day, and Cataplex E2, two tablets three times per day), I have seen a huge improvement in the lives of patients with this otherwise devastating condition. (Note: Both digitialis leaf and Strodival are prescription-only items which need to be prescribed by a doctor who is well versed in their use.)

The final irony is that the traditional Chinese doctors were correct. The kidneys (their way of referring to the adrenal glands) help the body deal with stress as well as make hormones (digoxin and ouabain) that keep our marvelous hearts healthy, strong and open to enjoy the full richness of life.

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CHOLESTEROL: FRIEND OR FOE?

By Natasha Campbell-McBride, MD

The art of medicine consists in amusing the patient while Nature cures the disease. Voltaire

In our modern world, cholesterol has become almost a swear word. Thanks to the promoters of the diet-heart hypothesis, everybody "knows" that cholesterol is "evil" and has to be fought at every turn. If you believe the popular media, you would think that there is simply no level of cholesterol low enough. If you are over a certain age, you are likely to be tested for how much cholesterol you have in your blood. If it is higher than about 200 mg/100ml (5.1 mol/l), you may be prescribed a "cholesterol pill." Millions of people around the world take these pills, thinking that this way they are taking good care of their health. What these people don't realize is just how far from the truth they are. The truth is that we humans cannot live without cholesterol. Let us see why.

Our bodies are made out of billions of cells. Almost every cell produces cholesterol all the time during all of our lives. Why? Because every cell of every organ has cholesterol as a part of its structure. Cholesterol is an integral and very important part of our cell membranes, the membranes that enclose each of our cells, and also of the membranes surrounding all the organelles inside the cell. What is cholesterol doing there? A number of things.

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Children deprived of cholesterol in infancy may end up with poor eyesight and brain function. Manufacturers of infant formulas are aware of this fact, but following the anti-cholesterol dogma they produce formulas with virtually no cholesterol in them.

STRUCTURAL INTEGRITY

First of all, saturated fats and cholesterol make the membranes of the cells firm—without them the cells would become flabby and fluid. If we humans didn't have cholesterol and saturated fats in the membranes of our cells, we would look like giant worms or slugs. And we are not talking about a few molecules of cholesterol here and there. In many cells, almost half of the cell membrane is made from cholesterol. Different kinds of cells in the body need different amounts of cholesterol, depending on their function and purpose. If the cell is part of a protective barrier, it will have a lot of cholesterol in it to make it strong, sturdy and resistant to any invasion. If a cell or an organelle inside the cell needs to be soft and fluid, it will have less cholesterol in its structure.

This ability of cholesterol and saturated fats to firm up and reinforce the tissues in the body is used by our blood vessels, particularly those that have to withstand the high pressure and turbulence of the blood flow. These are usually large or medium arteries in places where they divide or bend. The flow of blood pounding through these arteries forces them to incorporate a layer of cholesterol and saturated fat in the membranes, which makes it stronger, tougher and more rigid. These layers of cholesterol and fat are called fatty streaks. They are completely normal and form in all of us, starting from birth and sometimes even before we are born. Various indigenous populations around the world, who never suffer from heart disease, have plenty of fatty streaks in their blood vessels in old and young, including children. Fatty streaks are not indicative of the disease called atherosclerosis.

LIPID LIFESAVERS

All the cells in our bodies have to communicate with each other. How do they do that? They use proteins embedded into the membrane of the cell. How are these proteins fixed to the membrane? With the help of cholesterol and saturated fats! Cholesterol and stiff saturated fatty acids form so-called lipid rafts, which make little homes for every protein in the membrane and allow it to perform its functions. Without cholesterol and saturated fats, our cells would not be able to communicate with each other or to transport various molecules into and out of the cell. As a result, our bodies would not be able to function the way they do.

The human brain is particularly rich in cholesterol: around 25 percent of all body cholesterol is accounted for by the brain. Every cell and every structure in the brain and the rest of our nervous system needs cholesterol, not only to build itself but also to accomplish its many functions. The developing brain and eyes of the fetus and a newborn infant require large amounts of cholesterol. If the fetus doesn't get enough cholesterol during development, the child may be born with a congenital abnormality called cyclopean eye.[1]

Human breast milk provides a lot of cholesterol. Not only that, mother's milk provides a specific enzyme to allow the baby's digestive tract to absorb almost 100 percent of that cholesterol, because the developing brain and eyes of an infant require large amounts of it. Children deprived of cholesterol in infancy may end up with poor eyesight and brain function. Manufacturers of infant formulas are aware of this fact, but following the anti-cholesterol dogma, they produce formulas with virtually no cholesterol in them.

VITAL BRAIN MATTER

One of the most abundant materials in the brain and the rest of our nervous system is a fatty substance called myelin. Myelin coats every nerve cell and every nerve fiber like the insulating cover around electric wires. Apart from insulation, it provides nourishment and protection for every tiny structure in our brain and the rest of the nervous system. People who start losing their myelin develop a condition called multiple sclerosis. Well, 20 percent of myelin is cholesterol. If you start interfering with the body's ability to produce cholesterol, you put the very structure of the brain and the rest of the nervous system under threat.

The synthesis of myelin in the brain is tightly connected with the synthesis of cholesterol. In my clinical experience, foods with high cholesterol and high animal fat content are an essential medicine for a person with multiple sclerosis.

One of the most wonderful abilities we humans are blessed with is the ability to remember things—our human memory. How do we form

memories? By our brain cells establishing connections with each other, called synapses. The more healthy synapses a person's brain can make, the more mentally able and intelligent that person is. Scientists have discovered that synapse formation is almost entirely dependent on cholesterol, which is produced by the brain cells in a form called apolipoprotein E. Without the presence of this factor we cannot form synapses, and hence we would not be able to learn or remember anything. Memory loss is one of the side effects of cholesterol-lowering drugs.

In my clinic, I see growing numbers of people with memory loss who have been taking cholesterol-lowering pills. Dr. Duane Graveline, former NASA scientist and astronaut, suffered such memory loss while taking his cholesterol pill. He managed to save his memory by stopping the pill and eating lots of cholesterol-rich foods. Since then he has described his experience in his book, *Lipitor: Thief of Memory, Statin Drugs and the Misguided War on Cholesterol*.

Dietary cholesterol in fresh eggs and other cholesterol-rich foods has been shown in scientific trials to improve memory in the elderly. In my clinical experience, any person with memory loss or learning problems needs to have plenty of these foods every single day in order to recover.

NECESSARY PRODUCT OF THE BODY

These foods give the body a hand in supplying cholesterol so it does not have to work as hard to produce its own. What a lot of people don't realize is that most cholesterol in the body does not come from food! The body produces cholesterol as it is needed. Scientific studies have conclusively demonstrated that cholesterol from food has no effect whatsoever on the level of our blood cholesterol. Why? Because cholesterol is such an essential part of our human physiology that the body has very efficient mechanisms to keep blood cholesterol at a certain level.

When we eat more cholesterol, the body produces less; when we eat less cholesterol, the body produces more. As a raw material for making cholesterol the body can use carbohydrates, proteins and fats, which means that your pasta and bread can be used for making cholesterol in the body. It has been estimated that, in an average person, about 85 percent of blood cholesterol is produced by the body, while only 15 percent comes from food. So, even if you religiously follow a completely cholesterol-free diet, you will still have a lot of cholesterol in your body. However, cholesterol-lowering drugs are a completely different matter! They interfere with the body's ability to produce cholesterol, and hence they do reduce the amount of cholesterol available for the body to use.

DANGERS OF LOW CHOLESTEROL

If we do not take cholesterol-lowering drugs, most of us don't have to worry about cholesterol. However, there are people whose bodies, for whatever reason, are unable to produce enough cholesterol. These people are prone to emotional instability and behavioral problems. Low blood cholesterol has been routinely recorded in criminals who have committed murder and other violent crimes, people with aggressive and violent personalities, people prone to suicide and people with aggressive social behavior and low self-control.

I would like to repeat what the late Oxford professor David Horrobin warned us about: "Reducing cholesterol in the population on a large scale could lead to a general shift to more violent patterns of behavior. Most of this increased violence would not result in death but in more aggression at work and in the family, more child abuse, more wife-beating and generally more unhappiness."

People whose bodies are unable to produce enough cholesterol do need to have plenty of foods rich in cholesterol in order to provide their organs with this essential-to-life substance.

What else does our body need all that cholesterol for?

ENDOCRINE SYSTEM

After the brain, the organs hungriest for cholesterol are our endocrine glands: adrenals and sex glands. They produce steroid hormones. Steroid hormones in the body are made from cholesterol: testosterone, progesterone, pregnenolone, androsterone, estrone, estradiol, corticosterone, aldosterone and others. These hormones accomplish a myriad of functions in the body, from regulation of our metabolism, energy

In my clinic, I see growing numbers of people with memory loss who have been taking cholesterol-lowering pills.

Without cholesterol we would not be able to have children because every sex hormone in our bodies is made from cholesterol.

production, mineral assimilation, brain, muscle and bone formation to behavior, emotions and reproduction. In our stressful modern lives we consume a lot of these hormones, leading to a condition called "adrenal exhaustion." This condition is diagnosed very often by naturopaths and other health practitioners. There are many herbal preparations on the market for adrenal exhaustion. However, the most important therapeutic measure is to provide your adrenal glands with plenty of dietary cholesterol.

Without cholesterol we would not be able to have children because every sex hormone in our bodies is made from cholesterol. A fair percentage of our infertility epidemic can be laid at the doorstep of the diet-heart hypothesis. The more eager we became to fight animal fats and cholesterol, the more problems with normal sexual development, fertility and reproduction we started to face. About a third of western men and women are infertile, and increasing numbers of our youngsters are growing up with abnormalities in their sex hormones. These abnormalities lead to many physical problems.

Recent research has "discovered" that eating full-cream dairy products cures infertility in women.[2] Researchers found that women who drink whole milk and eat high-fat dairy products are more fertile than those who stick to low-fat products. Study leader Dr. Jorge Chavarro, of the Harvard School of Public Health, emphasized: "Women wanting to conceive should examine their diet. They should consider changing low-fat dairy foods for high-fat dairy foods, for instance by swapping skimmed milk for whole milk and eating cream, not low-fat yoghurt."

THE LIVER AND VITAMIN REGULATION

One of the busiest organs in terms of cholesterol production in our bodies is the liver, which regulates the level of our blood cholesterol. The liver also puts a lot of cholesterol into bile production. Yes, bile is made out of cholesterol. Without bile we would not be able to digest and absorb fats and fat-soluble vitamins. Bile emulsifies fats; in other words, it mixes them with water, so that digestive enzymes can get to them. After it completes its mission, most of the bile gets reabsorbed in the digestive system and brought back to the liver for recycling. In fact, 95 percent of our bile is recycled because the building blocks of bile, one of which is cholesterol, are too precious for the body to waste. Nature doesn't do anything without good reason. This example of the careful recycling of cholesterol alone should have given us a good idea about its importance for the body!

Bile is essential for absorbing fat-soluble vitamins: vitamin A, vitamin D, vitamin K and vitamin E. We cannot live without these vitamins. Apart from ensuring that fat-soluble vitamins get digested and absorbed properly, cholesterol is the major building block of one of these vitamins: vitamin D. Vitamin D is made from the cholesterol in our skin when it is exposed to sunlight. In those times of the year when there isn't much sunlight, we can get this vitamin from cholesterol-rich foods: cod liver oil, fish, shellfish, butter, lard and egg yolks. Our recent misguided fears of the sun and avoidance of cholesterol-rich foods have created an epidemic of vitamin D deficiency in the western world.

DIETARY SOURCES OF CHOLESTEROL

1. Caviar is the richest source; it provides 588 mg of cholesterol per 100 grams. Obviously, this is not a common food for the majority of us, so let us have a look at the next item on the list.
2. Cod liver oil follows closely with 570 mg of cholesterol per 100 grams. There is no doubt that the cholesterol element of cod liver oil plays an important role in all the well-known health benefits of this time-honored health food.
3. Fresh egg yolk takes third place, with 424 mg of cholesterol per 100 gram. I would like to repeat: fresh egg yolk, not chemically mutilated egg powders (they contain chemically mutilated cholesterol)!
4. Butter provides a good 218 mg of cholesterol per 100 gram. We are talking about natural butter, not butter substitutes.
5. Cold-water fish and shellfish, such as salmon, sardines, mackerel and shrimps, provide good amounts of cholesterol, ranging from 81 mg to 173 mg per 100 gram. The proponents of low-cholesterol diets tell you to replace meats with fish. Obviously, they are not aware of the fact that fish is almost twice as rich in cholesterol as meat.
6. Lard provides 94 mg of cholesterol per 100 gram. Other animal fats follow.

Unfortunately, apart from sunlight and cholesterol-rich foods there is no other appropriate way to get vitamin D. Of course, there are supplements, but most of them contain vitamin D_2, which is made by irradiating mushrooms and other plants. This vitamin is not the same as the natural vitamin D. It does not work as effectively and it is easy to get a toxic level of it. In fact, almost all cases of vitamin D toxicity ever recorded were cases where this synthetic vitamin D_2 had been used. Toxicity is almost impossible with natural vitamin D obtained from sunlight or cholesterol-rich foods because the body knows how to deal with an excess of natural substances. What the body does not know how to deal with is an excess of synthetic vitamin D_2.

Vitamin D has been designed to work as a team with another fat-soluble vitamin: vitamin A. That is why foods rich in one tend to be rich in the other. So, by taking cod liver oil, for example, we can obtain both vitamins at the same time. As we grow older, our ability to produce vitamin D in the skin under sunlight is considerably diminished. Taking foods rich in vitamin D is therefore particularly important for older people. For the rest of us, sensible sunbathing is a wonderful, healthy and enjoyable way of getting a good supply of vitamin D.

Skin cancer, blamed on sunshine, is not caused by the sun. It is caused by *trans* fats from vegetable oils and margarine and other toxins stored in the skin. In addition, some of the sunscreens that people use contain chemicals that have been proven to cause skin cancer.

IMMUNE SYSTEM HEALTH

Cholesterol is essential for our immune system to function properly. Animal experiments and human studies have demonstrated that immune cells rely on cholesterol in fighting infections and repairing themselves after the fight. In addition, LDL-cholesterol (low-density lipoprotein cholesterol), the so-called "bad" cholesterol, directly binds and inactivates dangerous bacterial toxins, preventing them from doing any damage in the body. One of the most lethal toxins is produced by a widely spread bacterium, *Staphylococcus aureus*, which is the cause of MRSA (Methicillin-resistant *Staphylococcus aureus*), a common hospital infection. This toxin can literally dissolve red blood cells. However, it does not work in the presence of LDL-cholesterol. People who fall prey to this toxin have low blood cholesterol. It has been recorded that people with high levels of cholesterol are protected from infections; they are four times less likely to contract AIDS, they rarely get common colds and they recover from infections more quickly than people with "normal" or low blood cholesterol.

People with low blood cholesterol are prone to various infections, suffer from them longer and are more likely to die from an infection. A diet rich in cholesterol has been demonstrated to improve these people's ability to recover from infections. So, any person suffering from an acute or chronic infection needs to eat high-cholesterol foods to recover. Cod liver oil, the richest source of cholesterol (after caviar), has long been prized as the best remedy for the immune system. Those familiar with old medical literature will tell you that until the discovery of antibiotics, a common cure for tuberculosis was a daily mixture of raw egg yolks and fresh cream.

VARYING BLOOD CHOLESTEROL LEVELS

The question is, why do some people have more cholesterol in their blood than others, and why can the same person have different levels of

VITAMIN D DEFICIENCY

What does it mean for our bodies to be deficient in vitamin D? A long list of suffering:

- Diabetes, as vitamin D is essential for blood sugar control
- Heart disease
- Mental illness
- Auto-immune illness, such as rheumatoid arthritis, lupus, inflammatory bowel disease and multiple sclerosis
- Obesity
- Osteoarthritis
- Rickets and osteomalacia
- Muscle weakness and poor neuro-muscular coordination
- High blood pressure
- Cancer
- Chronic pain
- Poor immunity and susceptibility to infections
- Hyperparathyroidism, which manifests itself as osteoporosis, kidney stones, depression, aches and pains, chronic fatigue, muscle weakness and digestive abnormalities

cholesterol at different times of the day? Why is our level of cholesterol different in different seasons of the year? In winter it goes up and in the summer it goes down. Why is it that blood cholesterol goes through the roof in people after any surgery? Why does blood cholesterol go up when we have an infection? Why does it go up after dental treatment? Why does it go up when we are under stress? And why does it become normal when we are relaxed and feel well?

The answer to all these questions is this: cholesterol is a healing agent in the body. When the body has some healing jobs to do, it produces cholesterol and sends it to the site of the damage. Depending on the time of day, the weather, the season and our exposure to various environmental agents, the damage to various tissues in the body varies. As a result, the production of cholesterol in the body also varies.

Since cholesterol is usually discussed in the context of disease and atherosclerosis, let us look at the blood vessels. Their inside walls are covered by a layer of cells called the endothelium. Any damaging agent we are exposed to will finish up in our bloodstream, whether it is a toxic chemical, an infectious organism, a free radical or anything else. Once such an agent is in the blood, what is it going to attack first? The endothelium, of course. The endothelium immediately sends a message to the liver. Whenever our liver receives a signal that a wound has been inflicted upon the endothelium somewhere in our vascular system, it gets into gear and sends cholesterol to the site of the damage in a shuttle, called LDL-cholesterol. Because this cholesterol travels from the liver to the wound in the form of LDL, our "science," in its wisdom calls LDL "bad" cholesterol. When the wound heals and the cholesterol is removed, it travels back to the liver in the form of HDL-cholesterol (high-density lipoprotein cholesterol). Because this cholesterol travels away from the artery back to the liver, our misguided "science" calls it "good" cholesterol. This is like calling an ambulance traveling from the hospital to the patient a "bad ambulance," and the one traveling from the patient back to the hospital a "good ambulance."

But the situation has gotten even more ridiculous. The latest thing that our science has "discovered" is that not all LDL-cholesterol is so bad. Most of it is actually good. So, now we are told to call that part of LDL the "good bad cholesterol" and the rest of it the "bad bad cholesterol."

MARVELOUS HEALING AGENT

Why does the liver send cholesterol to the site of the injury? Because the body cannot clear the infection, remove toxic elements or heal the wound without cholesterol and fats. Any healing involves the birth, growth and functioning of thousands of cells: immune cells, endothelial cells and many others. As these cells, to a considerable degree, are made out of cholesterol and fats, they cannot form and grow without a good

JACK SPRATT - REVISITED
By Harvey J. Gardner

Jack Spratt could eat no fat,
His wife could eat no lean,
And so betwixt the two of them
They licked the platter clean.

Now that was when they both were young
And filled with youthful zest.
But soon the health of one went wrong.
Was it hers you've guessed?

"Imagine," prattled Mrs. Spratt,
"Jack's shed this mortal scene.
"And though he'd never chewed the fat
"His girth was most obscene."

So down she sat to a plate of fat.
No bread, no meat, no bean.
Upon her lap her purring cat,
Both sated and serene.

Said Mrs. Spratt who felt just fine,
"My folks loved fat like me,
"My mother died at ninety-nine
"And dad at a hundred and three."

So listen misinformèd folks,
Eat bacon, butter, lard.
Toss the whites and eat the yolks
And watch your abs get hard.

See the pallid veggie boy
And veggie girl who languish:
Their fats replaced by chips of soy
And a soy baloney sandwich.

Sat. . . fat. . . is where it's at
To revitalize our nation.
Go on-line, come on, let's chat
@ Weston A. Price Foundation.

supply of these substances. When the cells are damaged, they require cholesterol and fats to repair themselves. It is a scientific fact that any scar tissue in the body contains good amounts of cholesterol.[3]

Another scientific fact is that cholesterol acts as an antioxidant in the body, dealing with free radical damage.[4] Any wound in the body contains plenty of free radicals because the immune cells use these highly reactive molecules for destroying microbes and toxins. Excess free radicals have to be neutralized, and cholesterol is one of the natural substances that accomplishes this function.

When we have surgery, our tissues are cut and many small arteries, veins and capillaries get damaged. The liver receives a very strong signal from this damage, so it floods the body with LDL-cholesterol to clean and heal every little wound in our blood vessels. That is why blood cholesterol goes high after any surgical procedure. After dental treatment, in addition to the damage to the tissues, a lot of bacteria from the tooth and the gums finish up in the blood, attacking the inside walls of our blood vessels. Once again, the liver gets a strong signal from that damage and produces lots of healing cholesterol to deal with it, so the blood cholesterol goes up.

The same thing happens when we have an infection: LDL-cholesterol goes up to deal with the bacterial or viral attack.

Apart from the endothelium, our immune cells need cholesterol to function and to heal themselves after the fight with the infection.

Our stress hormones are made out of cholesterol in the body. Stressful situations increase our blood cholesterol levels because cholesterol is being sent to the adrenal glands for stress hormone production. Apart from that, when we are under stress, a storm of free radicals and other damaging biochemical reactions occur in the blood. So the liver works hard to produce and send out as much cholesterol as possible to deal with the free radical attack. In situations like this, your blood cholesterol will test high.

DAMAGE CONTROL

In short, when we have high blood cholesterol level, it means that the body is dealing with some kind of damage. The last thing we should do is interfere with this process! When the damage has been dealt with, the blood cholesterol will naturally go down. If we have an ongoing disease in the body that constantly inflicts damage, then the blood cholesterol will be permanently high. So, when a doctor finds high cholesterol in a patient, what this doctor should do is to look for the reason. The doctor should ask, "What is damaging the body so that the liver has to produce all that cholesterol to deal with the damage?" Unfortunately, instead of this sensible procedure, our doctors are trained to attack the cholesterol.

Many natural herbs, antioxidants and vitamins have an ability to reduce our blood cholesterol. How do they do that? By helping the body remove the damaging agents, be they free radicals, bacteria, viruses or toxins. As a result, the liver does not have to produce so much cholesterol to deal with the damage. At the same time, vitamins, minerals, antioxidants, herbs and other natural remedies help to heal the wound. When the wound heals there is no need for high levels of cholesterol any more, so the body removes it in the form of HDL-cholesterol or so-called "good" cholesterol. That is why herbs, vitamins, antioxidants and other natural remedies increase the level of HDL-cholesterol in the blood.

In conclusion, cholesterol is one of the most important substances in the body. We cannot live without it, let alone function well. The pernicious diet-heart hypothesis has vilified this essential substance. Unfortunately, this hypothesis has served many commercial and political interests far too well, so they ensure its long survival. However, the life of the diet-heart hypothesis is coming to an end as we become aware that cholesterol has been mistakenly blamed for the crime just because it was found at the scene.

Dr. Campbell-McBride runs the Cambridge Nutrition Clinic where she specializes in using nutritional approaches as a treatment for learning disabilities and other mental disorders. She is recognized as one of the world's leading experts in treating children and adults with these conditions, as well as children and adults with digestive and immune disorders. She is the author of Gut And Psychology Syndrome*:* Natural Treatment for Autism, ADHD, Dyslexia, Dysopraxia, Depression and Schizophrenia. *This article is a chapter from her new book,* Put Your Heart in Your Mouth! Natural Treatments for Atherosclerosis, Angina, Heart Attack, High Blood Pressure, Stroke, Arrhythmia and Peripheral Vascular Disease. *Her books are available from Gut Health, Inc., www.guthealth.info.*

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The Benefits of High Cholesterol

By Uffe Ravnskov, MD, PhD

People with high cholesterol live the longest. This statement seems so incredible that it takes a long time to clear one´s brainwashed mind to fully understand its importance. Yet the fact that people with high cholesterol live the longest emerges clearly from many scientific papers. Consider the finding of Dr. Harlan Krumholz of the Department of Cardiovascular Medicine at Yale University, who reported in 1994 that old people with low cholesterol died twice as often from a heart attack as did old people with a high cholesterol.[1] Supporters of the cholesterol campaign consistently ignore his observation, or consider it as a rare exception, produced by chance among a huge number of studies finding the opposite.

But it is not an exception; there are now a large number of findings that contradict the lipid hypothesis. To be more specific, most studies of old people have shown that high cholesterol is not a risk factor for coronary heart disease. This was the result of my search in the Medline database for studies addressing that question.[2] Eleven studies of old people came up with that result, and a further seven studies found that high cholesterol did not predict all-cause mortality either.

Dr. Ravnskov is the author of *The Cholesterol Myths* and chairman of The International Network of Cholesterol Skeptics (thincs.org)

Now consider that more than 90 percent of all cardiovascular disease occurs in people above age 60 and that almost all studies have found that high cholesterol is not a risk factor for women.[2] This means that high cholesterol is only a risk factor for less than 10 percent of mankind, namely for young and middle-aged men.

But there is more comfort for those who have high cholesterol; six of the studies found that total mortality was *inversely* associated with either total or LDL-cholesterol, or both. This means that it is actually much better to have high than to have low cholesterol if you want to live to be very old.

HIGH CHOLESTEROL PROTECTS AGAINST INFECTION

Many studies have found that low cholesterol is in certain respects worse than high cholesterol. For instance, in 19 large studies of more than 68,000 deaths, reviewed by Professor David R. Jacobs and his co-workers from the Division of Epidemiology at the University of Minnesota, low cholesterol predicted an increased risk of dying from gastrointestinal and respiratory diseases.[3]

Most gastrointestinal and respiratory diseases have an infectious origin. Therefore, a relevant question is whether it is the infection that lowers cholesterol or the low cholesterol that predisposes to infection? To answer this question Professor Jacobs and his group, together with Dr. Carlos Iribarren, followed more than 100,000 healthy individuals in the San Francisco area for fifteen years. At the end of the study those who had low cholesterol at the start of the study had more often been admitted to the hospital because of an infectious disease.[4,5] This finding cannot be explained away with the argument that the infection had caused cholesterol to go down, because how could low cholesterol, recorded when these people had no evidence of infection, be caused by a disease they had not yet encountered? Isn´t it more likely that low cholesterol in some way made them more vulnerable to infection, or that high cholesterol protected those who did not become infected? Much evidence exists to support that interpretation.

LOW CHOLESTEROL AND HIV/AIDS

Young, unmarried men with a previous sexually transmitted disease or liver disease run a much greater risk of becoming infected with HIV virus than other people. The Minnesota researchers, now led by Dr. Ami Claxton, followed such individuals for 7-8 years. After having excluded those who became HIV-positive during the first four years, they ended up with a group of 2446 men. At the end of the study, 140 of these people tested positive for HIV; those who had low cholesterol at the beginning of the study were twice as likely to test postitive for HIV than those with the highest cholesterol.[6]

Similar results come from a study of the participants in the famous MRFIT study, including more than 300,000 young and middle-aged men. The data showed that 16 years after the first cholesterol analysis the number of men whose cholesterol was lower than 160 and who had died from AIDS was four times higher than the number of men who had died from AIDS with a cholesterol above 240.[7]

CHOLESTEROL AND CHRONIC HEART FAILURE

Heart disease may lead to a weakening of the heart muscle. A weak heart means that less blood and therefore less oxygen is delivered to the arteries. To compensate for the decreased power, the heart beat goes up, but in cases of severe heart failure the heart's extra effort is not sufficient. Patients with severe heart failure become short of breath because too little oxygen is delivered to the tissues, the pressure in their veins increases because the heart cannot deliver the blood away from the heart with sufficient power, and they become edematous, meaning that fluid accumulates in the legs and in serious cases also in the lungs and other parts of the body. This condition is called congestive or chronic heart failure.

There are many indications that bacteria or other microorganisms play an important role in chronic heart failure. For instance, patients with severe chronic heart failure have high levels of endotoxin and various types of cytokines in their blood. Endotoxin, also named lipopolysaccharide, is the most toxic substance produced by gram-negative bacteria such as *Escherichia coli*, *Klebsiella*, *Salmonella*, *Serratia* and *Pseudomo-*

At the end of the study those who had low cholesterol at the start of the study had more often been admitted to the hospital because of an infectious disease.

It is interesting to note that much evidence supports the theory that people born with very high cholesterol, so-called familial hypercholesteroemia, are protected against infection.

nas. Cytokines are hormones secreted by white blood cells in their battle against microorganisms; high levels of cytokines in the blood indicate that inflammatory processes are going on somewhere in the body.

The role of infection in chronic heart failure has been studied by Dr. Mathias Rauchhaus and his team at the Medical Department, Martin-Luther-University in Halle, Germany (Universitätsklinik und Poliklinik für Innere Medizin III, Martin-Luther-Universität, Halle). They found that the strongest predictor of death for patients with chronic heart failure was the concentration of cytokines in the blood, in particular in patients with heart failure due to coronary heart disease.[8] To explain their finding they suggested that bacteria from the gut may more easily penetrate into the tissues when the pressure in the abdominal veins is increased because of heart failure. In accordance with this theory, they found more endotoxin in the blood of patients with congestive heart failure and edema than in patients with non-congestive heart failure without edema, and endotoxin concentrations decreased significantly when the heart's function was improved by medical treatment.[9]

A simple way to test the functional state of the immune system is to inject antigens from microorganisms that most people have been exposed to under the skin. If the immune system is normal, an induration (hard spot) will appear about 48 hours later at the place of the injection. If the induration is very small, with a diameter of less than a few millimeters, this indicates the presence of "anergy," a reduction in or failure of response to recognize antigens. In accordance, anergy has been found associated with an increased risk of infection and mortality in healthy elderly individuals, in surgical patients and in heart transplant patients.[10]

Dr. Donna Vredevoe and her group from the School of Nursing and the School of Medicine, University of California at Los Angeles tested more than 200 patients with severe heart failure with five different antigens and followed them for twelve months. The cause of heart failure was coronary heart disease in half of them and other types of heart disease (such as congenital or infectious valvular heart disease, various cardiomyopathies and endocarditis) in the rest. Almost half of all the patients were anergic, and those who were anergic and had coronary heart disease had a much higher mortality than the rest.[10]

Now to the salient point: to their surprise the researchers found that mortality was higher, not only in the patients with anergy, but also in the patients with the lowest lipid values, including total cholesterol, LDL-cholesterol and HDL-cholesterol as well as triglycerides.

The latter finding was confirmed by Dr. Rauchhaus, this time in co-operation with researchers at several German and British university hospitals. They found that the risk of dying for patients with chronic heart failure was strongly and inversely associated with total cholesterol, LDL-cholesterol and also triglycerides; those with high lipid values lived much longer than those with low values.[11,12]

Other researchers have made similar observations. The largest study has been performed by Professor Gregg C. Fonorow and his team at the UCLA Department of Medicine and Cardiomyopathy Center in Los Angeles.[13] The study, led by Dr. Tamara Horwich, included more than a thousand patients with severe heart failure. After five years, 62 percent of the patients with cholesterol below 129 mg/l had died, but only half as many of the patients with cholesterol above 223 mg/l.

When proponents of the cholesterol hypothesis are confronted with findings showing a bad outcome associated with low cholesterol—and there are many such observations—they usually argue that severely ill patients are often malnourished, and malnourishment is therefore said to cause low cholesterol. However, the mortality of the patients in this study was independent of their degree of nourishment; low cholesterol predicted early mortality whether the patients were malnourished or not.

SMITH-LEMLI-OPITZ SYNDROME

It is interesting to note that much evidence supports the theory that people born with very high cholesterol, so-called familial hypercholesterolemia, are protected against infection (see sidebar). But if inborn high cholesterol protects against infection, inborn low cholesterol should have the opposite effect. Indeed, this seems to be true.

FAMILIAL HYPERCHOLESTEROLEMIA - NOT AS RISKY AS YOU MAY THINK

Many doctors believe that most patients with familial hypercholesterolemia (FH) die from CHD at a young age. Obviously, they do not know the surprising finding of the Scientific Steering Committee at the Department of Public Health and Primary Care at Ratcliffe Infirmary in Oxford, England. For several years, these researchers followed more than 500 FH patients between the ages of 20 and 74 and compared patient mortality during this period with that of the general population.

During a three- to four-year period, six of 214 FH patients below age 40 died from CHD. This may not seem particularly frightening but as it is rare to die from CHD before the age of 40, the risk for these FH patients was almost 100 times that of the general population.

During a four- to five-year period, eight of 237 FH patients between ages 40 and 59 died, which was five times more than the general population. But during a similar period of time, only one of 75 FH patients between the ages of 60 and 74 died from CHD, when the expected number was two.

If these results are typical for FH, you could say that between ages 20 and 59, about 3 percent of the patients die from CHD, and between ages 60 and 74, less than 2 percent die, in both cases during a period of 3-4 years. The authors stressed the fact that the patients had been referred for treatment because of a personal or family history of premature vascular disease and therefore were at a particularly high risk for CHD. Most patients with FH in the general population are unrecognized and untreated. Had the patients studied been representative for all FH patients, their prognosis would probably have been even better.

This view was recently confirmed by Dr. Eric Sijbrands and his coworkers from various medical departments in Amsterdam and Leiden, Netherlands. Out of a large group they found three individuals with very high cholesterol. A genetic analysis confirmed the diagnosis of FH and by tracing their family members backward in time, they came up with a total of 412 individuals. The coronary and total mortality of these members was compared with the mortality of the general Dutch population.

The striking finding was that those who lived during the 19th and early 20th century had normal mortality and lived a normal life span. In fact, those living in the 19th century had a *lower* mortality than the general population. After 1915 the mortality rose to a maximum between 1935 and 1964, but even at the peak, mortality was less than twice as high as in the general population.

Again, very high cholesterol levels alone do not lead to a heart attack. In fact, high cholesterol may even be protective against other diseases. This was the conclusion of Dr. Sijbrands and his colleagues. As support they cited the fact that genetically modified mice with high cholesterol are protected against severe bacterial infections.

"Doctor, don't be afraid because of my high cholesterol." These were the words of a 36-year-old lawyer who visited me for the first time for a health examination. And indeed, his cholesterol was high, over 400 mg/dl.

"My father's cholesterol was even higher," he added. "But he lived happily until he died at age 79 from cancer. And his brother, who also had FH, died at age 83. None of them ever complained of any heart problems." My "patient" is now 53, his brother is 56 and his cousin 61. All of them have extremely high cholesterol values, but none of them has any heart troubles, and none of them has ever taken cholesterol-lowering drugs.

So, if you happen to have FH, don't be too anxious. Your chances of surviving are pretty good, even surviving to old age.

Scientific Steering Committee on behalf of the Simon Broome Register Group. Risk of fatal coronary heart disease in familial hypercholesterolaemia. *British Medical Journal* 303, 893-896, 1991; Sijbrands EJG and others. Mortality over two centuries in large pedigree with familial hypercholesterolaemia: family tree mortality study. British Medical Journal 322, 1019-1023, 2001.

From *The Cholesterol Myths* by Uffe Ravnvskov, MD, PhD, NewTrends Publishing, pp 64-65.

Children with the Smith-Lemli-Opitz syndrome produce very little cholesterol because the enzyme that is necessary for the last step in the body's synthesis of cholesterol does not function properly. Most children with this syndrome are either stillborn or they die early because of serious central nervous system malformations. Those who survive are imbecile, they have extremely low cholesterol and suffer from frequent and severe infections. However, if their diet is supplemented with pure cholesterol or extra eggs, their cholesterol goes up and their bouts of infection become less serious and less frequent.[14]

LABORATORY EVIDENCE

Laboratory studies are crucial for learning more about the mechanisms by which the lipids exert their protective function. One of the first to study this phenomenon was Dr Sucharit Bhakdi from the Institute of Medical Microbiology, University of Giessen (*Institut für Medizinsche Mikrobiologie, Justus-Liebig-Universität Gießen*), Germany along with his team of researchers

from various institutions in Germany and Denmark.[15]

Staphylococcus aureus a-toxin is the most toxic substance produced by strains of the disease-promoting bacteria called staphylococci. It is able to destroy a wide variety of human cells, including red blood cells. For instance, if minute amounts of the toxin are added to a test tube with red blood cells dissolved in 0.9 percent saline, the blood is hemolyzed, that is the membranes of the red blood cells burst and hemoglobin from the interior of the red blood cells leaks out into the solvent. Dr. Bhakdi and his team mixed purified a-toxin with human serum (not just the blood cells alone, but the cells and the fluid in which the blood cells reside) and saw that 90 percent of its hemolyzing effect disappeared. By various complicated methods they identified the protective substance as LDL, the carrier of the so-called bad cholesterol. In accordance with these findings, no hemolysis occurred when they mixed a-toxin with purified human LDL, whereas HDL or other plasma constituents were ineffective in this respect.

Dr. Willy Flegel and his co-workers at the Department of Transfusion Medicine, University of Ulm, and the Institute of Immunology and Genetics at the German Cancer Research Center in Heidelberg, Germany (*DRK-Blutspendezentrale und Abteilung für Transfusionsmedizin, Universität Ulm, und Deutsches Krebsforschungszentrum, Heidelberg*) studied endotoxin in another way.[16] As mentioned, one of the effects of endotoxin is the stimulation of white blood cells to produce cytokines. The German researchers found that the cytokine-stimulating effect of endotoxin on the white blood cells disappeared almost completely if the endotoxin was mixed with human serum for 24 hours before they added the white blood cells to the test tubes. In a subsequent study[17] they found that purified LDL from patients with familial hypercholesterolemia had the same inhibitory effect as the serum.

LDL may not only bind and inactivate dangerous bacterial toxins; it seems to have a direct beneficial influence on the immune system also, possibly explaining the observed relationship between low cholesterol and various chronic diseases. This was the starting point for a study by Professor Matthew Muldoon and his team at the University of Pittsburgh, Pennsylvania. They studied healthy young and middle-aged men and found that the total number of white blood cells and the number of various types of white blood cells were significantly lower in men with LDL-cholesterol below 160 mg/dl (mean 88.3 mg/l), than in men with LDL-cholesterol above 160 mg/l (mean 185.5 mg/l).[18] The researchers cautiously concluded that there were immune system differences between men with low and high cholesterol, but that it was too early to state whether these differences had any importance for human health. Now, seven years later with many of the results discussed here, we can state with confidence that the immune-supporting properties of LDL-cholesterol do indeed play an important role in human health.

LDL may not only bind and inactivate dangerous bacterial toxins; it seems to have a direct beneficial influence on the immune system also, possibly explaining the observed relationship between low cholesterol and various chronic diseases.

ANIMAL EXPERIMENTS

The immune systems in various mammals including human beings have many similarities. Therefore, it is interesting to see what experiments with rats and mice can tell us. Professor Kenneth Feingold at the Department of Medicine, University of California, San Francisco, and his group have published several interesting results from such research. In one of them they lowered LDL-cholesterol in rats by giving them either a drug that prevents the liver from secreting lipoproteins, or a drug that increases their disappearance. In both models, injection of endotoxin was followed by a much higher mortality in the low-cholesterol rats than in normal rats. The high mortality was not due to the drugs because if the drug-treated animals were injected with lipoproteins just before the injection of endotoxin, their mortality was reduced to normal.[19]

Dr. Mihai Netea and his team from the Departments of Internal and Nuclear Medicine at the University Hospital in Nijmegen, The Netherlands, injected purified endotoxin into normal mice and into mice with familial hypercholesterolemia that had LDL-cholesterol four times higher than normal. Whereas all normal mice died, they had to inject eight times as much endotoxin to kill the mice with familial hypercholesterolemia. In another experiment they injected live bacteria and found that twice as many mice with familial hypercholesterolemia survived compared with normal mice.[20]

OTHER PROTECTING LIPIDS

As seen from the above, many of the roles played by LDL-cholesterol are shared by HDL. This should not be too surprising considering the fact that high HDL-cholesterol is associated with cardiovascular health and longevity. But there is more.

Triglycerides, molecules consisting of three fatty acids linked to glycerol, are insoluble in water and are therefore carried through the blood inside lipoproteins, just as cholesterol. All lipoproteins carry triglycerides, but most of them are carried by a lipoprotein named VLDL (very low-density lipoprotein) and by chylomicrons, a mixture of emulsified triglycerides appearing in large amounts after a fat-rich meal, particularly in the blood that flows from the gut to the liver.

For many years it has been known that sepsis, a life-threatening condition caused by bacterial growth in the blood, is associated with a high level of triglycerides. The serious symptoms of sepsis are due to endotoxin, most often produced by gut bacteria. In a number of studies, Professor Hobart W. Harris at the Surgical Research Laboratory at San Francisco General Hospital and his team found that solutions rich in triglycerides but with practically no cholesterol were able to protect experimental animals from the toxic effects of endotoxin and they concluded that the high level of triglycerides seen in sepsis is a normal immune response to infection.[21] Usually the bacteria responsible for sepsis come from the gut. It is therefore fortunate that the blood draining the gut is especially rich in triglycerides.

EXCEPTIONS

So far, animal experiments have confirmed the hypothesis that high cholesterol protects against infection, at least against infections caused by bacteria. In a similar experiment using injections of *Candida albicans*, a common fungus, Dr. Netea and his team found that mice with familial hypercholesterolemia died more easily than normal mice.[22] Serious infections caused by *Candida albicans* are rare in normal human beings; however, they are mainly seen in patients treated with immunosuppressive drugs, but the finding shows that we need more knowledge in this area. However, the many findings mentioned above indicate that the protective effects of the blood lipids against infections in human beings seem to be greater than any possible adverse effects.

CHOLESTEROL AS A RISK FACTOR

Most studies of young and middle-aged men have found high cholesterol to be a risk factor for coronary heart disease, seemingly a contradiction to the idea that high cholesterol is protective. Why is high cholesterol a risk factor in young and middle-aged men? A likely explanation is that men of that age are often in the midst of their professional career. High cholesterol may therefore reflect mental stress, a well-known cause of high cholesterol and also a risk factor for heart disease. Again, high cholesterol is not necessarily the direct cause but may only be a marker. High cholesterol in young and middle-aged men could, for instance, reflect the body's need for more cholesterol because cholesterol is the building material of many stress hormones. Any possible protective effect of high cholesterol may therefore be counteracted by the negative influence of a stressful life on the vascular system.

High cholesterol in young and middle-aged men could, for instance, reflect the body's need for more cholesterol because cholesterol is the building material of many stress hormones.

RESPONSE TO INJURY

In 1976 one of the most promising theories about the cause of atherosclerosis was the Response-to-Injury Hypothesis, presented by Russell Ross, a professor of pathology, and John Glomset, a professor of biochemistry and medicine at the Medical School, University of Washington in Seattle.[23,24] They suggested that atherosclerosis is the consequence of an inflammatory process, where the first step is a localized injury to the thin layer of cells lining the inside of the arteries, the intima. The injury causes inflammation and the raised plaques that form are simply healing lesions.

Their idea is not new. In 1911, two American pathologists from the Pathological Laboratories, University of Pittsburgh, Pennsylvania, Oskar Klotz and M.F. Manning, published a summary of their studies on human arteries and concluded that "there is every indication that the production of tissue in the intima is the result of a direct irritation of that tissue by the presence of infection or toxins or the stimulation by the products of a primary degeneration in that layer."[25] Other researchers have presented similar theories.[26]

Researchers have proposed many potential causes of vascular injury, including mechanical stress, exposure to tobacco fumes, high LDL-cholesterol, oxidized cholesterol, homocysteine, the metabolic consequences of diabetes, iron overload, copper deficiency, deficiencies of vitamins A and D, consumption of *trans* fatty acids, microorganisms and many more. With one exception, there is evidence to support roles for all of these factors, but the degree to which each of them participates remains uncertain. The exception is of course LDL-cholesterol. Much research allows us to exclude high LDL-cholesterol from the list. Whether we look directly with the naked eye at the inside of the arteries at autopsy, or we do it indirectly in living people using x-rays, ultrasound or electron beams, no association worth mentioning has ever been found between the amount of lipid in the blood and the degree of atherosclerosis in the arteries. Also, whether cholesterol goes up or down, by itself or due to medical intervention, the changes of cholesterol level have never been followed by parallel changes in the atherosclerotic plaques; there is no dose-response.

Proponents of the cholesterol campaign often claim that the trials indeed have found dose-response, but here they refer to calculations between the mean changes of the different trials with the outcome of the whole treatment group. However, true dose-response demands that the individual changes of the putative causal factor are followed by parallel, individual changes of the disease outcome, and this has never occurred in the trials where researchers have calculated true dose-response.

A detailed discussion of the many factors accused of harming the arterial endothelium is beyond the scope of this article. However, the protective role of the blood lipids against infections obviously demands a closer look at the alleged role of one of the alleged causes, the microorganisms.

During the weeks preceding an acute cardiovascular attack many patients have reported a bacterial or viral infection.

IS ATHEROSCLEROSIS AN INFECTIOUS DISEASE?

For many years scientists have suspected that viruses and bacteria, in particular cytomegalovirus and *Chlamydia pneumonia* (also named TWAR bacteria) participate in the development of atherosclerosis.

Research within this area has exploded during the last decade and by January 2004, at least 200 reviews of the issue had been published in medical journals. Due to the widespread preoccupation with cholesterol and other lipids, there has been little general interest in the subject, however, and few doctors know much about it. Here I shall mention some of the most interesting findings.[26]

Electron microscopy, immunofluorescence microscopy and other advanced techniques have allowed us to detect microorganisms and their DNA in the atherosclerotic lesions in a large proportion of patients. Bacterial toxins and cytokines, hormones secreted by the white blood cells during infections, are seen more often in the blood from patients with recent heart disease and stroke, in particular during and after an acute cardiovascular event, and some of them are strong predictors of cardiovascular disease. The same is valid for bacterial and viral antibodies, and a protein secreted by the liver during infections, named C-reactive protein (CRP), is a much stronger risk factor for coronary heart disease than cholesterol.

Clinical evidence also supports this theory. During the weeks preceding an acute cardiovascular attack many patients have reported a bacterial or viral infection. For instance, Dr. Armin J. Grau from the Department of Neurology at the University of Heidelberg and his team asked 166 patients with acute stroke, 166 patients hospitalized for other neurological diseases and 166 healthy individuals matched individually for age and sex about recent infectious disease. Within the first week before the stroke, 37 of the stroke patients, but only 14 of the control individuals had suffered an infectious disease. In half of the patients the infection was of bacterial origin, in the other half of viral origin.[27]

Similar observations apply to patients with acute myocardial infarction (heart attack). For instance, Dr. Kimmo J. Mattila at the Department of Medicine, Helsinki University Hospital, Finland, found that 11 of 40 male patients with an acute heart attack before age 50 had an influenza-like infection with fever within 36 hours prior to admittance to hospital, but only 4 out of 41 patients with chronic coronary disease

(such as recurrent angina or pervious myocardial infarction) and 4 out of 40 control individuals without chronic disease randomly selected from the general population.[28]

Attempts have been made to prevent cardiovascular disease by treatment with antibiotics. In five trials treatment of patients with coronary heart disease using azithromyzin or roxithromyzin, antibiotics that are effective against *Chlamydia pneumonia*,yielded successful results; a total of 104 cardiovascular events occurred among the 412 non-treated patients, but only 61 events among the 410 patients in the treatment groups.[28a-e] In one further trial a significant decreased progression of atherosclerosis in the carotid arteries occurred with antibiotic treatment.[28f] However, in four other trials,[30a-d] one of which included more than 7000 patients,[28d] antibiotic treatment had no significant effect.

The reason for these inconsistent results may be that the treatment was too short (in one of the trials treatment lasted only five days). Also, *Chlamydia pneumonia,* the TWAR bacteria, can only propagate inside human cells and when located in white blood cells they are resistant to antibiotics.[31] Treatment may also have been ineffective because the antibiotics used have no effect on viruses. In this connection it is interesting to mention a controlled trial performed by Dr. Enrique Gurfinkel and his team from Fundación Favaloro in Buenos Aires, Argentina.[32] They vaccinated half of 301 patients with coronary heart disease against influenza, a viral disease. After six months 8 percent of the control patients had died, but only 2 percent of the vaccinated patients. It is worth mentioning that this effect was much better than that achieved by any statin trial, and in a much shorter time.

DOES HIGH CHOLESTEROL PROTECT AGAINST CARDIOVASCULAR DISEASE?

Apparently, microorganisms play a role in cardiovascular disease. They may be one of the factors that start the process by injuring the arterial endothelium. A secondary role may be inferred from the association between acute cardiovascular disease and infection. The infectious agent may preferably become located in parts of the arterial walls that have been previously damaged by other agents, initiating local coagulation and the creation of a thrombus (clot) and in this way cause obstruction of the blood flow. But if so, high cholesterol may protect against cardiovascular disease instead of being the cause!

HIGH CHOLESTEROL TO THE RESCUE

In any case, the diet-heart idea, with its demonization of high cholesterol, is obviously in conflict with the idea that high cholesterol protects against infections. Both ideas cannot be true. Let me summarize the many facts that conflict with the idea that high cholesterol is bad.

If high cholesterol were the most important cause of atherosclerosis, people with high cholesterol would be more atherosclerotic than people with low cholesterol. But as you know by now this is very far from the truth.

If high cholesterol were the most important cause of atherosclerosis, lowering of cholesterol would influence the atherosclerotic process in proportion to the degree of its lowering. But as you know by now, this does not happen.

If high cholesterol were the most important cause of cardiovascular disease, it would be a risk factor in all populations, in both sexes, at all ages, in all disease categories, and for both heart disease and stroke. But as you know by now, this is not the case

I have only two arguments for the idea that high cholesterol is good for the blood vessels, but in contrast to the arguments claiming the opposite they are very strong. The first one stems from the statin trials. If high cholesterol were the most important cause of cardiovascular disease, the greatest effect of statin treatment would have been seen in patients with the highest cholesterol, and in patients whose cholesterol was lowered the most. Lack of dose-response cannot be attributed

RISK FACTOR

There is one risk factor that is known to be certain to cause death. It is such a strong risk factor that it has a 100 percent mortality rate. Thus I can guarantee that if we stop this risk factor, which would take no great research and cost nothing in monetary terms, within a century human deaths would be completely eliminated. This risk factor is called "Life."

Barry Groves, second-opinions.com

It is difficult to explain away the fact that during the period of life in which most cardiovascular disease occurs and from which most people die (and most of us die from cardiovascular disease), high cholesterol occurs most often in people with the lowest mortality.

to the knowledge that the statins have other effects on plaque stabilization, as this would not have masked the effect of cholesterol-lowering considering the pronounced lowering that was achieved. On the contrary, if a drug that effectively lowers the concentration of a molecule assumed to be harmful to the cardiovascular system and at the same time exerts several beneficial effects on the same system, a pronounced dose-response should be seen.

On the other hand, if high cholesterol has a protective function, as suggested, its lowering would counterbalance the beneficial effects of the statins and thus work against a dose-response, which would be more in accord with the results from the various trials.

I have already mentioned my second argument, but it can't be said too often: *High cholesterol is associated with longevity in old people*. It is difficult to explain away the fact that during the period of life in which most cardiovascular disease occurs and from which most people die (and most of us die from cardiovascular disease), high cholesterol occurs most often in people with the lowest mortality. How is it possible that high cholesterol is harmful to the artery walls and causes fatal coronary heart disease, the most common cause of death, if those whose cholesterol is the highest, live longer than those whose cholesterol is low?

To the public and the scientific community I say, "Wake up!"

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The Dangers of Statin Drugs

What You Haven't Been Told About Popular Cholesterol-Lowering Medicines

By Sally Fallon and Mary G. Enig, PhD

The authors wish to thank the members of The International Network of Cholesterol Skeptics (thincs.org) for their invaluable contribution to this article.

Hypercholesterol is *the* health issue of the 21st century. It is actually an invented disease, a "problem" that emerged when health professionals learned how to measure cholesterol levels in the blood. High cholesterol exhibits no outward signs—unlike other conditions of the blood, such as diabetes or anemia, diseases that manifest telltale symptoms like thirst or weakness, hypercholesterolemia requires the services of a physician to detect its presence. Many people who feel perfectly healthy suffer from high cholesterol—in fact, feeling good is actually a symptom of high cholesterol!

Doctors who treat this new disease must first convince their patients that they are sick and need to take one or more expensive drugs for the rest of their lives, drugs that require regular checkups and blood tests. But such doctors do not work in a vacuum—their efforts to convert healthy people into patients are bolstered by the full weight of the US government, the media and the medical establishment, agencies that have worked in concert to disseminate the cholesterol dogma and convince the population that high cholesterol is the forerunner of heart disease and possibly other diseases as well.

The penance is a lifetime of cholesterol-lowering medications along with a boring lowfat diet. But why wait until you have a heart attack? Since we all labor under the stigma of original sin, we are all candidates for treatment.

Who suffers from hypercholesterolemia? Peruse the medical literature of 25 or 30 years ago and you'll get the following answer: any middle-aged man whose cholesterol is over 240 with other risk factors, such as smoking or overweight. After the Cholesterol Consensus Conference in 1984, the parameters changed; anyone (male or female) with cholesterol over 200 could receive the dreaded diagnosis and a prescription for pills. Recently that number has been moved down to 180. If you have had a heart attack, you get to take cholesterol-lowering medicines even if your cholesterol is already very low—after all, you have committed the sin of having a heart attack so your cholesterol must therefore be too high. The penance is a lifetime of cholesterol-lowering medications along with a boring lowfat diet. But why wait until you have a heart attack? Since we all labor under the stigma of original sin, we are all candidates for treatment. Current edicts stipulate cholesterol testing and treatment for young adults and even children.

The drugs that doctors use to treat the new disease are called statins—sold under a variety of names including Lipitor (atorvastatin), Zocor (simvastatin), Mevacor (lovastatin) and Pravachol (pravastatin).

HOW STATINS WORK

The diagram on page 57 illustrates the pathways involved in cholesterol production. The process begins with acetyl-CoA, a two-carbon molecule sometimes referred to as the "building block of life." Three acetyl-CoA molecules combine to form six-carbon hydroxymethyl glutaric acid (HMG). The step from HMG to mevalonate requires an enzyme, HMG-CoA reductase. Statin drugs work by inhibiting this enzyme—hence the formal name of HMG-CoA reductase inhibitors. Herein lies the potential for numerous side effects, because statin drugs inhibit not just the production of cholesterol, but a whole family of intermediary substances, many if not all of which have important biochemical functions in their own right.

Consider the findings of pediatricians at the University of California, San Diego who published a description of a child with an hereditary defect of mevalonic kinase, the enzyme that facilitates the next step beyond HMG-CoA reductase.[1] The child was mentally retarded, microcephalic (very small head), small for his age, profoundly anemic, acidotic and febrile. He also had cataracts. Predictably, his cholesterol was consistently low—70-79 mg/dl. He died at the age of 24 months. The child represents an extreme example of cholesterol inhibition, but his case illuminates the possible consequences of taking statins in strong doses or for a lengthy period of time—depression of mental acuity, anemia, acidosis, frequent fevers and cataracts.

Cholesterol is one of three end products in the mevalonate chain. The two others are ubiquinone and dilochol. Ubiquinone or Co-Enzyme Q_{10} is a critical cellular nutrient biosynthesized in the mitochondria. It plays a role in ATP production in the cells and functions as an electron carrier to cytochrome oxidase, our main respiratory enzyme. The heart requires high levels of Co-Q_{10}. A form of Co-Q_{10} called ubiquinone is found in all cell membranes where it plays a role in maintaining membrane integrity so critical to nerve conduction and muscle integrity. Co-Q_{10} is also vital to the formation of elastin and collagen. Side effects of Co-Q_{10} deficiency include muscle wasting leading to weakness and severe back pain, heart failure (the heart is a muscle!), neuropathy and inflammation of the tendons and ligaments, often leading to rupture.

Dolichols also play a role of immense importance. In the cells they direct various proteins manufactured in response to DNA directives to their proper targets, ensuring that the cells respond correctly to genetically programmed instruction. Thus statin drugs can lead to unpredictable chaos on the cellular level, much like a computer virus that wipes out certain pathways or files.

Squalene, the immediate precursor to cholesterol, has anti-cancer effects, according to research.

The fact that some studies have shown that statins can prevent heart disease, at least in the short term, is most likely explained not by the inhibition of cholesterol production but because they block the creation of mevalonate. Reduced amounts of mevalonate seem to make smooth muscle cells less active, and platelets less able to produce thromboxane. Atherosclerosis begins with the growth of smooth muscle cells in side

artery walls and thromboxane is necessary for blood clotting.

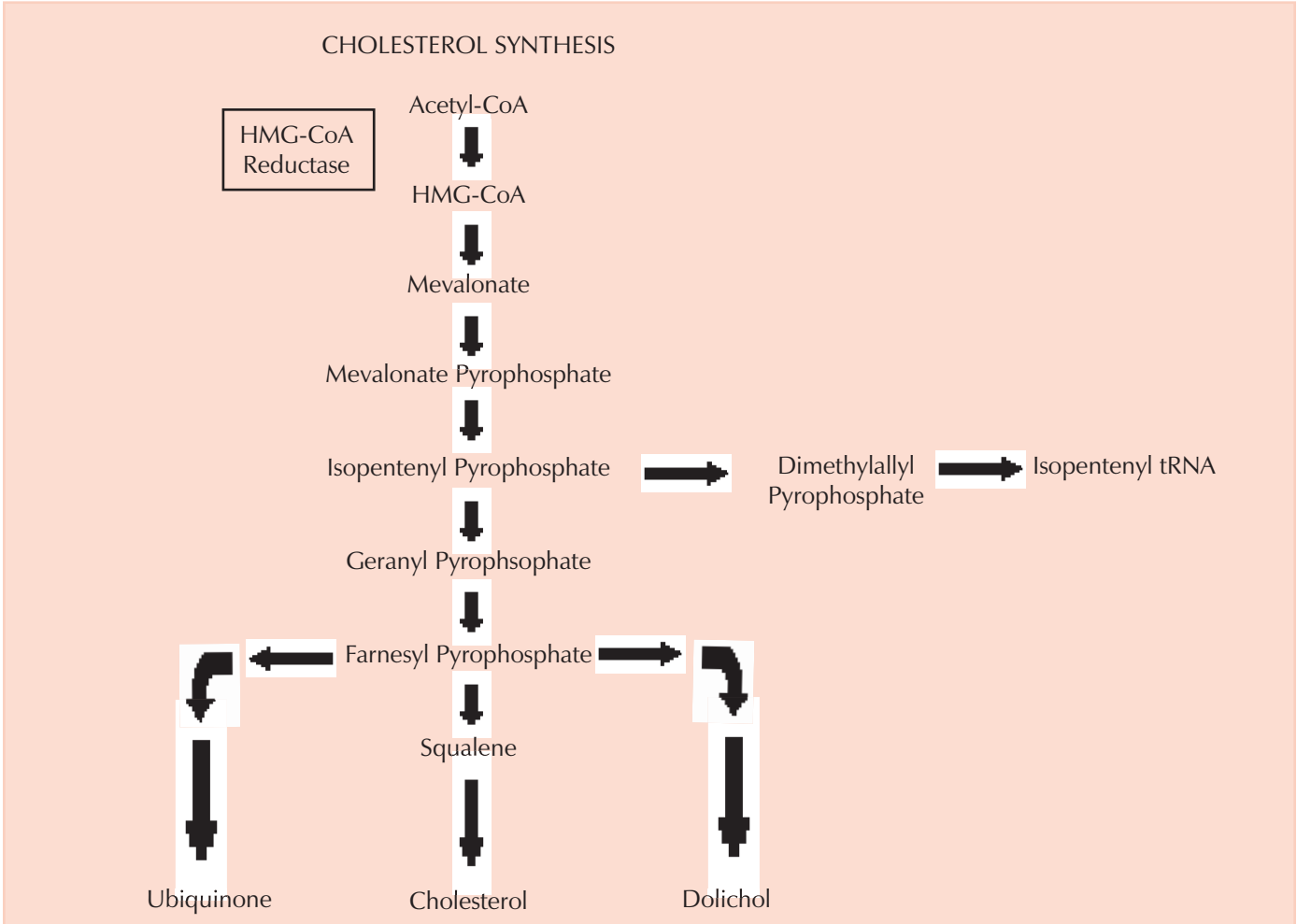
CHOLESTEROL

Of course, statins inhibit the production of cholesterol—they do this very well. Nowhere is the failing of our medical system more evident than in the wholesale acceptance of cholesterol reduction as a way to prevent disease—have all these doctors forgotten what they learned in biochemistry 101 about the many roles of cholesterol in the human biochemistry? Every cell membrane in our body contains cholesterol because cholesterol is what makes our cells waterproof—without cholesterol we could not have a different biochemistry on the inside and the outside of the cell. When cholesterol levels are not adequate, the cell membrane becomes leaky or porous, a situation the body interprets as an emergency, releasing a flood of corticoid hormones that work by sequestering cholesterol from one part of the body and transporting it to areas where it is lacking. Cholesterol is the body's repair substance: scar tissue contains high levels of cholesterol, including scar tissue in the arteries.

Cholesterol is the precursor to vitamin D, necessary for numerous biochemical processes including mineral metabolism. The bile salts, required for the digestion of fat, are made of cholesterol. Those who suffer from low cholesterol often have trouble digesting fats. Cholesterol also functions as a powerful antioxidant, thus protecting us against cancer and aging.

Cholesterol is vital to proper neurological function. It plays a key role in the formation of memory and the uptake of hormones in the brain, including serotonin, the body's feel-good chemical. When cholesterol levels drop too low, the serotonin receptors cannot work. Cholesterol is the main organic molecule in the brain, constituting over half the dry weight of the cerebral cortex.

Finally, cholesterol is the precursor to all the hormones produced in the adrenal cortex including glucocorticoids, which regulate blood sugar levels, and mineralocorticoids, which regulate mineral balance. Corticoids are the cholesterol-based adrenal hormones that the body uses in response to stress of various types; it promotes healing and balances the tendency to inflammation. The adrenal cortex also produces sex hormones, including testosterone, estrogen and progesterone, out of cholesterol. Thus, low



During the last 20 years, the industry has mounted an incredible promotional campaign—enlisting scientists, advertising agencies, the media and the medical profession in a blitz that turned the statins into one of the bestselling pharmaceuticals of all time.

cholesterol—whether due to an innate error of metabolism or induced by cholesterol-lowering diets and drugs—can be expected to disrupt the production of adrenal hormones and lead to blood sugar problems, edema, mineral deficiencies, chronic inflammation, difficulty in healing, allergies, asthma, reduced libido, infertility and various reproductive problems.

ENTER THE STATINS

Statin drugs entered the market with great promise. They replaced a class of pharmaceuticals that lowered cholesterol by preventing its absorption from the gut. These drugs often had immediate and unpleasant side effects, including nausea, indigestion and constipation, and in the typical patient they lowered cholesterol levels only slightly. Patient compliance was low: the benefit did not seem worth the side effects and the potential for use very limited. By contrast, statin drugs had no immediate side effects: they did not cause nausea or indigestion and they were consistently effective, often lowering cholesterol levels by 50 points or more. During the last 20 years, the industry has mounted an incredible promotional campaign—enlisting scientists, advertising agencies, the media and the medical profession in a blitz that turned the statins into one of the bestselling pharmaceuticals of all time. Sixteen million Americans now take Lipitor, the most popular statin, and drug company officials claim that 36 million Americans are candidates for statin drug therapy. What bedevils the industry is growing reports of side effects that manifest many months after the commencement of therapy; the November 2003 issue of *Smart Money* magazine reports on a 1999 study at St. Thomas' Hospital in London (apparently unpublished), which found that 36 percent of patients on Lipitor's highest dose reported side effects; even at the lowest dose, 10 percent reported side effects.[2]

MUSCLE PAIN AND WEAKNESS

The most common side effect is muscle pain and weakness, a condition called rhabdomyolysis, most likely due to the depletion of Co-Q_{10}, a nutrient that supports muscle function. Dr. Beatrice Golomb of San Diego, California is currently conducting a series of studies on statin side effects. The industry insists that only 2-3 percent of patients get muscle aches and cramps but in one study, Golomb found that 98 percent of patients taking Lipitor and one-third of the patients taking Mevachor (a lower-dose statin) suffered from muscle problems.[3] A message board devoted to Lipitor at forum.ditonline.com contains more than 800 posts, many detailing severe side effects. The Lipitor board at www.rxlist.com contains more than 2,600 posts.

The test for muscle wasting or rhabdomyolysis is elevated levels of a chemical called creatine

A BETTER WAY

If statins work, they do so by reducing inflammation, not because they lower cholesterol. Statins block the production of mevalonate leading to inhibition of platelet clumping and reduction of inflammation in the artery walls. However, simple changes in the diet can achieve the same effect without also cutting off the body's vital supply of cholesterol:

- Avoid *trans* fats, known to contribute to inflammation
- Avoid refined sugars, especially fructose, known to stimulate clumping of the blood platelets
- Take cod liver oil, an excellent dietary source of anti-inflammatory vitamin A, vitamin D and EPA
- Eat plenty of saturated fats, which encourage the production of anti-inflammatory prostaglandins
- Take evening primrose, borage or black currant oil, sources of GLA which the body uses to make anti-inflammatory prostaglandins
- Eat foods high in copper, especially liver; copper deficiency is associatied with clot formation and inflammation in the arteries
- Eat coconut oil and coconut products; coconut oil protects against bacteria and viruses that can lead to inflammation in the artery wall
- Avoid reduced-fat milks and powdered milk products (such as powdered whey); they contain oxidized cholesterol, shown to cause irritation of the artery wall

kinase (CK). But many people experience pain and fatigue even though they have normal CK levels.[4]

Tahoe City resident Doug Peterson developed slurred speech, balance problems and severe fatigue after three years on Lipitor—for two and a half years, he had no side effects at all.[5] It began with restless sleep patterns—twitching and flailing his arms. Loss of balance followed and the beginning of what Doug calls the "statin shuffle"—a slow, wobbly walk across the room. Fine motor skills suffered next. It took him five minutes to write four words, much of which was illegible. Cognitive function also declined. It was hard to convince his doctors that Lipitor could be the culprit, but when he finally stopped taking it, his coordination and memory improved.

John Altrocchi took Mevacor for three years without side effects; then he developed calf pain so severe he could hardly walk. He also experienced episodes of temporary memory loss.

For some, however, muscle problems show up shortly after treatment begins. Ed Ontiveros began having muscle problems within 30 days of taking Lipitor. He fell in the bathroom and had trouble getting up. The weakness subsided when he went off Lipitor. In another case, reported in the medical journal *Heart*, a patient developed rhabdomyolysis after a single dose of a statin.[6]

Heel pain from plantar fascitis (heel spurs) is another common complaint among those taking statin drugs. One correspondent reported the onset of pain in the feet shortly after beginning statin treatment. She had visited an evangelist, requesting that he pray for her sore feet. He enquired whether she was taking Lipitor. When she said yes, he told her that his feet had also hurt when he took Lipitor.[7]

Active people are much more likely to develop problems from statin use than those who are sedentary. In a study carried out in Austria, only six out of 22 athletes with familial hypercholesterolemia were able to endure statin treatment.[8] The others discontinued treatment because of muscle pain.

Active people are much more likely to develop problems from statin use than those who are sedentary.

By the way, other cholesterol-lowering agents besides statin drugs can cause joint pain and muscle weakness. A report in *Southern Medical Journal* described muscle pains and weakness in a man who took Chinese red rice, an herbal preparation that lowers cholesterol.[9] Anyone suffering from myopathy, fibromyalgia, coordination problems and fatigue needs to look at low cholesterol plus Co-Q_{10} deficiency as a possible cause.

NEUROPATHY

Polyneuropathy, also known as peripheral neuropathy, is characterized by weakness, tingling and pain in the hands and feet as well as

DIETARY TRIALS

Doctors and other health professionals claim there is ample proof that animal fats cause heart disease while they confidently advise us to adopt a lowfat diet; actually the literature contains only two studies involving humans that compared the outcome (not markers like cholesterol levels) of a diet high in animal fat with a diet based on vegetable oils, and both showed that animal fats are protective.

The Anti-Coronary Club project, launched in 1957 and published in 1966 in the *Journal of the American Medical Association*, compared two groups of New York businessmen, aged 40 to 59 years. One group followed the so-called "Prudent Diet" consisting of corn oil and margarine instead of butter, cold breakfast cereals instead of eggs and chicken and fish instead of beef; a control group ate eggs for breakfast and meat three times per day. The final report noted that the Prudent Dieters had average serum cholesterol of 220 mg/l, compared to 250 mg/l in the eggs-and-meat group. But there were *eight* deaths from heart disease among Prudent Dieter group, and *none* among those who ate meat three times a day

In a study published in the *British Medical Journal*, 1965, patients who had already had a heart attack were divided into three groups: one group got polyunsaturated corn oil, the second got monounsaturated olive oil and the third group was told to eat animal fat. After two years, the corn oil group had 30 percent lower cholesterol, but only 52 percent of them were still alive. The olive oil groups fared little better—only 57 percent were alive after two years. But of the group that ate mostly animal fat, 75 percent were still alive after two years.

The question is, does widespread statin-induced neuropathy make our elderly drivers (and even not-so-elderly drivers) more accident prone?

difficulty walking. Researchers who studied 500,000 residents of Denmark, about 9 percent of that country's population, found that people who took statins were more likely to develop polyneuropathy.[10] Taking statins for one year raised the risk of nerve damage by about 15 percent—about one case for every 2,200 patients. For those who took statins for two or more years, the additional risk rose to 26 percent.

According to the research of Dr. Golomb, nerve problems are a common side effect from statin use; patients who use statins for two or more years are at a four to 14-fold increased risk of developing idiopathic polyneuropathy compared to controls.[11] She reports that in many cases, patients told her they had complained to their doctors about neurological problems, only to be assured that their symptoms could not be related to cholesterol-lowering medications.

The damage is often irreversible. People who take large doses for a long time may be left with permanent nerve damage, even after they stop taking the drug.

The question is, does widespread statin-induced neuropathy make our elderly drivers (and even not-so-elderly drivers) more accident prone? In July of 2003, an 86-year-old man with an excellent driving record plowed into a farmers' market in Santa Monica, California, killing ten people. Several days later, a most interesting letter from a Lake Oswego, Oregon woman appeared in the *Washington Post*:[12]

"My husband, at age 68, backed into the garage and stepped on the gas, wrecking a lot of stuff. He said his foot slipped off the brake. He had health problems and is on medication, including a cholesterol drug, which is now known to cause problems with feeling in one's legs.

"In my little community, older drivers have missed a turn and taken out the end of a music store, the double doors of the post office and the front of a bakery. In Portland, a bank had to do without its drive-up window for some time.

"It is easy to say that one's foot slipped, but the problem could be lack of sensation. My husband's sister-in-law thought her car was malfunctioning when it refused to go when a light turned green, until she looked down and saw that her foot was on the brake. I have another friend who mentioned having no feeling in her lower extremities. She thought about having her car retrofitted with hand controls but opted for the handicapped bus instead."

HEART FAILURE

We are currently in the midst of a congestive heart failure epidemic in the United States—while the incidence of heart attack has declined slightly, an increase in the number heart failure cases has outpaced these gains. Deaths attributed to heart failure more than doubled from 1989 to 1997.[13] (Statins were first given pre-market approval in 1987.) Interference with production of Co-Q_{10} by statin drugs is the most likely explanation. The heart is a muscle and it cannot work when deprived of Co-Q_{10}.

Cardiologist Peter Langsjoen studied 20 patients with completely normal heart function. After six months on a low dose of 20 mg of Lipitor a day, two-thirds of the patients had abnormalities in the heart's filling phase, when the muscle fills with blood. According to Langsjoen, this malfunction is due to Co-Q_{10} depletion. Without Co-Q_{10}, the cell's mitochondria are inhibited from producing energy, leading to muscle pain and weakness. The heart is especially susceptible because it uses so much energy.[14]

Co-Q_{10} depletion becomes more and more of a problem as the pharmaceutical industry encourages doctors to lower cholesterol levels in their patients by greater and greater amounts. Fifteen animal studies in six different animal species have documented statin-induced Co-Q_{10} depletion leading to decreased ATP production, increased injury from heart failure, skeletal muscle injury and increased mortality. Of the nine controlled trials on statin-induced Co-Q_{10} depletion in humans, eight showed significant Co-Q_{10} depletion leading to decline in left ventricular function and biochemical imbalances.[15]

Yet virtually all patients with heart failure are put on statin drugs, even if their cholesterol is already low. Of interest is a recent study indicating that patients with chronic heart failure benefit from having high levels of cholesterol rather than low. Researchers in Hull, UK followed 114 heart failure patients for at least 12 months.[16] Survival was 78 percent at 12 months and 56 percent at 36 months. They found that for every point of *decrease* in serum cholesterol, there was a 36

percent *increase* in the risk of death within 3 years.

DIZZINESS

Dizziness is commonly associated with statin use, possibly due to pressure-lowering effects. One woman reported dizziness one half hour after taking Pravachol.[17] When she stopped taking it, the dizziness cleared up. Blood pressure lowering has been reported with several statins in published studies. According to Dr. Golumb, who notes that dizziness is a common adverse effect, the elderly may be particularly sensitive to drops in blood pressure.[18]

COGNITIVE IMPAIRMENT

The November 2003 issue of *Smart Money*[19] describes the case of Mike Hope, owner of a successful ophthalmologic supply company: "There's an awkward silence when you ask Mike Hope his age. He doesn't change the subject or stammer, or make a silly joke about how he stopped counting at 21. He simply doesn't remember. Ten seconds pass. Then 20. Finally an answer comes to him. 'I'm 56,' he says. Close, but not quite. 'I will be 56 this year.' Later, if you happen to ask him about the book he's reading, you'll hit another roadblock. He can't recall the title, the author or the plot." Statin use since 1998 has caused his speech and memory to fade. He was forced to close his business and went on Social Security 10 years early. Things improved when he discontinued Lipitor in 2002, but he is far from complete recovery—he still cannot sustain a conversation. What Lipitor did was turn Mike Hope into an old man when he was in the prime of life.

Cases like Mike's have shown up in the medical literature as well. An article in *Pharmacotherapy*, December 2003, for example, reports two cases of cognitive impairment associated with Lipitor and Zocor.[20] Both patients suffered progressive cognitive decline that reversed completely within a month after discontinuation of the statins. A study conducted at the University of Pittsburgh showed that patients treated with statins for six months compared poorly with patients on a placebo in solving complex mazes, psychomotor skills and memory tests.[21]

Dr. Golomb has found that 15 percent of statin patients develop some cognitive side effects.[22] The most harrowing involve global transient amnesia—complete memory loss for a brief or lengthy period—described by former astronaut Duane Graveline in his book *Lipitor: Thief of Memory*.[23] Sufferers report baffling incidents involving complete loss of memory—arriving at a store and not remembering why they are there, unable to remember their name or the names of their loved ones, unable to find their way home in the car. These episodes occur suddenly and disappear just as suddenly. Graveline points out that we are all at risk when the general public is taking statins—do you want to be in an airplane when your pilot develops statin-induced amnesia?

While the pharmaceutical industry denies the fact that statins can cause amnesia, memory loss has shown up in several statin trials. In a trial involving 2502 subjects, amnesia occurred in 7 receiving Lipitor; amnesia also occurred in 2 of 742 subjects during comparative trials with other statins. In addition, "abnormal thinking" was reported in 4 of the 2502 clinical trial subjects.[24] The total recorded side effects was therefore 0.5 percent; a figure that likely under-represents the true frequency since memory loss was not specifically studied in these trials.

CANCER

In every study with rodents to date, statins have caused cancer.[25] Why have we not seen such a dramatic correlation in human studies? Because cancer takes a long time to develop and most of the statin trials do not go on longer than two or three years. Still, in one trial, the CARE trial, breast cancer rates of those taking a statin went up 1500 percent.[26] In the Heart Protection Study, non-melanoma skin cancer occurred in 243 patients treated with simvastatin compared with 202 cases in the control group.[27]

Manufacturers of statin drugs have recognized the fact that statins depress the immune system, an effect that can lead to cancer and infectious disease, recommending statin use for inflammatory arthritis and as an immune suppressor for transplant patients.[28]

PANCREATIC ROT

The medical literature contains several

A study conducted at the University of Pittsburgh showed that patients treated with statins for six months compared poorly with patients on a placebo in solving complex mazes, psychomotor skills and memory tests.

Numerous studies have linked low cholesterol with depression.

reports of pancreatitis in patients taking statins. One paper describes the case of a 49-year-old woman who was admitted to the hospital with diarrhea and septic shock one month after beginning treatment with lovastatin.[29] She died after prolonged hospitalization; the cause of death was necrotizing pancreatitis. Her doctors noted that the patient had no evidence of common risk factors for acute pancreatitis, such as biliary tract disease or alcohol use. "Prescribers of statins (particularly simvastatin and lovastatin) should take into account the possibility of acute pancreatitis in patients who develop abdominal pain within the first weeks of treatment with these drugs," they warned.

DEPRESSION

Numerous studies have linked low cholesterol with depression. One of the most recent found that women with low cholesterol are twice as likely to suffer from depression and anxiety. Researchers from Duke University Medical Center carried out personality trait measurements on 121 young women aged 18 to 27.[30] They found that 39 percent of the women with low cholesterol levels scored high on personality traits that signalled proneness to depression, compared to 19 percent of women with normal or high levels of cholesterol. In addition, one in three of the women with low cholesterol levels scored high on anxiety indicators, compared to 21 percent with normal levels. Yet the author of the study, Dr. Edward Suarez, cautioned women with low cholesterol against eating "foods such as cream cakes" to raise cholesterol, warning that these types of food "can cause heart disease." In previous studies on men, Dr. Suarez found that men who lower their cholesterol levels with medication have increased rates of suicide and violent death, leading the researchers to theorize "that low cholesterol levels were causing mood disturbances."

How many elderly statin-takers eke through their golden years feeling miserable and depressed, when they should be enjoying their grandchildren and looking back with pride on their accomplishments? But that is the new dogma—you may have a long life as long as it is experienced as a vale of tears.

ANY BENEFITS?

Most doctors are convinced—and seek to convince their patients—that the benefits of statin drugs far outweigh the side effects. They can cite a number of studies in which statin use has lowered the number of coronary deaths compared to controls. But as Dr. Ravnskov has pointed out in his book *The Cholesterol Myths*,[31] the results of the major studies up to the year 2000—the 4S, WOSCOPS, CARE, AFCAPS and LIPID studies—generally showed only small differences and these differences were often statistically insignificant and independent of the amount of cholesterol lowering achieved. In two studies, EXCEL, and FACAPT/TexCAPS, more deaths occurred in the treatment group compared to controls. Dr. Ravnskov's 1992 meta-analysis of 26 controlled cholesterol-lowering trials found an equal number of cardiovascular deaths in the treatment and control groups and a greater number of total deaths in the treatment groups.[32] An analysis of all the big controlled trials reported before 2000 found that long-term use of statins for primary prevention of heart disase produced a 1 percent *greater* risk of death over 10 years compared to a placebo.[33]

Recently published studies do not provide any more justification for the current campaign to put as many people as possible on statin drugs.

HONOLULU HEART PROGRAM (2001)

This report, part of an ongoing study, looked at cholesterol lowering in the elderly. Researchers compared changes in cholesterol concentrations over 20 years with all-cause mortality.[34] To quote: "Our data accords with previous findings of increased mortality 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. . . The most striking findings were related to changes in cholesterol between examination three (1971-74) and examination four (1991-93). 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 con-

centrations of cholesterol 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* [emphasis ours]."

MIRACL (2001)

The MIRACL study looked at the effects of a high dose of Lipitor on 3086 patients in the hospital after angina or nonfatal MI and followed them for 16 weeks.[35] According to the abstract: "For patients with acute coronary syndrome, lipid-lowering therapy with atorvastatin, 80 mg/day, reduced recurrent ischemic events in the first 16 weeks, mostly recurrent symptomatic ischemia requiring rehospitalization." What the abstract did not mention was that there was no change in death rate compared to controls and no significant change in re-infarction rate or need for resuscitation from cardiac arrest. The only change was a significant drop in chest pain requiring rehospitalization.

ALLHAT (2002)

ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial), the largest North American cholesterol-lowering trial ever and the largest trial in the world using Lipitor, showed mortality of the treatment group and controls after 3 or 6 years was identical.[36] Researchers used data from more than 10,000 participants and followed them over a period of four years, comparing the use of a statin drug to "usual care," namely maintaining proper body weight, no smoking, regular exercise, etc., in treating subjects with moderately high levels of LDL cholesterol. Of the 5170 subjects in the group that received statin drugs, 28 percent lowered their LDL cholesterol significantly. And of the 5185 usual-care subjects, about 11 percent had a similar drop in LDL. *But both groups showed the same rates of death, heart attack and heart disease*.

HEART PROTECTION STUDY (2002)

Carried out at Oxford University,[37] this study received widespread press coverage; researchers claimed "massive benefits" from cholesterol-lowering,[38] leading one commentator to predict that statin drugs were "the new aspirin."[39] But as Dr. Ravnskov points out,[40] the benefits were far from massive. Those who took simvastatin had an 87.1 percent survival rate after five years compared to an 85.4 percent survival rate for the controls and these results were independent of the amount of cholesterol lowering. The authors of the Heart Protection Study never published cumulative mortality data, even though they received many requests to do so and even though they received funding and carried out a study to look at cumulative data. According to the authors, providing year-by-year mortality data would be an "inappropriate" way of publishing their study results.[41]

PROSPER (2002)

PROSPER (Prospective Study of Pravastatin in the Elderly at Risk) studied the effect of pravastatin compared to placebo in two older populations of patients of which 56 percent were primary prevention cases (no past or symptomatic cardiovascular disease) and 44 percent were secondary prevention cases (past or symptomatic cardiovascular disease).[42] Pravastatin did not reduce total myocardial infarction or total stroke in the primary prevention population but did so in the secondary. However, measures of overall health impact in the combined populations, total mortality and total serious adverse events were unchanged by pravastatin as compared to the placebo and those in the treatment group had increased cancer. In other words: not one life saved.

J-LIT (2002)

Japanese Lipid Intervention Trial was a six-year study of 47,294 patients treated with the same dose of simvastatin.[43] Patients were grouped by the amount of cholesterol lowering. Some patients had no reduction in LDL levels, some had a moderate fall in LDL and some had very large LDL reductions. The results: *no* correlation between the amount of LDL lowering and death rate at five years. Those with LDL cholesterol lower than 80 had a death rate of just over 3.5 at five years; those whose LDL was over 200 had a death rate of just over 3.5 at five years.

META-ANALYSIS (2003)

In a meta-analysis of 44 trials involving al-

Those individuals with a low serum cholesterol maintained over a 20-year period will have the worst outlook for all-cause mortality.

most 10,000 patients, the death rate was identical at 1 percent of patients in each of the three groups—those taking atorvastatin (Lipitor), those taking other statins and those taking nothing.[44] Furthermore, 65 percent of those on treatment versus 45 percent of the controls experienced an adverse event. Researchers claimed that the incidence of adverse effects was the same in all three groups, but 3 percent of the atorvastatin-treated patients and 4 percent of those receiving other statins withdrew due to treatment-associated adverse events, compared with 1 percent of patients on the placebo.

STATINS AND PLAQUE (2003)

A study published in the *American Journal of Cardiology* casts serious doubt on the commonly held belief that lowering your LDL-cholesterol, the so-called bad cholesterol, is the most effective way to reduced arterial plaque.[45] Researchers at Beth Israel Medical Center in New York City examined the coronary plaque buildup in 182 subjects who took statin drugs to lower cholesterol levels. One group of subjects used the drug aggressively (more than 80 mg per day) while the balance of the subjects took less than 80 mg per day. Using electron beam tomography, the researchers measured plaque in all of the subjects before and after a study period of more than one year. The subjects were generally successful in lowering their cholesterol, but in the end there was no statistical difference in the two groups in the progression of arterial calcified plaque. On average, subjects in *both* groups showed a 9.2 percent increase in plaque *buildup*.

STATINS AND WOMEN (2003)

No study has shown a significant reduction in mortality in women treated with statins. The University of British Columbia Therapeutics Initiative came to the same conclusion, with the finding that statins offer no benefit to women for prevention of heart disease.[46] Yet in February of

HOW THEY CHEAT

Researchers use many statistical tricks to present results that conform with the reigning medical paradigm and the expectation of study sponsors. These include:

- Exaggerating trivial results using the concept of "relative risk." For example, if the CHD death rate at cholesterol levels of 240 mg/dl is 2/1000 and at 160 mg/dl is 1/1000, the rate of difference (called the *absolute risk*) is 1/1000 or 0.001 percent but the difference in *relative risk* is 100 percent (2 is 100 percent greater than 1). If the CHD death rate at 240 mg/ml is 2/1billion and at 160 mg/dl is 1/1billion, the rate of difference (absolute risk) is 1/1billion or 0.0000001 percent but the difference in relative risk is still 100 percent (2 is 100 percent greater than 1). In other words, the concept of relative risk eliminates the sample size and makes trivial results seem very important. Cholesterol theory proponents usually exaggerate benefits by reporting them in terms of relative risk and minimize side effects by reporting them in terms of absolute risk.
- Using surrogate end points (such as lower LDL-cholesterol) rather than meaningful end points (such as death from heart disease or from all causes). The recently published ENHANCE study, which looked at meaningful endpoints, showed that significantly lowering LDL-cholesterol did not result in the prolongation of life or prevention of heart attacks. Approval of statin drugs was based on studies using only surrogate end points.
- Assignment of data to unequal intervals. This can be done in such a way as to exaggerate trivial findings, making them seem very important.
- Leaving out data in epidemiological studies. Ancel Keys, who published the famous Six and Seven Countries studies in the 1950s, used this method to create graphs showing a strong correlation between fat consumption and heart disease in carefully selected countries.
- Cherry picking results to find chance correlations. In a large study that looks at many risk factors, it is always possible to find positive correlations in certain groups even when the overall results are disappointing.
- Changing trial's endpoint (the final result that the study was supposed to measure) to conform to data received.
- Determining nutrient intake with dietary recall questionnaires. These are notoriously inaccurate and create erroneous conclusions about nutrient intake.
- Confounding a risk factor with a cause. There are hundreds of known risk factors for heart disease, including short stature, television ownership and hairy earlobes. Many of these are stronger risk factors than elevated cholesterol (which is a mild risk factor only for middle aged men). But a risk factor is not necessarily a cause!
- Abstracts do not accurately reflect findings. One egregious example is the Anti-Coronary Club study, in which those put on diets that restricted animal fats had eight deaths from heart attack while those in the control group had none (Bulletin NY Academy of Medicine 1968). This important finding was omitted from the abstract and relegated to fine print at the end of the article.
- Omission of contradictory studies in review articles. Review articles look at combined data from many studies. Often contradictory studies that would change the findings are excluded.

2004, *Circulation* published an article in which more than 20 organizations endorsed cardiovascular disease prevention guidelines for women with several mentions of "preferably a statin."[47]

ASCOT-LLA (2003)

ASCOT-LLA (Anglo-Scandinavian Cardiac Outcomes Trial—Lipid Lowering Arm) was designed to assess the benefits of atorvastatin (Lipitor) versus a placebo in patients who had high blood pressure with average or lower-than-average cholesterol concentrations and at least three other cardiovascular risk factors.[48] The trial was originally planned for five years but was stopped after a median follow-up of 3.3 years because of a significant reduction in cardiac events. Lipitor did reduce total myocardial infarction and total stroke; however, total *mortality* was not significantly reduced. In fact, women were worse off with treatment. The trial report stated that total serious adverse events "did not differ between patients assigned atorvastatin or placebo," but did not supply the actual numbers of serious events.

CHOLESTEROL LEVELS IN DIALYSIS PATIENTS (2004)

In a study of dialysis patients, those with *higher* cholesterol levels had *lower* mortality than those with low cholesterol.[49] Yet the authors claimed that the "inverse association of total cholesterol level with mortality in dialysis patients is likely due to the cholesterol-lowering effect of systemic inflammation and malnutrition, not to a protective effect of high cholesterol concentrations." Keeping an eye on further funding opportunities, the authors concluded: "These findings support treatment of hypercholesterolemia in this population."

PROVE-IT (2004)

PROVE-IT (PRavastatin Or AtorVastatin Evaluation and Infection Study),[50] led by researchers at Harvard University Medical School, attracted immense media attention. "Study of Two Cholesterol Drugs Finds One Halts Heart Disease," was the headline in the *New York Times*.[51] In an editorial entitled "Extra-Low Cholesterol," the paper predicted that "The findings could certainly presage a significant change in the way heart disease patients are treated. It should also start a careful evaluation of whether normally healthy people could benefit from a sharp drug-induced reduction in their cholesterol levels."[52]

The Washington Post was even more effusive, with a headline "Striking Benefits Found in Ultra-Low Cholesterol."[53] "Heart patients who achieved ultra-low cholesterol levels in one study were 16 percent less likely to get sicker or to die than those who hit what are usually considered optimal levels. The findings should prompt doctors to give much higher doses of drugs known as statins to hundreds of thousands of patients who already have severe heart problems, experts said. In addition, it will probably encourage physicians to start giving the medications to millions of healthy people who are not yet on them, and to boost dosages for some of those already taking them to lower their cholesterol even more, they said."

The study compared two statin drugs, Lipitor and Pravachol. Although Bristol Myers-Squibb (BMS), makers of Pravachol, sponsored the study, Lipitor (made by Pfizer) outperformed its rival Pravachol in lowering LDL. The "striking benefit" was a 22 percent rate of death or further adverse coronary events in the Lipitor patients compared to 26 percent in the Pravachol patients.

PROVE-IT investigators studied 4162 patients who had been in the hospital following an MI or unstable angina. Half got Pravachol and half got Lipitor. Those taking Lipitor had the greatest reduction of LDL-cholesterol—LDL in the Pravachol group was 95, in the Lipitor group it was 62—a 32 percent greater reduction in LDL levels and a 16 percent reduction in all-cause mortality. But that 16 percent was a reduction in relative risk. As pointed out by Red Flags Daily columnist Dr. Malcolm Kendrick, the absolute reduction in the rate of the death rate of those taking Lipitor rather than Pravachol, was one percent, a decrease from 3.2 percent to 2.2 percent over two years.[54] Or, to put it another way, a 0.5 percent absolute risk reduction per year—these were the figures that launched the massive campaign for cholesterol-lowering in people with no risk factors for heart disease, not even high cholesterol.

In a study of dialysis patients, those with higher cholesterol levels had lower mortality than those with low cholesterol.

Kendrick notes that the carefully constructed J-LIT study, published two years earlier, found no correlation whatsoever between the amount of LDL lowering and the death rate.

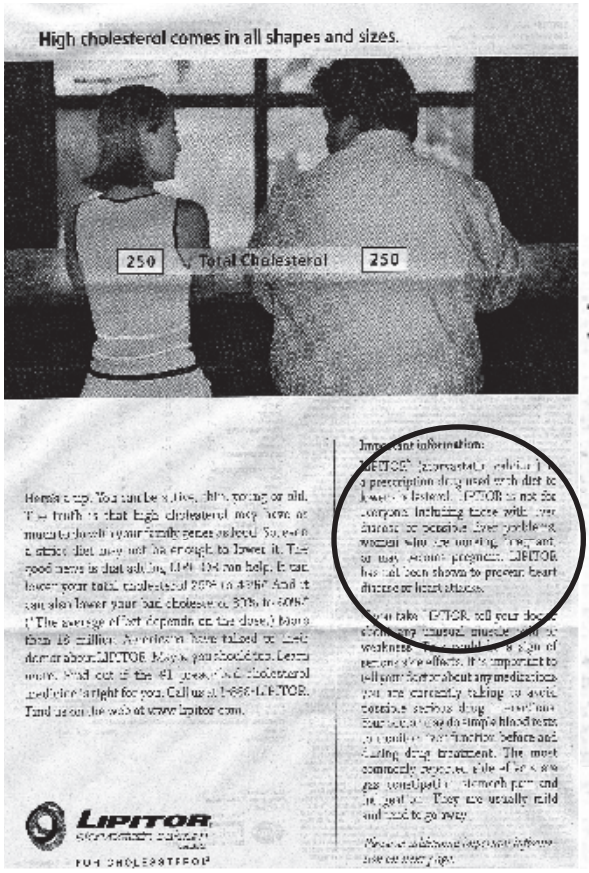
And the study was seriously flawed with what Kendrick calls "the two-variables conundrum." "It is true that those with the greatest LDL lowering were protected against death. However, . . . those who were protected not only had a greater degree of LDL lowering, *they were also on a different drug!* which is rather important, yet seems to have been swept aside on a wave of hype. If you really want to prove that the more you lower the LDL level, the greater the protection, then you *must* use the same drug. This achieves the absolutely critical requirement of any scientific experiment, which is to remove all possible uncontrolled variables. . . As this study presently stands, because they used different drugs, anyone can make the case that the benefits seen in the patients on atorvastatin [Lipitor] had nothing to do with greater LDL lowering; they were purely due to the direct drug effects of atorvastatin." Kendrick notes that the carefully constructed J-LIT study, published two years earlier, found no correlation whatsoever between the amount of LDL lowering and the death rate. This study had ten times as many patients, lasted almost three times as long and used the same drug at the same dose in all patients. Not surprisingly, J-LIT attracted virtually no media attention.

PROVE-IT did not look at side effects but Dr. Andrew G. Bodnar, senior vice president for strategy and medical and external affairs at Bristol Meyer Squibb, makers of the losing statin, indicated that liver enzymes were elevated in 3.3 percent of the Lipitor group but only in 1.1 percent of the Pravachol group, noting that when liver enzyme levels rise, patients must be advised to stop taking the drug or reduce the dose.[55] And withdrawal rates were very high: thirty-three percent of patients discontinued Pravachol and 30 percent discontinued Lipitor after two years due to adverse events or other reasons.[56]

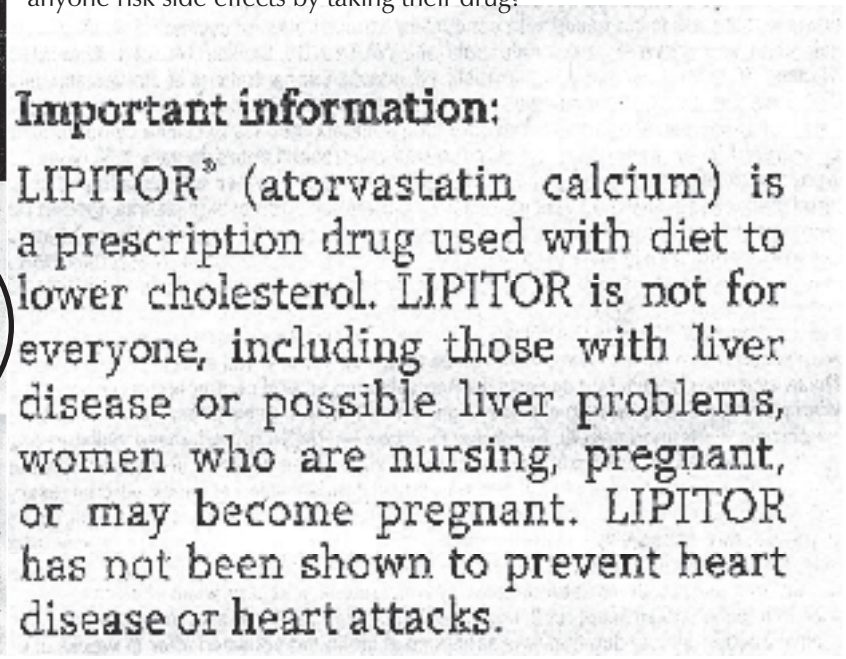
REVERSAL (2004)

In a similar study, carried out at the Cleveland Clinic, patients were given either Lipitor

READ THE FINE PRINT!



The picture in a recent ad for Lipitor implies that cholesterol-lowering is for everyone, even slim young women. However, in the fine print we learn that Lipitor "has not been shown to prevent heart disease or heart attacks"! If the makers of Lipitor need to provide this disclaimer, after millions of dollars invested in studies, why should anyone risk side effects by taking their drug?



or Pravachol. Those receiving Lipitor achieved much lower LDL-cholesterol levels and a reversal in "the progression of coronary plaque aggregation."[57] Those who took Lipitor had plaque reduced by 0.4 percent over 18 months, based on intravascular ultrasound (not the more accurate tool of electron beam tomography); Dr. Eric Topol of the Cleveland Clinic claimed these decidedly unspectacular results "Herald a shake-up in the field of cardiovascular prevention.. . . the implications of this turning point—that is, of the new era of intensive statin therapy—are profound. Even today, only a fraction of the patients who should be treated with a statin are actually receiving such therapy. . . More than 200 million people worldwide meet the criteria for treatment, but fewer than 25 million take statins."[58] Not surprisingly, an article in *The Wall Street Journal* noted "Lipitor Prescriptions Surge in Wake of Big Study."[59]

But as Dr. Ravnskov points out, the investigators looked at change in atheroma volume, not the change in lumen area, "a more important parameter because it determines the amount of blood that can be delivered to the myocardium. Change of atheroma volume cannot be translated to clinical events because adaptive mechansims try to maintain a normal lumen area during early atherogenesis."[60]

OTHER USES

With such paltry evidence of benefit, statin drugs hardly merit the hyperbole heaped upon them. Yet the industry maintains a full court press, urging their use for greater and greater numbers of people, not only for cholesterol lowering but also as treatment for other diseases—cancer, multiple sclerosis, osteoporosis, stroke, macular degeneration, arthritis and even mental disorders such as memory and learning problems, Alzheimer's and dementia.[61] New guidelines published by the American College of Physicians call for statin use by all people with diabetes older than 55 and for younger diabetes patients who have any other risk factor for heart disease, such as high blood pressure or a history of smoking.[62] David A. Drachman, professor of neurology at the University of Massachusetts Medical School calls statins "Viagra for the brain."[63] Other medical writers have heralded the polypill, composed of a statin drug mixed with a blood pressure medication, aspirin and niacin, as a prevent-all that everyone can take. The industry is also seeking the right to sell statins over the counter.

Can honest assessment find any possible use for these dangerous drugs? Dr. Peter Langsjoen of Tyler, Texas, suggests that statin drugs are appropriate only as a treatment for cases of advanced Cholesterol Neurosis, created by the industry's anti-cholesterol propaganda. If you are concerned about your cholesterol, a statin drug will relieve you of your worries.

CREATIVE ADVERTISING

The best advertising for statin drugs is free front-page coverage following gushy press releases. But not everyone reads the paper or goes in for regular medical exams, so statin manufacturers pay big money for creative ways to create new users. For example, a new health awareness group called the Boomer Coalition supported ABC's Academy Awards telecast in March of 2004 with a 30-second spot flashing nostalgic images of celebrities lost to cardiovascular disease—actor James Coburn, baseball star Don Drysdale and comedian Redd Foxx. While the Boomer Coalition sounds like a grass roots group of health activists, it is actually a creation of Pfizer, manufacturers of Lipitor. "We're always looking for creative ways to break through what we've found to be a lack of awareness and action," says Michal Fishman, a Pfizer spokeswoman. "We're always looking for what people really think and what's going to make people take action," adding that there is a stigma about seeking treatment and many people "wrongly assume that if they are physically fit, they aren't at risk for heart disease."[64] The Boomer Coalition website allows visitors to "sign up and take responsibility for your heart health," by providing a user name, age, email address and blood pressure and cholesterol level.

A television ad in Canada admonished viewers to "Ask your doctor about the Heart Protection Study from Oxford University." The ad did not urge viewers to ask their doctors about EXCEL, ALLHAT, ASCOT, MIRACL or PROSPER, studies that showed no benefit—and the potential for great harm—from taking statin drugs.

With such paltry evidence of benefit, statin drugs hardly merit the hyperbole heaped upon them. Yet the industry maintains a full court press, urging their use for greater and greater numbers of people, not only for cholesterol lowering but also as treatment for other diseases.

THE COSTS

Statin drugs are very expensive—a course of statins for a year costs between $900 and $1400. They constitute the mostly widely sold pharmaceutical drug, accounting for 6.5 percent of market share and 12.5 billion dollars in revenue for the industry. Your insurance company may pay most of that cost, but consumers always ultimately pay with higher insurance premiums. Payment for statin drugs poses a huge burden for Medicare, so much so that funds may not be available for truly lifesaving medical measures.

In the UK, according to the National Health Service, doctors wrote 31 million prescriptions for statins in 2003, up from one million in 1995 at a cost of 7 billion pounds—and that's just in one tiny island.[65] In the US, statins currently bring in $12.5 billion annually for the pharmaceutical industry. Sales of Lipitor, the number-one-selling statin, are projected to hit $10 billion in 2005.

Even if statin drugs do provide some benefit, the cost is very high. In the WOSCOP clinical trial where healthy people with high cholesterol were treated with statins, the five-year death rate for treated subjects was reduced by a mere 0.6 percent. As Dr. Ravnskov points out,[66] to achieve that slight reduction about 165 healthy people had to be treated for five years to extend one life by five years. The cost for that one life comes to $1.2 million dollars. In the most optimistic calculations, the costs to save one year of life in patients with CHD is estimated at $10,000, and much more for healthy individuals. "This may not sound unreasonable," says Dr. Ravnskov. "Isn't a human life worth $10,000 or more?"

"The implication of such reasoning is that to add as many years as possible, more than half of mankind should take statin drugs every day from an early age to the end of life. It is easy to calculate that the costs for such treatment would consume most of any government's health budget. And if money is spent to give statin treatment to all healthy people, what will remain for the care of those who really need it? Shouldn't health care be given primarily to the sick and the crippled?"

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Cholesterol and Stroke

By Chris Masterjohn

Cholesterol is essential to human life. It is a primary constituent of cell membranes, essential to learning and memory, and the fundamental building block of bile acids, vitamin D, and the steroid hormones.[1] Because in most cases the body synthesizes all the cholesterol it needs, however, scientists do not consider it an essential nutrient. Weston Price did not study the cholesterol content of primitive and modern diets, and he wrote his epic work, *Nutrition and Physical Degeneration*, decades before the medical establishment began measuring blood cholesterol levels and recommending cholesterol-lowering drugs and diets.[2]

Although Price's work did not directly concern this vital molecule, the medical establishment's campaign against it has nevertheless produced a nutritional paradigm that is antithetical to his findings: the American Heart Association, for example, recommends lowering cholesterol by limiting the consumption of butter, egg yolks, and organ meats – foods that formed the centerpiece of the primitive diets that Price's work esteemed.[3]

Careful examination of these findings allows us to assemble a strategy for preventing stroke within the context of a diet rich in traditional whole foods—including those rich in cholesterol.

STROKE THEN AND NOW

Stroke is the third leading cause of death in the United States.[4] Through the early 1990s, neither epidemiological studies[5] nor controlled trials of cholesterol-lowering drugs[6] were able to generate any evidence for an association between cholesterol levels and the risk of this disease. Today, however, the scene is very different. Recent news articles have reported that cholesterol-lowering drugs do, in fact, lower the risk of stroke,[7] and that women with high cholesterol levels are at risk even if they are otherwise healthy.[8] The American Heart Association now lists reducing the risk of stroke as the second most important reason for avoiding cholesterol-rich foods.[3]

Those who wish to believe in the association may be tempted to dismiss the old research as inferior by virtue of its comparative antiquity; those who wish to deny the association may be tempted to dismiss the new research as the product of an increasingly entrenched command of research dollars wielded by the manufacturers of cholesterol-lowering drugs. The truth lies somewhere between these two extremes.

Research over the last two decades has overcome many of the methodological flaws of earlier research and allowed us to recognize that cholesterol levels are indeed related to stroke—increasing the risk of some forms and decreasing the risk of others. Most important, this research has shown that oxidative stress, inflammation, and the health of the cells that line the blood vessel walls are the true causes underlying the efficacy of cholesterol-lowering statins. Careful examination of these findings allows us to assemble a strategy for preventing stroke within the context of a diet rich in traditional whole foods—including those rich in cholesterol.

DIFFERENT STROKES

Although a given stroke may have more than one hundred potential causes, the vast majority of strokes fall roughly into two categories: hemorrhagic and ischemic. Hemorrhagic stroke occurs when a blood vessel ruptures, causing uncontrolled bleeding into brain tissue. Ischemic stroke occurs when a blockage closes off the flow of blood within a vessel, depriving brain tissue of the oxygen and nutrients it needs to survive. (In the US, over 90 percent of all strokes are ischemic.) The cell death that results from this deprivation is called an infarction. Severe buildup of atherosclerotic plaque can occasionally narrow a blood vessel sufficiently to produce an ischemic stroke, but it is usually a clot formed at the site of a ruptured plaque that causes such a stroke. Both forms of the disease cause lasting damage to the delicate tissue of the brain. Stroke is therefore not only the third leading cause of death but also the greatest single cause of disability in most developed countries.[9]

FINDING THE CORRELATION

In 1995, researchers pooled together the results of 45 prospective studies investigating the potential link between cholesterol levels and the

ARTICLE SUMMARY

- Stroke is the leading cause of disability and the third leading cause of death in the United States and most developed countries. There are two types of stroke: ischemic and hemorrhagic. The more common ischemic stroke results from blockage of a blood vessel supplying the brain, whereas the more damaging hemorrhagic stroke results from rupture of such a vessel.
- As cholesterol levels increase, the risk of hemorrhagic stroke decreases and the risk of ischemic stroke increases.
- Stroke mortality is lowest at cholesterol levels between 180 and 200. Mortality increases substantially below 180 and above 240. Mortality is highest below 160 and above 300.
- Although cholesterol itself does not cause stroke, a diet high in polyunsaturated fat and low in antioxidants can make LDL-cholesterol within the blood vulnerable to oxidation. Oxidized LDL can contribute to the development of stroke.
- High blood pressure is a far more important contributor to stroke than high or low cholesterol.
- Animal fat and fatty fish are associated with a lower risk of stroke. Polyunsaturated fat and carbohydrates are associated with a higher risk of stroke.
- Exercise, stress management, proper control of oral or systemic infections, and adequate nutrition can lower the risk of stroke. Important protective nutrients include magnesium, potassium, antioxidants and adequate protein.

risk of stroke. Together, these studies examined this relationship in 450,000 people. The risk of stroke declined slightly with increasing cholesterol levels—an effect so small that it could easily have been due to chance.[5]

Most studies that began in the 1960s and 1970s, before the advent of computed axial tomography (the CAT scan), failed to distinguish between ischemic and hemmhoragic strokes. Many of them were also primarily designed to study heart disease. Since strokes occur at a later age than heart attacks, the average age of the subjects in these studies was too young and the incidence of stroke too low to detect modest associations with risk factors. These two problems obscured the true relationship of cholesterol to stroke.[10]

The Honolulu Heart Study enrolled over 8,000 Japanese American men between 1965 and 1968, measured their cholesterol levels and recorded which of them died of stroke over the following six years. The men were between the ages of 45 and 68 at the time of enrollment. Although CAT scans were not yet available, the researchers distinguished between ischemic and hemorrhagic strokes using signs and symptoms, findings at surgery or autopsy. As published in a 1980 issue of the journal *Stroke*, they found no association between serum cholesterol and ischemic stroke and an inverse association between serum cholesterol and hemorrhagic stroke, meaning a higher cholesterol level was associated with a lower risk of hemorrhagic stroke.[11]

A second report from the Honolulu Heart Study, published in 1994 with a fifteen year follow-up, however, demonstrated a direct association between serum cholesterol and ischemic stroke. The association was only found among subjects with cholesterol levels higher than 213 milligrams per deciliter (mg/dL) and was very small—over the course of ten years, subjects with cholesterol levels under 213 had a 2.5 percent chance of stroke and those with levels over 240 had a 3.2 chance of stroke.[12]

This report also showed that the incidence of stroke increases with age to a much greater degree than does the incidence of heart disease. In men younger than sixty, the ratio of heart disease to stroke was greater than three; in men older than sixty, it was less than two.[12] This pattern made the very meager association of ischemic stroke with serum cholesterol impossible to detect after only six years of follow-up.

The Multiple Risk Factor Intervention Trial (MR FIT) confirmed the findings of the Honolulu Heart Study in over 350,000 men. Those with cholesterol levels below 160 had three times the risk of hemorrhagic stroke as those with higher levels, while those with levels over 200 had a higher risk of ischemic stroke compared to those with lower levels. Between 200 and 240, the risk increased by only 20 percent. Those with levels above 280, however, had 2.5 times the risk of ischemic stroke as those with the lowest levels.[13]

The Eastern Stroke and Coronary Heart Disease Collaborative Research Group confirmed these findings in eastern Asian countries as well. The group pooled the results of 18 prospective studies conducted in China and Japan involving nearly 125,000 people. For every 23 point drop in serum cholesterol, the risk of ischemic stroke decreased by 23 percent and the risk of hemorrhagic stroke increased by 27 percent.[14]

It was then clear that the inability to

A QUESTION OF BIAS

A case control study of 180 subjects published in 1996 showed that LDL-cholesterol had a strong, positive association with ischemic stroke and that HDL-cholesterol had a strong, negative association with ischemic stroke. The authors were surprised to find, however, that saturated fat intake was over 25 percent lower in stroke patients than in controls.[19]

Case control studies are conducted retrospectively—that is, after the endpoints or results one is trying to learn about have already occurred. For this reason, they are subject to a number of biases to which prospective studies are not. The authors of this report suggested that the inverse association of stroke with the intake of saturated fat resulted from one of these biases: patients who have a history of high cholesterol and triglycerides would be counseled to avoid saturated fat. Therefore, they argued, the low intake of saturated fat would not have caused the disease; rather, the disease would have caused the low intake of saturated fat.

To support this proposition, they examined the medical records of a subset of their subjects to see whether a prior diagnosis of high lipid levels was associated with a low intake of saturated fat during the time of the study. Indeed, this was the case. They produced no evidence, however, that the diet followed rather than preceded the diagnosis. If it is true that the patients only followed a low-fat diet after being diagnosed with high lipid levels, it leaves open the question of why the diet did not lower their lipid levels or their risk of stroke. The suggestion that the low intake of saturated fat was caused by the disease rather than the disease by the low intake of saturated fat may have been easier to reconcile with the researchers' presuppositions, but it is more difficult to reconcile with the consistent evidence from prospective studies that total fat and animal fat consumption is inversely associated with the incidence of stroke.

discover a relationship of cholesterol levels to "stroke" resulted from the two opposing relationships of cholesterol levels to the two different types of stroke. The relationship between diet and stroke was less clear. Conventional wisdom would have had us believe that if high cholesterol levels increased the risk of ischemic stroke, so would a diet rich in total fat, saturated fat, and cholesterol. Conventional wisdom turned out to be very, very wrong.

A GREASY SITUATION

The authors of the 1980 Honolulu Heart Study report noted that stroke mortality and incidence among Japanese Americans was far lower than that among residents of Japan. They suggested that the difference owed to the substantially higher intakes of fat and protein among Japanese Americans.[11] These authors published another report from the same study in 1985, which found an inverse association between ischemic stroke and the dietary intake of total and saturated fat.[15]

Other authors examined 198 autopsies of fatal stroke within the same study. They dissected small and large blood vessels of the brain and assessed the severity of atherosclerosis within them. Among 104 men who also had heart disease, there was no association between atherosclerosis and any dietary factors. Among the other 94, intake of fish was inversely associated with atherosclerosis of the small arteries and intake of animal protein and total fat was inversely associated with atherosclerosis of the large arteries. Intake of carbohydrates, by contrast, was positively associated with atherosclerosis of the large arteries.[16]

A decade later, the Framingham Heart Study found similar results

TAKE YOUR PICK

In his book *Eat to Live*, Dr. Joel Fuhrman argues that avoiding animal foods is an important strategy for stroke prevention. Although low cholesterol levels increase the risk of hemorrhagic stroke, this type only represents eight percent of the total; since the vast majority of strokes are of ischemic origin, he maintains, eating a cholesterol-lowering diet will lower the risk of stroke.[20]

This point of view suffers from three fundamental flaws: first, the data indicates that the rate of hemorrhagic stroke is low in western countries because our cholesterol levels tend to be high; second, hemorrhagic strokes are far more dangerous than ischemic strokes; and third, as already shown, animal foods are associated with a decrease—not an increase—in the risk of ischemic stroke.

Among Japanese American men living in Hawaii, whose intakes of animal fat and protein are lower than those of mainland Americans but higher than those of men living in Japan, hemorrhagic stroke constitutes 25 percent of all strokes.[11] In China and Japan, where serum cholesterol levels correspond to the bottom two thirds of the range of western levels, hemorrhagic stroke constitutes 42 percent of all strokes. The authors of a collaborative research project pooling the results of 18 studies conducted in this region found that as cholesterol levels increased, hemorrhagic stroke constituted a lower proportion of total strokes; as cholesterol levels decreased, hemorrhagic stroke constituted a higher proportion of total strokes.[14] Although the risk of hemorrhagic stroke may be low in the United States, the rates of this more dangerous type of stroke would almost certainly be higher if we vigorously maintained low cholesterol levels by eating vegan or semi-vegan diets.

Hemorrhagic stroke is much more dangerous than ischemic stroke. Victims of the former suffer greater neurological deficits, are more likely to be institutionalized, and are four times more likely to die within thirty days than victims of the latter. A recent comparison of the two types showed that only seven percent of ischemic stroke victims die within thirty days, whereas 28 percent of hemorrhagic stroke victims die within the same period of time.[21] Moreover, low cholesterol levels are associated with decreased survival even from ischemic stroke. A Scottish study found that every 40 point decrease in serum cholesterol was associated with a nine percent increase in the risk of mortality for all types of stroke.[22]

In the MR FIT trial, involving over 350,000 men, stroke mortality was lowest among those with cholesterol levels between 180 and 200. Substantial increases in mortality occurred below 180 and above 240. The largest increases in mortality occurred among those with levels below 160 and over 300.[13] Although these correlations do not demonstrate that the cholesterol levels actually cause the increase or decrease in risk, in the absence of a comprehensive understanding of causation they may justify optimizing these levels with exercise, nutritional supplements or moderation of carbohydrate intake. They cannot, however, justify a diet low in animal protein and fat when the evidence has consistently shown the consumption of these foods to be associated with an equal or lower risk of stroke.

In the Diet and Reinfarction Trial (DART), subjects who reduced their total fat intake and replaced saturated fat with polyunsaturated fat doubled their risk of suffering a fatal stroke.[23] The reduction of total fat was small, from 35 percent of calories to 32 percent of calories. The increase in the polyunsaturated-to-saturated fat ratio was larger: it doubled from 0.4 to 0.8. These results taken together suggest that maintaining low cholesterol levels with diets low in fat and saturated fat is likely to increase the risk and severity of stroke.

among 800 men whom the researchers followed over the course of 19 years. Although there was no association of stroke with polyunsaturated fat consumption, each additional three percent of calories from total fat was associated with a fifteen percent decrease in the risk of ischemic stroke; each additional one percent of calories from monounsaturated fat was associated with an eleven percent decrease in risk; and each additional one percent of calories from saturated fat was associated with a nine percent decrease in risk.[17]

In the last decade, some studies have shown no relationship between the intake of animal fat and the risk of ischemic stroke, but most have continued to show that the risk of this disease is inversely associated with the intake of animal fat and fish.[18]

HOW LOW CAN IT GO?

Early trials with cholesterol-lowering drugs were less than promising. A 1993 report pooled together the results of 13 trials conducted between 1966 and 1992 involving over 45,000 men. Cholesterol lowering had no effect on the incidence of stroke. There was a general tendency for it to decrease the risk of nonfatal stroke and increase the risk of fatal stroke, but the only trials in which the magnitudes of these differences were strong enough to be distinguished from the effects of chance were those that used the drug clofibrate. Clofibrate belongs to a class of drugs, called fibrates, that increase the excretion of lipids into the bile. Treatment with clofibrate more than doubled the risk of fatal stroke. The only trial that specifically reported the effect of treatment on hemorrhagic stroke used another fibrate called gemfibrozil. Treatment with this drug resulted in five times the risk of fatal hemorrhagic stroke.[25]

Hemorrhagic stroke is four times as deadly as ischemic stroke,[21] and survival of both types is positively associated with cholesterol levels.[22] The results of the early trials with cholesterol-lowering drugs may well reflect a tradeoff between hemorrhagic and ischemic stroke as well as a decreased ability to survive either type.

The results of later trials with statin drugs proved very different. A 2004 report that pooled together the results of 120 lipid-lowering trials, including 24 using statins, showed that treatment with statins lowered the risk of stroke by 18 percent. Among ten trials that distinguished hemorrhagic from ischemic stroke, treatment with lipid-lowering therapy in general increased the risk of hemorrhagic stroke by 21 percent, whereas statins themselves increased this risk by only 3 percent; neither effect was large enough to be distinguished from the effect of chance.[26] Statins are apparently more successful than older cholesterol-lowering drugs because they more effectively reduce the risk of ischemic stroke and less severely aggravate the risk of hemorrhagic stroke.

Some authors have suggested that statins effectively reduce the risk of stroke because they are more than twice as effective at reducing cholesterol levels compared to older drugs. Indeed, the older drugs only reduced cholesterol levels by an average of eight percent. Statins, by contrast, have reduced them by an average of 22 percent.[27] Moreover, the reduction of LDL in these trials corresponds to both the reduction in the risk of stroke and the reduction in the degree of atherosclerosis within the arteries of the neck that supply the brain.[28]

In the Diet and Reinfarction Trial (DART), subjects who reduced their total fat intake and replaced saturated fat with polyunsaturated fat *doubled* their risk of suffering a fatal stroke.

A CONVENIENT CHOICE OF WORDS

On February 20, 2007, *Science Daily* reported that researchers had shown total cholesterol levels to predict the risk of stroke in women. Women with the highest cholesterol levels, according to the article, had twice the risk of stroke as women with lower levels. The researchers claimed their findings underscored "the importance of cholesterol levels as a risk factor for stroke, even if you have no history of heart disease and are otherwise healthy."[8]

The article left out one important fact: the study only looked at ischemic stroke.[24] Did the women with high cholesterol levels have not only twice the risk of ischemic stroke but also half the risk of the much more dangerous and fatal hemorrhagic stroke? Was the incidence of total stroke any higher or lower in women with high cholesterol levels? We simply do not know; the study did not address the question.

If the key to the success of statins were limited simply to cholesterol reduction, however, we would expect them to not only lower the risk of ischemic stroke to a greater degree than other drugs, but also to raise the risk of hemorrhagic stroke to a greater degree than other drugs. Instead, we find that statins have no effect on the risk of hemorrhagic stroke at all. Clearly, these drugs are affecting the risk of stroke by some means other than lowering cholesterol.

CORRELATION VERSUS CAUSATION

It is a fundamental principle of science that correlation does not prove causation. The risk of ischemic stroke is higher among people with high cholesterol levels, but this does not in and of itself show that high cholesterol levels cause ischemic stroke. If a drug that lowers cholesterol also lowers the risk of this disease and the reduction in risk is proportionate to the reduction in cholesterol, this provides evidence that high cholesterol causes the disease—as long as the drug only lowers cholesterol. With statins, however, this is not the case.

Statins do not directly inhibit the synthesis of cholesterol. Instead, they inhibit the synthesis of mevalonate (see Figure 1). Cells use mevalonate to synthesize a number of different chemicals, only one of which is cholesterol. The degree of cholesterol reduction is dependent on the degree of mevalonate reduction; it therefore can also act as a marker for the degree of reduction of other products made from mevalonate. Before concluding which of these products underlies the efficacy of statins, we must look beyond statistical correlations and examine more deeply the molecular mechanisms of the disease process.

NITRIC OXIDE

One of the many products of mevalonate activates the enzyme Rho. Rho is a stress signal which, in response to inflammation, changes the shape and tension of the protein fibers that form the cell's skeleton.[29] Rho also decreases the production of endothelial nitric oxide synthase (eNOS), the enzyme that synthesizes nitric oxide.[30] Nitric oxide itself is a gas that

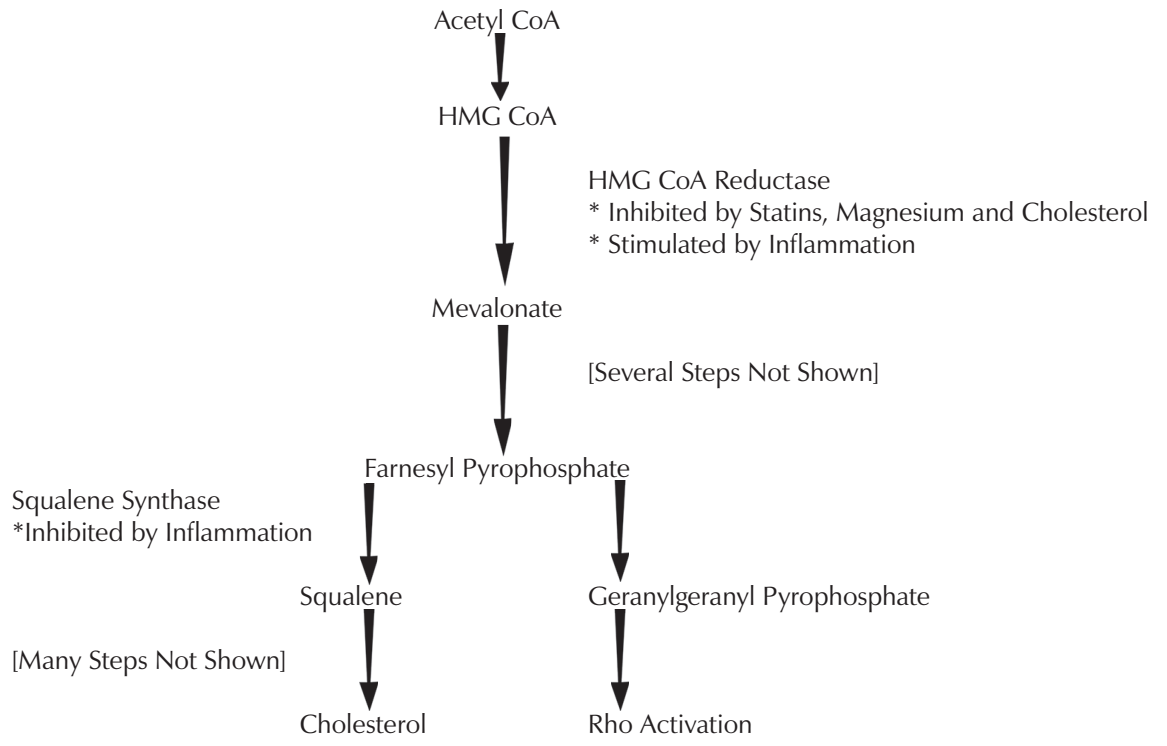

FIGURE 1. CHOLESTEROL SYNTHESIS AND RHO ACTIVATION
Many steps in the cholesterol synthesis pathway are omitted in the figure for the sake of simplicity. The key regulated enzymes within this pathway are HMG CoA reductase and squalene synthase. The former converts HMG CoA to mevalonate; the latter converts farnesyl pyrophosphate to squalene. The synthesis of squalene is the first step in this pathway that is committed to the synthesis of cholesterol. Statins suppress both the synthesis of cholesterol and the activation of Rho by inhibiting HMG CoA reductase. Inflammation increases cholesterol levels by stimulating HMG CoA reductase; because it also inhibits squalene synthase, however, most of the mevalonate it produces is diverted into other pathways, such as Rho activation. Because they inhibit HMG CoA reductase, it is possible that magnesium and dietary cholesterol also suppress Rho activation.

dilates blood vessels, relaxing the smooth muscle cells within their walls and increasing the flow of blood. It also decreases the adhesion of white blood cells to the lining of these vessels, the migration of smooth muscle cells to the sites of atherosclerotic lesions and the formation of blood clots, all of which are involved in the disease process that leads to ischemic stroke.[31]

By inhibiting the activation of Rho,[30] statins greatly increase the levels of eNOS and the amount and activity of nitric oxide within the blood vessel lining.[32,33] When researchers experimentally induce a stroke in mice with normal cholesterol levels, prior administration of statins greatly increases the flow of blood within the brain and reduces the damage to brain tissue and the neurological deficits that follow. The benefit occurs even when the dose and length of administration is insufficient to reduce cholesterol; in mice that are genetically engineered to lack the eNOS enzyme, by contrast, statins have no effect.[34] These drugs clearly protect against stroke in ways that are dependent on eNOS and have nothing to do with cholesterol.

There is also evidence that the inhibition of Rho offers additional protection against stroke in some animal models by increasing the body's ability to dissolve blood clots independently of both eNOS and cholesterol.[35]

Because both Rho activation and cholesterol synthesis depend on the availability of mevalonate, the ability of a given drug or a given dose of that drug to inhibit one substance is going to correlate with its ability to inhibit the other. These things make it clear that a protective effect of statins—even one that correlates with cholesterol reduction—is not necessarily evidence that cholesterol itself causes stroke.

IS CHOLESTEROL IRRELEVANT?

It would be a mistake to conclude from these observations that cholesterol is entirely irrelevant to the process that leads to ischemic stroke. Most likely, the level of low-density lipoprotein (LDL) cholesterol is a loose indicator of the level of oxidized LDL, a particle that can, indeed, contribute to the disease process that underlies this form of stroke.

An LDL particle carries cholesterol and triglycerides through the blood within a membrane made of lipid and protein. The membrane consists primarily of many phospholipids interwoven with one large protein molecule; although most of the cholesterol is contained within the core of the particle, a small amount is also dis-

CHRONIC NITRIC OXIDE DEFICIENCY

Many dietary, lifestyle, and physiological factors directly or indirectly regulate nitric oxide levels. Nitric oxide relaxes blood vessels, increases blood flow and inhibits the formation of atherosclerotic plaque. A chronic deficiency of this compound may be among the most important causes of atherosclerosis and ischemic stroke.

The enzyme endothelial nitric oxide synthase (eNOS) produces nitric oxide within the lining of the blood vessels. Many factors regulate the production of this enzyme and thereby indirectly regulate the production of nitric oxide itself. Sheer stress stimulates the production of eNOS and may be an important mediator of the atherosclerotic process. The blood vessel lining experiences this type of stress as blood runs parallel to it. Exercise increases sheer stress because it causes the blood to move more vigorously, and thereby increases the production of eNOS.[36] Atherosclerotic lesions tend to develop at specific sites where disturbed blood flow increases the force running perpendicular to the blood vessel lining and decreases the force running parallel to it.[37]

Oxidized LDL-cholesterol and insufficient oxygen decrease the production of eNOS. Free radicals are able to destroy nitric oxide itself after eNOS has synthesized it.[38]

The cellular enzyme Rho is capable of powerfully suppressing the production of eNOS. Ordinarily, the vast majority of Rho within a cell is inactivated. One of the products of the cholesterol synthesis pathway, geranylgeranyl pyrophosphate (GGPP), is responsible for activating it. Cellular enzymes synthesize GGPP from mevalonate. When the cell is producing more mevalonate than it uses for cholesterol synthesis, more GGPP is available for the activation of Rho (see Figure 1). Dietary nutrients such as cholesterol and magnesium, as well as the cholesterol that we synthesize ourselves, keep the production of mevalonate from exceeding the level needed by the cell.[39] Inflammation, however, not only increases the production of mevalonate but inhibits its conversion to cholesterol. When researchers fed hamsters endotoxin, a pro-inflammatory chemical released from the breakdown of bacterial cell walls, it increased the production of mevalonate by a factor of ten but only increased the production of cholesterol by a factor of two.[40] The result of chronic inflammation is a small excess of cholesterol and a much larger excess of other mevalonate products such as GGPP, which our cells then use to activate Rho.

Magnesium deficiency, chronic inflammation—and perhaps even a lack of dietary cholesterol—might contribute to chronic activation of Rho and suppression of nitric oxide synthesis. Inadequate exercise, excessive consumption of easily oxidized materials such as polyunsaturated fat and inadequate dietary antioxidants may further aggravate the deficiency of this important compound and thereby facilitate the atherosclerotic process and the impairment of blood flow that characterize an ischemic stroke.

Animal experiments have shown that diets high in animal fat and cholesterol reduce the incidence of stroke in rats with high blood pressure.

tributed throughout the membrane. The amino acids within the protein and the unsaturated fatty acids within the phospholipids are vulnerable to oxidation; they are also protected by certain vitamins, polyphenols and other antioxidants that are carried within the lipoprotein. When we refer to the oxidation of LDL, we refer primarily to the oxidation of the phospholipids and protein at the surface of the particle rather than the cholesterol within its core.

Oxidation of LDL causes it to accumulate in certain scavenger white blood cells called macrophages—an instrumental event in its accumulation within atherosclerotic plaque.[41] Oxidized LDL also suppresses the production of eNOS. Since nitric oxide inhibits the oxidation of LDL, the loss of nitric oxide and the oxidation of LDL could produce a vicious cycle.[33] When the level of sugar in the blood rises, it can similarly damage LDL in a process called glycation. Although less powerfully than oxidized LDL, glycated LDL also accumulates in macrophages[42] and suppresses the production of eNOS.[43]

Selectively filtering LDL from the blood of patients with high cholesterol—most of which is oxidized—appears to improve nitric oxide production and blood flow.[44] Researchers have unfortunately only tested the effect of this treatment on these parameters in small, uncontrolled trials.

We cannot with any confidence quantify the contribution of oxidized LDL to the development of stroke, but the evidence strongly suggests that it plays some part. The very meager association between cholesterol levels and ischemic stroke probably reflects both the indirect association of cholesterol with chronic inflammation (see sidebar on page 34) and the causal contribution of oxidized LDL.

LOW CHOLESTEROL AND HEMORRHAGIC STROKE

Whether and how low cholesterol causes the increased risk of hemorrhagic stroke with which it is associated is an open question. Animal experiments have shown that diets high in animal fat and cholesterol reduce the incidence of stroke in rats with high blood pressure.[17] In humans, the inverse association between cholesterol levels and hemorrhagic stroke primarily exists among those with diastolic blood pressure above 90 millimeters mercury (mm Hg).[13] Cholesterol probably protects against hemorrhage by strengthening and stabilizing the blood vessel walls, especially when these walls need extra strength to withstand the constant onslaught of high blood pressure.

PRACTICAL PREVENTION

Despite the opposing relationships of cholesterol levels to ischemic and hemorrhagic strokes, the distribution of risk is not even across cholesterol levels. In the large MR FIT trial, for

BLOOD PRESSURE AND STROKE

The overwhelmingly powerful and consistent risk factor for stroke is not high cholesterol but high blood pressure.

The 1980 report of the Honolulu Heart Study found the risk of ischemic stroke to increase with systolic blood pressure beginning at the lowest levels—the risk was lowest at levels under 121 millimeters mercury (mm Hg). The relationship of blood pressure to hemorrhagic stroke was similar but the lowest risk existed at levels between 122 and 134 mm Hg. The authors concluded that, "It seems to be a universal finding among all stroke epidemiology studies that the single most important risk factor for stroke, whether of cerebral infarction or intracranial hemorrhage, is hypertension."[15]

In a 1995 report of the pooled results of 45 prospective studies involving 450,000 people, the risk of stroke had no association with cholesterol, but it increased consistently with diastolic blood pressure from the group with the lowest to the group with the highest. The difference was dramatic: subjects with a diastolic blood pressure of 102 mm Hg had five times the risk of stroke as those with a diastolic blood pressure of 75 mm Hg.[5]

In a 1998 report of the pooled results of 18 prospective studies conducted in eastern Asia involving nearly 125,000 people, the effect of blood pressure was even more pronounced. While serum cholesterol had modest but opposing associations with ischemic and hemorrhagic strokes that canceled each other out, the risk of both types of stroke increased consistently as diastolic blood pressure increased. Those with levels higher than 110 mm Hg had 13 times the risk of stroke as those with levels lower than 79 mm Hg.[14]

Clearly, maintaining optimal blood pressure is far more important to preventing stroke than maintaining low cholesterol levels.

THE WORK OF WESTON PRICE: ITS ENDURING VALUE

Weston Price promoted two theories about the relationship of nutrition and oral health to degenerative disease, which have long been ignored but have more recently gained support. In his earlier career, Price conducted 25 years of research demonstrating the ability of oral pathogens to cause cardiovascular and other systemic diseases. His work focused primarily on the tendency of the root canal procedure to facilitate this process.[45] In his classic work on nutrition, *Nutrition and Physical Degeneration*, he connected nutritional status during development to deformities of the oral palate as well as to the risk of tuberculosis. Price believed that developmental deformities of the chest cavity—produced by the same nutritional causes as the deformities of the oral palate—made a person more vulnerable to the tuberculosis bacterium. For these reasons, he placed a special emphasis on the importance of nutritional preparation for and support of pregnancy and lactation—practices he universally observed among the healthy indigenous groups he studied.[2] Modern science is now rediscovering the links between vascular disease and oral health and fetal nutrition.

ORAL HEALTH AND STROKE

Recent research has been focusing on the association of vascular disease with periodontitis rather than with root canals, but nevertheless has been confirming the general principle observed by Price, namely that oral pathogens can cause systemic, degenerative diseases. Oral pathogens and immune cells specific to them inhabit arterial plaque. A number of studies have associated periodontitis with the severity of atherosclerosis and the incidence of heart disease and stroke. The pooled results of these studies conducted up to the year 2004 associate the presence of periodontitis with a 50 percent increase in the risk of ischemic stroke. Several studies have associated the presence of severe periodontitis with a nearly three-fold increase in risk of total stroke or fatal ischemic stroke. Among those strokes that are preceded by a fever, dental infection is associated with a nine-fold increase in risk.[46]

Preliminary evidence suggests that periodontitis increases the systemic marker of inflammation known as C-reactive protein as well as total and LDL-cholesterol. Intensive treatment of periodontitis using antibiotics decreases these levels when compared to standard treatment without antibiotics.[47] Although it is possible that LDL and total cholesterol may be an important part of the body's response to inflammation, it is also possible that their increase is largely coincident with the increase in mevalonate production used for other purposes, such as the activation of Rho, an important mediator of the stress response (see sidebar on page 34).

Oral pathogens may cause immune cells specific to them to directly adhere to the blood vessel lining and initiate atherosclerosis. By increasing the activation of Rho, however, we should also expect systemic inflammation to interfere with nitric oxide functioning and thereby contribute to atherosclerosis by a second mechanism.

FETAL NUTRITION AND STROKE

Recent research has focused on the association of fetal nutrition with certain physical deformities and the risk of vascular diseases rather than the risk of tuberculosis; nevertheless, this research confirms the general principle that early nutritional status has a lasting influence on the risk of degenerative disease. Inadequate nutrition during various stages of fetal development causes corresponding changes in various physical parameters; these parameters are in turn associated with the risk of diabetes, heart disease and stroke.

Soon after conception, inadequate nutrition causes the womb to reallocate cells from the fetus to the placenta in order to extract more nutrition from the blood supply of the mother; the inevitable result is a loss of raw material from which to generate fetal tissue. During later stages of growth, the fetus compensates for inadequate nutrition by sacrificing the supply of nutrients to muscles and internal organs such as the liver and pancreas in favor of supplying the brain; the exception is the left ventricle of the heart, which may grow larger than is normal in order to pump hard enough to supply the brain with extra blood. This general pattern of compensation results in a reduced ratio of body length to head circumference (that is, short stature with a proportionately larger head).[48]

Excluding premature births, low birth weight is associated with a three-fold increase in the risk of type-2 diabetes, a two-fold increase in the risk of heart disease and a modest increase in life-long blood pressure. A reduced length-to-head circumference ratio is associated with defects in the regulation of cholesterol and blood clotting metabolism. One study found stroke mortality to be more common among men who were born to mothers with flat, bony pelvises (considered abnormal), suggesting a relationship between the mother's childhood nutrition and her offspring's risk of stroke.[48]

These findings illustrate the enduring importance of Price's research and the benefits that would accrue should modern science incorporate his research into the paradigm with which it approaches questions of health and disease.

Nutrient-dense animal foods are gifts for which we should all be grateful. They supply the body with the resources it needs both to build itself up during youth and to maintain its integrity into old age.

example, stroke mortality was lowest between 180 and 200. Mortality substantially increased below 180 and above 240; it was highest below 160 and above 300.[13] While it is unlikely that total or LDL cholesterol themselves play a major role in the development of stroke, oxidation or glycation of that LDL will contribute to the atherosclerotic process and the breakdown of nitric oxide functioning. It therefore may be a sensible precaution to use exercise and, where needed, nutritional supplements to maintain moderate levels of cholesterol and to eat a diet rich in antioxidants to protect that cholesterol from damage.

Magnesium is essential to the regulation of cholesterol synthesis. Like statin drugs, magnesium inhibits the enzyme that produces mevalonate, a precursor to cholesterol. Unlike statins, however, cellular enzymes also use magnesium to increase the production of mevalonate when needed. Supplements with this mineral can lower levels of LDL and raise levels of HDL; since they provide the body with the resources it needs to regulate these levels rather than interfere with the body's physiological processes, they are unlikely to exhibit any of the adverse effects exhibited by statins.[39]

Diets low in fat or animal products should not be used to lower cholesterol for the prevention of ischemic stroke. The available data clearly indicate that these foods are associated with a decreased risk, not an increased risk, of this type of stroke.[17] Since carbohydrate intake is associated with atherosclerosis of the large arteries within the brain,[16] eating lower amounts of carbohydrates would be a wiser dietary modification.

Additionally, increasing polyunsaturated fat intake in the DART trial doubled stroke mortality.[23] Polyunsaturated fatty acids are vulnerable to oxidation within the body[49] and their incorporation into lipoproteins would make those lipoproteins more likely to oxidize. Since there is evidence that fatty fish is protective against stroke,[18] however, we should give priority to reducing omega-6 fatty acids from vegetable oils rather than the elongated omega-3 fatty acids from fish. Even among traditional fats, an overemphasis on olive oil, flax oil, chicken fat, or even very large amounts of lard could lead us to consume an excess of polyunsaturated fat. These nourishing foods have their place, but more saturated fats like butter, tallow and coconut oil should form the mainstay of a diet rich in fat.

Exercise, antioxidant-rich foods and proper treatment of chronic oral or systemic infections will help maintain the rich supply of nitric oxide that blood vessels need. Since the amino acid L-arginine is the raw material for nitric oxide production, adequate protein is also important.

Blood pressure is far more critical to the development of stroke than is cholesterol. Exercise, stress management and adequate intake of potassium can be useful in controlling blood pressure. Some individuals may also need to moderate their intake of salt.

Were it true, as many contend, that the consumption of cholesterol-rich animal foods such as butter, egg yolks and organ meats puts one at risk for stroke, we would be forced to make the difficult choice between the diet that builds robust and sturdy bodies and the diet that allows us to live safely into old age. Thankfully, there is no evidence that these foods will do anything but protect us from stroke. Nutrient-dense animal foods are gifts for which we should all be grateful. They supply the body with the resources it needs both to build itself up during youth and to maintain its integrity into old age.

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The Weston A. Price Foundation has over 400 local chapters worldwide, which help consumers find raw milk, meat, eggs and other nutrient-dense products from local pasture-based farms. Local chapters host potluck dinners, cooking classes, seminars and speakers. Visit www.westonaprice.org to find the local chapter closest to you.

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CoEnzyme Q_{10} for Healthy Hearts

by John Williamson Cameron

Coenzyme Q_{10} is a fat-soluble vitamin-like substance present in every cell of the body, which serves as a coenzyme for several of the key enzymatic steps in synthesis of ATP on which production of energy within all cells depends. In its reduced form, called ubiquinol, Coenzyme Q_{10} is a potent anti-oxidant that protects cells from damage by free radicals. It also regenerates other anti-oxidants, including vitamins C and E.

CoQ_{10} was first isolated from beef heart mitochondria in 1957, and it was first synthesized in 1958. In 1972, Gian Paolo Littaru of Italy along with Professor Karl Folkers of the US documented a deficiency of CoQ_{10} in human heart disease. By the mid 1970s, the Japanese had perfected the industrial technology to produce pure CoQ_{10} in quantities sufficient for larger studies.

In the early 1980s, scientists were able to conduct a number of clinical trials due to the availability of CoQ_{10} in large quantities from Japan and from the newly acquired capacity to measure CoQ_{10} in blood and tissue. Professor Karl Folkers received the Priestly Medal in 1986 and the National Medal of Science from President Bush in 1990 for his work with CoQ_{10} and other vitamins. Internationally, a dozen placebo controlled studies on treatment of heart disease with CoQ_{10} have confirmed the effectiveness of CoQ_{10} in improving heart muscle function while producing no adverse side effects or drug interactions.

John Cameron, a Professional Engineer with a master's degree in environmental engineering, worked as a consultant in the field of water and wastewater systems design until his retirement in 1984. In recent years, John has applied his problem-solving experience to the analysis of often conflicting nutritional and biomedical theories.

Because CoQ_{10} is a natural substance, it can not be patented, so patent-protected profits have not been available to educate physicians and the public about the proven benefits of CoQ_{10} for the treatment of heart failure. The lack of patent-protected profits has also prevented the automation of the complex, seventeen-step process required for measurement of CoQ_{10} blood levels by gas chromatography. Even today, only a half dozen labs in the United States are capable of testing CoQ_{10} levels. CoQ_{10} has shown great promise in preliminary studies in treatment of many other conditions, but lack of patent-protected profits and lack of an automated economical system for testing CoQ_{10} have severely limited large-scale clinical studies.[1]

CoQ_{10} DEFICIENCY

CoQ_{10} is present in small amounts in a wide variety of foods but the dominant source of CoQ_{10} in humans is biosynthesis. CoQ_{10} required by blood cells is synthesized in the liver, while the majority of CoQ_{10} synthesis occurs in cells throughout the body.

The biosynthesis of CoQ_{10} is a complex multi-step process that requires at least seven vitamins (B_2, B_3, B_5, B_6, B_{12}, C and folic acid) and several trace elements, and is, by its nature, highly vulnerable. Sub-optimal nutrient intake impairing CoQ_{10} synthesis is almost universal, and deficiency of any of the vitamins or trace elements required for CoQ_{10} synthesis can cause deficiency of CoQ_{10}. Decreased absorption of nutrients necessary for synthesis of CoQ_{10} can be caused by aging,[2] digestive problems such as irritable bowel syndrome,[3] liver diseases,[4] and many common prescription drugs[5] including oral contraceptives[6] and HRT.[7]

CoQ10 synthesis can also be impaired by the widely prescribed HMG-CoA reductase inhibitors (statins) which block the synthesis of melovonic acid and thereby block synthesis of cholesterol, CoQ_{10} and other compounds, such as squalene and isoprene, all of which are important to health.

Increased utilization of CoQ_{10} by the body is the cause of low blood CoQ_{10} levels seen in many conditions, including excessive exertion,[1] hyperthyroidism,[8] aortic valve stenosis,[9] hypertrophic cardiomyopathy,[10] diabetes,[11] rheumatoid arthritis,[12] lupus,[13] HIV,[1] asthma,[14] certain cancers,[15] hyperlipidemia,[16] and atherosclerosis.[17]

Researchers now consider CoQ_{10} deficiency to be a significant cause of heart failure and coronary artery disease. CoQ_{10} deficiency also is thought to contribute to cancer, infertility in men[18] and migraine headache.[19]

Sub-optimal nutrient intake impairing CoQ_{10} synthesis is almost universal.

ARTICLE SUMMARY

- $CoenzymeQ_{10}$ is a substance synthesized in all cells of the body which is necessary for synthesis of ATP, the substance that provides energy to all cells.
- $CoenzymeQ_{10}$ deficiency can be caused by reduced synthesis of CoQ_{10} due to nutrient deficiencies or statin drug use, or by increased utilization of CoQ_{10} by the body due to certain diseases, aging, or by an inflammatory atherogenic physiological state resulting from excess consumption of carbohydrates, calories and omega-6 fatty acids and inadequate intake of omega-3 fatty acids.
- The filling or diastolic phase of the heart cycle uses more energy than the contraction or systolic phase and CoQ_{10} deficiency can cause impairment of the filling cycle of the heart leading to heart failure if not corrected. CoQ_{10} supplementation to provide adequate CoQ_{10} levels can prevent diastolic heart failure. For those with diastolic dysfunction, CoQ_{10} supplementation will improve diastolic function and can normalize heart function if irreversible damage to heart muscle has not occurred. CoQ_{10} also regenerates alpha-tocopherol to the active, reduced form.
- Ubiquinol, the reduced form of CoQ_{10}, is a potent anti-oxidant that helps protect cells of the body from oxidative damage. Increased oxidative stress due to aging, poor diet or inflammatory disease results in decreased levels of ubiquinol and total CoQ_{10}. The level of oxidative stress can be reduced by adoption of a good diet and by supplementation with CoQ_{10} to increase the anti-oxidant protection available. For those who have coronary artery disease, these measures will reduce the progression of the disease and reduce the risk of plaque rupture.
- Those with conditions such as type-2 diabetes, asthma, arthritis and hypertension, and those over 65 years of age or taking statin drugs, are likely to be CoQ_{10} deficient and would therefore benefit from CoQ_{10} supplementation. CoQ_{10} is a substance that occurs naturally in the body so there are no significant side effects. While it would be desirable to have blood levels tested, there are very few labs in the US capable of accurately testing CoQ_{10} levels.

Much of the media coverage and advertising on the subject of heart disease is misleading, unbalanced or erroneous.

OVERVIEW OF HEART FAILURE

Much of the media coverage and advertising on the subject of heart disease is misleading, unbalanced or erroneous. The term "cardiovascular disease" is often carelessly used in public discourse as though it were synonymous with "heart disease," thereby implying that all heart disease is due to blockage of coronary arteries. As a result of widespread misinformation, many incorrectly believe that all heart failure is the result of coronary artery disease.

The primary cause of impaired systolic function is heart muscle damage caused by reduced blood flow due to coronary artery disease. While the damage can result from chronic reduced blood flow through narrowed arteries, it is most often due to plaque rupture, which causes a sudden complete artery blockage that precipitates a heart attack. The common measure of systolic function is "ejection fraction," the percent of ventricle volume discharged with each heart contraction. Systolic function is considered impaired when the ejection fraction is less than 45 percent compared to the normal range of 55 to 70 percent and the borderline range of 45 to 54 percent. Clinical trials of heart disease treatments have focused for decades on young men with "impaired" systolic function. It is now recognized that over 90 percent of cardiac deaths occur in men and women over 65 years of age, and more than half of cardiac deaths occur in those with "normal" systolic function.[20]

Past focus on impaired systolic function occurred largely because the importance of the filling phase of the heart cycle, the diastolic phase, had been little studied and was poorly understood. The term "diastolic heart failure," meaning heart failure resulting from impaired diastolic function in those with "normal" ejection fraction, first appeared in clinical studies about ten years ago, and one medical texts described diastolic heart failure as a "new" type of heart failure.[21] Diastolic dysfunction leading to diastolic heart failure is due primarily to deficiency of CoQ_{10} which causes energy starvation of the heart.

Whenever systolic function is impaired, diastolic dysfunction is also present, and the degree of diastolic dysfunction has been found to more accurately predict the prognosis of systolic heart failure than ejection fraction, the common measure of systolic impairment. While coronary artery disease is the primary cause of heart failure with impaired systolic function, the conditions that lead to coronary artery disease also increase CoQ_{10} utilization and can result in CoQ_{10} deficiency and diastolic heart failure.

The mortality rate of heart failure cases with impaired systolic function is almost double the mortality rate of diastolic heart failure, but the total number of deaths due to the two conditions is approximately equal due to the greater number of those with diastolic heart failure.[20]

CAUSES OF CORONARY ARTERY DISEASE (CAD)

There are many factors that contribute to development of atherosclerosis, but the primary cause is the profound changes that have taken place in the American diet during the past century, particularly:

- Imbalance in consumption of essential fatty acids (too little omega-3 as in fish, too much omega-6 as in corn oil, etc.) which has an adverse effect on the balance of eicosanoids (localized tissue hormones) that control many functions of the body and mind.
- Excess consumption of carbohydrates, particularly sugars and high fructose corn syrup.
- Eating too much (too many calories).
- Free radicals in processed liquid vegetable oils and *trans* fatty acids partially hydrogenated vegetable oils.
- Nutrient deficiencies.[22]

The typical American diet results in increased production of triglycerides (TG), decreased levels of HDL-cholesterol, and a preponderance of small, dense LDL-cholesterol particles, a condition referred to as the atherogenic lipid triad. The increase in the atherogenic potential of LDL arises from the increase in the number of small, dense LDL particles, not from the cholesterol content per se. Small dense LDL particles more easily penetrate the arterial wall, initiating atherosclerotic injury, which leads to the development of inflammation and plaque.[23]

The development of highly atherogenic, small dense LDL particles is thought to be due

to high insulin levels and excess triglycerides that result from excessive carbohydrate and caloric intake and from an imbalance of essential fatty acids.[24,25] Researchers have noted a high degree of correlation between the TG/HDL ratio, insulin intolerance, particle size and the presence of coronary artery disease. Because TG and HDL are commonly measured, the ratio TG/HDL is considered proxy for LDL particle size and a good indicator of the presence of coronary artery disease (CAD) and risk of adverse coronary events.[26]

High insulin levels cause insulin intolerance and diabetes, and together these greatly increase the risk of coronary artery disease. In one study, 58 percent of CAD patients were found to be insulin resistant, including 22 percent with diabetes. Diabetes and insulin intolerance greatly increase the risk of cardiac death.[27]

The increased oxidative stress resulting from coronary artery disease increases utilization of ubiquinol, the reduced form of CoQ_{10}, resulting in low levels of both total CoQ_{10} and ubiquinol. Levels of ubiquinol in those with CAD have been found to be inversely proportional to triglyceride levels.[15] Diabetes causes severe depression of CoQ_{10} levels, and because development of CAD usually occurs slowly, diastolic heart failure is common in diabetic patients.

Other factors that contribute to atherosclerosis are smoking, inactivity and stress. The stress factor can result from an imbalance of essential fatty acids due to excess production of "bad" eicosanoids (hormones) that promote the "fight or flight response" and which cause an exaggerated response to normal daily stress. Exaggerated stress response can result in many physiological changes that contribute to coronary artery disease, including increased adrenaline production leading to constriction of blood vessels, increased blood clotting factors, and stimulation of smooth muscle cell production.[22]

In addition, *trans* fatty acids and nutrient deficiencies, particularly deficiency in vitamin

CoENZYME Q_{10} AND CANCER

Abnormally low plasma CoQ_{10} levels have been found in patients with melanoma and cancer of the breast, lung and pancreas.[1] Early studies have hinted that CoQ_{10} may be effective in treating some cancers. In one study, six of 32 patients who took 90 mg per day of CoQ_{10} showed partial tumor reduction. One of the six then began taking 390 mg per day, and within two months there was no mammographic evidence of the tumor.[2] An additional three patients undergoing conventional treatment took 390 mg of CoQ_{10} over three to five years. The results: in patient one, liver metastases disappeared; in patient two, the tumor in the pleural cavity disappeared; in patient three, there was no sign of cancer in the tumor bed nor of metastases.[3]

Abnormally low concentrations of CoQ_{10} were found to be a strong predictor of metastasis in patients with melanoma. Patients with melanoma and matched controls were followed over seven and one-half years. The average CoQ_{10} levels of patients at baseline was 0.50 mcg/ml compared to 1.27 mcg/ml in controls. Researchers found that 33 percent of melanoma patients developed metastises during the follow-up period. The patients who developed metastises during follow-up had baseline CoQ_{10} levels of 0.34 mcg/ml compared with a level of 0.57 mcg/ml in patients who did not develop metasises. Patients with low baseline CoQ_{10} levels had an approximate eight-fold risk of metastatic disease compared with patients with high levels. It was concluded the baseline CoQ_{10} levels are a powerful and independent prognostic factor that can be used to estimate the risk for melanoma progression.[1] The foregoing study suggests the probability that CoQ_{10} supplementation may greatly reduce the risk of metastises in melanoma patients.

In cancer, abnormal cell growth occurs because cells have lost there ability to kill themselves, a process called apoptosis. A recent study suggests that supplementing with CoQ_{10} can restore the ability of the cancer cell to kill itself. Gene analysis has found that the bci-2 genes regulate cell division and programmed cell death. Cells normally divide and unneeded or sick cells are eliminated, but in cancer there is a decrease in cell death and the cells keep dividing. Both CoQ_{10} and bci-2 are present in normal and malignant cells, but in cancer patients there is an over-expression of bci-2 and a deficiency of CoQ_{10}. Under these conditions, the cells can't self destruct, resulting in cell proliferation.The researchers concluded that CoQ_{10} supplementation helps restore the ability of cancer cells to kill themselves.[4]

Another study found that CoQ_{10} supplementation reduces the side effects of chemotherapy.[5]

While no large long-term studies have yet been carried out on use of CoQ_{10} to treat cancer, it would seem prudent and beneficial for cancer patients to take CoQ_{10} supplements based on information available to date.

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A, make it difficult for the body to produce hormones needed for dealing with stress.

Adoption of a nutrient-dense, low-carbohydrate diet with a balance of essential fatty acids can profoundly shift the physiological state to one that is anti-atherogenic, with normalized insulin and lipid levels. Such improvements in diet will not significantly reverse established diabetes or coronary artery disease, but will reduce their rate of progression. It is not unusual for those who adopt a healthy low-carbohydrate diet to experience a reduction of the TG/HDL ratio by 50 to 75 percent, indicating a dramatic decrease in insulin resistance, inflammation, and levels of small LDL particles, and further indicating reduced risk of diabetes, coronary artery disease and adverse cardiac events.[28]

The beneficial effects of a nutrient-dense, low-carbohydrate diet with balanced essential fatty acids is seen in the low rates of diabetes and heart disease in those who follow this type of diet, such as those in some fishing villages in Japan and Inuit natives.[29,30] Autopsies have found significantly lower levels of atherosclerosis in such populations compared to neighboring populations on diets containing modern processed foods.

Cholesterol performs many important functions in the body and cholesterol levels increase with age in response to increased need. Research indicates that those with cholesterol above 240 have better brain function and live longer than those with cholesterol below the prescribed "healthy" level of 200. The increased incidence of cancer observed in statin users may be due in part to reduced cholesterol levels, which result in reduced synthesis of vitamin D from sunlight.[31,32] Saturated fat, which has been erroneously demonized as a cause of high cholesterol levels, does not stimulate insulin production and thus cannot cause increased cholesterol levels.[22]

The typical pro-atherogenic American diet has been made far worse by ill-advised government policies, which encourage increased consumption of carbohydrates and omega-6 polyunsaturated oils and discourage consumption of healthy saturated fat and protein. As a result, the majority of older people have some degree of atherosclerosis and many have coronary artery disease.

CoQ_{10} DEFICIENCY AND DIASTOLIC DYSFUNCTION

The filling phase of the heart requires more energy than the contraction phase, and is therefore more sensitive to CoQ_{10} deficiency. Energy starvation of the heart due to CoQ_{10} deficiency causes stiffening of the heart walls in the left ventricle and results in impaired filling of the heart, or diastolic

CoENZYME Q_{10} AND FOOD

Research indicates that the body requires replacement of about 500 mg per day of CoQ_{10}.[1] The average CoQ_{10} content of the western diet is about 5 mg per day, so for most people, food contributes only about 1 percent of daily CoQ_{10} requirements—the balance comes from endogenous synthesis. The highest level of CoQ_{10} is found in heart meat, and significant amounts are found in cold water fish, beef, pork, chicken and nuts. About 10 percent of daily CoQ_{10} requirements can be obtained by eating 12 ounces of beef or pork heart, two pounds of sardines or mackerel, three pounds of beef or pork, or four pounds of peanuts. Milk, eggs, and most grains and vegetables contain small amounts of CoQ_{10}.[2]

Synthesis of CoQ_{10} indispensably requires vitamins B_2, B_6, B_{12}, C, folic acid, niacin and pantothenic acid along with several trace elements, including selenium, which protects CoQ_{10} from oxidation.[3,4] Deficiencies in any of these nutrients can result in reduced synthesis of CoQ_{10} and cause many other adverse effects as well. The vitamin and mineral content of foods is therefore of greater importance for maintaining CoQ_{10} levels than their CoQ_{10} content. Most of the foods that contain significant amounts of CoQ_{10} are also rich in many of the nutrients required for CoQ_{10} synthesis.

Synthesis of CoQ_{10} declines with age.[5] A study of plasma levels of CoQ_{10} and vitamin B_6 in the elderly found that indicators of vitamin B_6 activity declined with age, and that CoQ_{10} levels were directly related to levels of B_6 activity,[6] indicating that reduced CoQ_{10} levels result from low levels of vitamin B_6. Vitamin B_{12} is also required for CoQ_{10} synthesis, and absorption of B_{12} declines with age. Advanced age therefore increases the importance of adequate intake of the nutrients required for synthesis of CoQ_{10}, including nutrient-dense foods like liver and raw animal foods as sources of vitamin B_6.

Many inflammatory conditions that result in oxidative stress and reduced CoQ_{10} levels, such as insulin intolerance, diabetes and atherosclerosis, can be prevented or improved by proper diet. A balanced, healthy diet is necessary to provide the nutrients needed for optimum CoQ_{10} synthesis and for maintenance of a physiological state that minimizes the oxidative stress that that leads to decreased CoQ_{10} levels.

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dysfunction. Fatigue or lack of energy is a common symptom of diastolic dysfunction. The stiffened heart walls increase energy requirements of the heart thereby increasing CoQ_{10} utilization and further depleting CoQ_{10} reserves. A vicious cycle ensues. As diastolic dysfunction worsens, blood pressure and heart rate increase, the heart walls thicken, and pulmonary hypertension often develops. When diastolic dysfunction is sufficient to produce pulmonary congestion (that is, a damming up of blood into the lungs causing shortness of breath), congestive heart failure is said to be present. Estimates indicate that 15 percent of those under age 50 and 50 percent of those over age 70 have diastolic dysfunction, and more than half of those presenting with acute pulmonary congestion have diastolic heart failure.[21]

The finding that diastolic dysfunction is caused by CoQ_{10} deficiency is not reflected in the majority of medical references. Mainstream medicine insists that diastolic dysfunction is due to many causes, including chronic hypertension, hypertrophic cardiomyopathy, aortic stenosis, coronary artery disease, diabetes and aging. All of the conditions considered "causes" of diastolic dysfunction increase utilization of CoQ_{10} and cause CoQ_{10} deficiency, and all can be improved with CoQ_{10} supplementation.

Systolic heart dysfunction caused by coronary artery disease and diastolic dysfunction due to CoQ_{10} deficiency are both common in the older population. In a recent study of men and women over age 65, about 5 percent were diagnosed with heart failure. Of those with heart failure, 63 percent had normal systolic function or diastolic heart failure, while only 15 percent had borderline systolic function and 22 percent had impaired systolic function. Men outnumbered women by three to one in those with impaired systolic function, while women slightly outnumbered men in those with normal systolic function.[20]

TREATMENT OF HEART DISEASE WITH COQ_{10}

The normal range of CoQ_{10} in the blood is 0.6 to 2.0 mcg/ml. CoQ_{10} levels in the low normal range are an indication of conditions that increase utilization or decrease synthesis of CoQ_{10}. Since CoQ_{10} deficiency is widespread, average levels of CoQ_{10} are actually considered to be less than optimum. In many of the studies of heart disease treatment with CoQ_{10}, patients were given a fixed amount of CoQ_{10} on the order of 100 mg/day. More recently it has become the practice of some physicians to adjust CoQ_{10} dosage to provide a minimum blood level of 2.0 mcg/ml, which usually requires dosages of from 200 to 500 mcg/ml per day. CoQ_{10} supplements are divided into doses of no more than 150 mg per day, usually taken with meals for most efficient absorption.[33]

The symptoms of fatigue and activity impairment, myalgia, and cardiac arrhythmia frequently precede by years the development of congestive heart failure and are the result of diastolic dysfunction caused by CoQ_{10} deficiency. Supplemental CoQ_{10} is unique in its ability to improve diastolic dysfunction and bring about a normalization of blood pressure, heart rate and ventricular hypertrophy that are symptoms of diastolic dysfunction. Accordingly, conditions that cause diastolic dysfunction are addressed first below.[34]

HYPERTHYROIDISM

An overactive thyroid causes a high rate of metabolism or energy use, which results in increased utilization of CoQ_{10} and can result in CoQ_{10} levels that are among the lowest detected in human diseases. Hyperthyroidism will lead to heart failure if not corrected. CoQ_{10} supplementation can normalize cardiac function, but correction of thyroid production usually will normalize metabolism, CoQ_{10} levels and diastolic function.[8]

STATIN DRUG USE

The depletion of the essential nutrient CoQ_{10} by the popular cholesterol-lowering drugs, HMG CoA reductase inhibitors (statins) is dose related. A clinical trial in which subjects took 80 mg per day of atorvastatin (Lipitor) resulted in a reduction of CoQ_{10} by 50 percent within 30 days. It was concluded that statin-induced CoQ_{10} deficiency was the probable cause of the most commonly

COQ_{10}, VITAMIN E, AND CORONARY ARTERY DISEASE

There are a number of forms of vitamin E that occur in foods, but alpha-tocopherol is the only form that is retained by the body in significant amounts and is therefore considered the form of vitamin E most important to health.[43] CoQ_{10} supplementation regenerates the oxidized form of alpha-tocopherol to the active reduced form. It has been hypothesized that CoQ_{10} is essential for the beneficial function of alpha-tocopherol.[44]

For a long time vitamin E was assumed to act by decreasing the oxidation of small dense LDL particles, which play a key role in atherosclerosis initiation. However, it has been found that at the cellular level, vitamin E acts by inhibiting many reactions involved in progression of atherosclerosis, including inhibition of smooth muscle cell proliferation, platelet aggregation, monocyte adhesion, oxLDL uptake, cykotine production and superoxide production. Oxidation impairs the beneficial functions of alpha-tocopherol, so regeneration of alpha-tocopherol by CoQ_{10} is important for preventing coronary artery disease.[45]

reported side effects of statins—muscle pain, exercise intolerance, and fatigue.[35]

Another clinical trial found that CoQ_{10} deficiency caused by atorvastatin therapy worsened left ventricular diastolic function in most patients. CoQ_{10} supplementation in patients with worsening diastolic function resulted in improved diastolic function.[36]

In a recent clinical study, patients who had been on statins for an average of 28 months were evaluated for adverse statin effects, including muscle pain, fatigue, shortness of breath, memory loss and peripheral neuropathy. All patients discontinued statins due to side effects and began supplemental CoQ_{10} at an average of 240 mg per day. After a period of 22 months, patients experienced a decrease in fatigue from 84 to 16 percent, muscle pain from 64 to 6 percent, shortness of breath from 58 to 12 percent, memory loss from 8 to 4 percent and peripheral neuropathy from 10 to 2 percent. Heart function improved or remained stable in the majority of patients and there were no adverse consequences from statin discontinuation. It was concluded that statin-related side effects are far more common than previously recognized and are reversible with a combination of statin discontinuation and supplemental CoQ_{10}.[37]

Cholesterol performs many valuable functions in the body that can be impaired by reduction of cholesterol levels with statins. For example, cells in the brain produce cholesterol because the cholesterol molecule is too large to pass the brain-blood barrier.[38] Statins can pass the blood-brain barrier and reduce brain levels of cholesterol. A recent clinical study found that those taking statins had minor impaired mental function compared to controls not on statins.[39]

HYPERTENSION

Secondary hypertension, or hypertension from known causes, such as renal artery stenosis and hardened arteries, is the cause of less than 10 percent of hypertension, while hypertension from unknown causes, or "essential hypertension," comprises the remaining 90 percent. Secondary hypertension increases the energy requirements of the heart, thereby increasing CoQ_{10} utilization and resulting diastolic dysfunction. Standard dogma in cardiology has long held that diastolic dysfunction is caused by both secondary and essential hypertension, but evidence from treatment of hypertension with CoQ_{10} suggests that diastolic dysfunction is the cause, not the result, of hypertension from unknown causes.

The vast majority of patients with hypertension have diastolic dysfunction regardless of whether the blood pressure is treated or untreated, or controlled or uncontrolled. Supplemental CoQ_{10} is unique in its ability to improve diastolic dysfunction, and as diastolic function improves, blood pressure in patients taking anti-hypertensive drugs drifts down so that more than one fourth of patients attain normal blood pressure and require no more anti-hypertensive drugs. The remaining patients require substantially less anti-hypertensive drug therapy.

Patients with diastolic dysfunction have impairment of the filling

CoQ_{10}, OMEGA-3 AND HEART DISEASE

More than 20,000 clinical studies have explored the health benefits of omega-3 fatty acids, a large portion of which involve the treatment of heart disease. A study in Italy of over 11,000 heart attack patients found that one gram per day of omega-3 fatty acids significantly reduced the mortality rate in the coming years, a record far better than that experienced by statin drug users, and without the adverse side effects. While the beneficial functions of omega-3 fatty acids are multiple, the researchers concluded that the reduction of mortality resulting in the aforementioned study was the result of a reduction in cardiac arrhythmias. As a result of the study, use of omega-3 supplementation following myocardial infarction has become standard protocol in Europe.[46]

EPA and DHA, the long-chain omega-3 fatty acids which are most important to heart health, are not present in plant foods but are abundant in cold water fish. EPA can be synthesized by the body from the omega-3 fatty acids found in plant food, but it is questionable whether DHA can be synthesized in adequate amounts, if at all. Synthesis of EPA is impaired by excessive omega-6 and carbohydrate intake and by *trans* fats. It has been hypothesized that CoQ_{10} may protect the sensitive DHA double bonds from destruction by oxidation.[47]

Animal studies illustrate that vitamin B_6 and folate metabolism are linked with those of long-chain fatty acids. Furthermore, a human study indicated synergistic effects of folic acid and vitamin B_6 together with omega-3 fatty acids on the atherogenic index.[48] Omega-3 fatty acids therefore enhance CoQ_{10} synthesis through enhancement of B vitamin metabolism.

The beneficial functions of alpha-tocopherol in inhibiting development of atherosclerosis are also brought about by supplementation with EPA and DHA. In addition, EPA and DHA supplements have been found to improve endothelial function and insulin resistance, reduce thrombosis, reduce triglycerides and increase HDL-cholesterol.[49,50]

CoQ_{10} has been found to improve endothelial function and peripheral resistance, both of which indicate poor circulation when compromised, leading to coronary artery disease. CoQ_{10} supplementation also reduces triglyceride levels, glucose levels and insulin resistance and increases HDL levels.[51,52]

phase of the cardiac cycle, which limits the ability to increase cardiac output, and cardiac output can only be increased by increasing heart rate and blood pressure. It has been postulated that the normalization of blood pressure which occurs as a result of CoQ_{10} supplementation is the result of the normalization of diastolic function—the ability of the heart to expand and fill more—and thus increase cardiac output.[40,41]

There have been numerous studies using CoQ_{10} for treatment of hypertension in patients not on anti-hypertensive drugs. In eight studies, the mean decrease in systolic blood pressure was 16 mmHg and in diastolic pressure, 10 mmHg.[42]

Other beneficial effects of CoQ_{10} include reduction of endothelial dysfunction, peripheral resistance and blood viscosity, thereby reducing blood pressure and improving circulation and delivery of oxygen to tissues. CoQ_{10} also normalizes blood pressure by reducing oxidative damage to cells through the anti-oxidant action of the reduced form of CoQ_{10}, ubiquinol.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM) is a genetic abnormality manifested by severe thickening of the left ventricle, a condition that increases energy requirements of the heart and increased utilization of CoQ_{10}. The resulting CoQ_{10} deficiency can cause significant diastolic dysfunction with disabling cardiac symptoms and increased risk of sudden death at any age. In a clinical trial on HCM treatment with CoQ_{10} supplementation, ventricular wall thicknesses were reduced by 25 percent to near normal levels and symptoms of diastolic dysfunction, including fatigue and shortness of breath, were greatly reduced.[10]

HEART DYSFUNCTION IN THE ELDERLY

Current medical texts often list aging as one of the many causes of diastolic dysfunction. Diastolic dysfunction is common in the elderly due to increased oxidative stress. Diastolic dysfunction shows clear improvement with use of CoQ_{10}, even in those of advanced age. Elderly patients, average age 84 years, experienced significant improvement in diastolic dysfunction, exercise tolerance and quality of life when treated with an average CoQ_{10} dose of 220 mg per day. These findings refute the common assertion that a stiff and non-compliant heart is a normal and irreversible aspect of the aging process.[53]

CONGESTIVE HEART FAILURE

The main clinical problems in patients with congestive heart failure are frequent hospitalizations due to the high incidence of life-threatening arrhythmias, pulmonary edema and other serious complications.

In a trial that studied the influence of long-term CoQ_{10} treatment on patients with chronic heart failure receiving conventional treatment, patients were randomized to receive a placebo or 2 mg per kilogram of body weight per day of CoQ_{10}. Compared to those in the placebo group, patients receiving CoQ_{10} in addition to conventional therapy had 38 percent fewer hospitalizations, 61 percent fewer episodes of pulmonary edema, and 51 percent fewer episodes of cardiac asthma. The results showed that the addition of CoQ_{10} to conventional therapy significantly reduced hospitalizations for worsening heart failure and the incidence of serious complications.[54]

AORTIC STENOSIS

Aortic stenosis, the incomplete opening of the aortic valve, increases the work load on the heart, thereby causing CoQ_{10} deficiency and diastolic dysfunction. A major cause of aortic stenosis is a bicuspid valve, a genetic abnormality in which the aortic valve has two cusps rather than the normal three cusps. Calcification and narrowing of a bicuspid aortic valve often begins in the fourth or fifth decade of life. Aortic stenosis can also occur in a normal valve, usually in the seventh and eighth decade of life, due to normal wear and tear. When symptoms of heart failure occur, valve replacement is the treatment of choice. Over half of valve replacements are in those with a bicuspid valve.

CoQ_{10} supplementation greatly improves heart function in aortic stenosis. While there have been no published studies on treatment of aortic stenosis with CoQ_{10}, cardiologists in private practice have observed near normalization of heart function in patients with mild to moderate aortic stenosis using CoQ_{10} treatment.[57] The hypothesis that CoQ_{10} supplements improve diastolic function by increasing ATP synthesis is illustrated by the fact that aortic stenosis patients have imparied ATP synthesis which normalizes after valve replacement.[9]

In practice, elderly aortic stenosis patients are often denied surgical valve replacement. An analysis of long-term survival of patients over 70 years of age found that only patients with high baseline risk had a significantly better three-year survival than patients denied surgical treatment. In low-risk patients, those denied valve replacement had a better survival rate than those who had valve replacement.[58]

Valve replacement in those with high baseline risk usually results in improvement of diastolic function, but often significant diastolic dysfunction remains, as indicated by pulmonary hypertension. Thus, it is reasonable to conclude that those patients who do benefit from valve replacement would also benefit from CoQ_{10} supplementation following valve replacement.

CoQ_{10} FOR HEART ATTACK PATIENTS

In a one-year, double-blind controlled trial of patients who had suffered a recent heart attack, the effects of 120 mg per day of CoQ_{10} were compared with effects of a placebo. The two groups were similar with respect to the extent and history of their heart disease, and both groups were receiving "optimal lipid therapy" with about half the patients in each group taking 10 mg per day of Lovastatin. Compared to those in the placebo group, patients receiving CoQ_{10} in addition to conventional therapy had 44 percent fewer episodes of total cardiac events, 44 percent fewer non-fatal infarctions, significantly lower cardiac deaths, 83 percent fewer patients reporting fatigue, and a significant decrease in markers of atherosclerosis.[55]

CoQ_{10} FOR DIABETIC PATIENTS

Diastolic dysfunction often in the absence of coronary artery disease is more prevalent in diabetics than in the general population. It has been estimated that approximately 75 percent of those with diabetes will eventually die from some kind of heart problem, compared to 25 percent of the general population. CoQ_{10} supplements of 200 mg per day have been found to improve blood pressure, glycemic control and endothelial (circulatory) function in patients with type-2 diabetes. Control of endothelial function is of great importance because of the prevalence of circulatory problems in diabetics.[51,56]

CONCLUSION

CoQ_{10}, a substance found in all cells of the body, is essential for using the energy you breath to make the cellular energy source ATP. CoQ_{10} deficiency results in impaired heart function, particularly dysfunction of the filling phase of the heart cycle. Diastolic dysfunction is present in all heart failure, whether the heart failure is due primarily to CoQ_{10} deficiency, coronary artery disease, or some other cause.

Synthesis of CoQ_{10} declines with age due to decreased absorption of the nutrients needed for CoQ_{10} synthesis and the increased utilization of CoQ_{10} from a natural increase in oxidative stress. CoQ_{10} synthesis can be further depressed by a number of congenital conditions such as type-1 diabetes and some autoimmune diseases, and by self-inflicted physiological conditions such as insulin intolerance, type-2 diabetes and coronary artery disease, all of which are caused by high insulin levels that result from a diet with excess carbohydrates and omega-6 fatty acids and inadequate omega-3. Thus, the misguided, "politically correct" dietary recommendations are a major contributor to coronary artery disease so prevalent in the western world. Depletion of CoQ_{10} levels can be exacerbated by many drugs that are widely prescribed to the elderly, particularly the cholesterol-lowering statins, which directly inhibit CoQ_{10} synthesis.

CoQ_{10} is naturally present in the body so CoQ_{10} supplementation causes virtually no side effects. CoQ_{10} supplementation can prevent development of diastolic dysfunction and the accompanying symptoms of hypertension and fatigue and, when used in conjunction with proper diet, can reduce the risk of coronary artery disease and diabetes. CoQ_{10} supplementation is also thought to reduce the risk of many other diseases and conditions, including cancer, immune diseases, migraine headache, male infertility and the increased oxidative stress that occurs with aging.

Of course, the first line of defence should be a nutrient-dense traditional diet, including liver and raw animal foods, which supply the vitamins and minerals needed for synthesis of CoQ_{10}. These foods are just as important for protecting seniors as they are for building strong bodies in the young.

LABORATORY TESTS FOR PLASMA CoQ_{10}

Measurement of blood levels of CoQ_{10} can determine whether or not supplementation is needed, as well as the effectiveness of supplementation in raising CoQ_{10} levels. Ubiquinol, the reduced form of CoQ_{10}, is a potent anti-oxidant that protects cells from damage by free radicals, and determination of the ratio of ubiquinol to total CoQ_{10} provides a measure of the risk of coronary artery disease. The ratio of CoQ_{10} to cholesterol indicates the level of protection of cholesterol against free radical damage. Vitamin E is also important because of the synergism between vitamin E and CoQ_{10},

At present there are only three labs in the US that provide testing for CoQ_{10} and three others that researchers use only for research. The most advanced lab for CoQ_{10} testing in the US is the Langsjoen Q_{10} Laboratory, Inc., located in the Langsjoen Cardiology Clinic in Tyler, Texas. This extremely accurate, high-pressure liquid chromatography laboratory can measure total and reduced CoQ_{10} levels in both blood and heart muscle. The unit also measures the ratio of CoQ_{10} to cholesterol and vitamin E levels. The only other such laboratories are located in Italy and Japan.

Individuals can have CoQ_{10} levels tested at the Langsjoen laboratory. Lab personnel can assist patients in arranging for samples to be drawn locally, preferably on a Monday. The samples must be shipped overnight packed in dry ice to the Langsjoen lab. Samples are tested and the results mailed to the patient. There is a significant degree of variation between individuals in absorption of CoQ_{10} supplements, particularly among those with significant heart impairment, so determination of supplemented blood levels is important for optimal treatment.

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The Oiling of America

How the Phony Cholesterol Theory Caused Americans to Abandon Healthy Whole Foods

By Mary G. Enig, PhD, and Sally Fallon

In 1954, a young researcher from Russia named David Kritchevsky published a paper describing the effects of feeding cholesterol to rabbits.[1] Cholesterol added to vegetarian rabbit chow caused the formation of atheromas—plaques that block arteries and contribute to heart disease. Cholesterol is a heavy weight molecule—an alcohol or a sterol—found only in animal foods such as meat, fish, cheese, eggs and butter.

In the same year, according to the American Oil Chemists Society, Kritchevsky published a paper describing the beneficial effects of polyunsaturated fatty acids for lowering cholesterol levels.[2] Polyunsaturated fatty acids are the kind of fats found in large amounts in highly liquid vegetable oils made from corn, soybeans, safflower seeds and sunflower seeds. (Monounsaturated fatty acids are found in large amounts in olive oil, palm oil and lard; saturated fatty acids are found in large amounts in fats and oils that are solid at room temperature, such as butter, tallows and coconut oil.)

Scientists of the period were grappling with a new threat to public health—a steep rise in heart disease. While turn-of-the-century mortality statistics are unreliable, they consistently indicate that heart disease caused no more than ten percent of all deaths, considerably less than infectious diseases such as pneumonia and tuberculosis.

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RISE OF CORONARY HEART DISEASE IN THE 20TH CENTURY

By 1950, coronary heart disease, or CHD, was the leading source of mortality in the United States, causing more than 30% of all deaths. The greatest increase came under the rubric of myocardial infarction (MI)—a massive blood clot leading to obstruction of a coronary artery and consequent death to the heart muscle. MI was almost nonexistent in 1910 and caused no more than three thousand deaths per year in 1930. By 1960, there were at least 500,000 MI deaths per year in the US. What life-style changes had caused this increase?

One change was a decrease in infectious disease, following the decline of the horse as a means of transport, the installation of more sanitary water supplies and the advent of better housing, all of which allowed more people to reach adulthood and the heart attack age. The other was a dietary change. Since the early part of the century, when the Department of Agriculture had begun to keep track of food "disappearance" data—the amount of various foods going into the food supply—a number of researchers had noticed a change in the kind of fats Americans were eating. Butter consumption was declining while the use of vegetable oils, especially oils that had been hardened to resemble butter by a process called hydrogenation, was increasing—dramatically increasing. By 1950 butter consumption had dropped from eighteen pounds per person per year to just over ten. Margarine filled in the gap, rising from about two pounds per person at the turn of the century to about eight. Consumption of vegetable shortening—used in crackers and baked goods—remained relatively steady at about twelve pounds per person per year but vegetable oil consumption had more than tripled—from just under three pounds per person per year to more than ten.[3]

The statistics pointed to one obvious conclusion—Americans should eat the traditional foods that nourished their ancestors, including meat, eggs, butter and cheese, and avoid the newfangled vegetable-oil-based foods that were flooding the grocers' shelves; but the Kritchevsky articles attracted immediate attention because they lent support to another theory—one that militated against the consumption of meat and dairy products. This was the lipid hypothesis, namely that saturated fat and cholesterol from animal sources raise cholesterol levels in the blood, leading to deposition of cholesterol and fatty material as pathogenic plaques in the arteries. Kritchevsky's rabbit trials were actually a repeat of studies carried out four decades earlier in St. Petersburg, in which rabbits fed saturated fats and cholesterol developed fatty deposits in their skin and other tissues—and in their arteries. By showing that feeding polyunsaturated oils from vegetable sources lowered serum cholesterol in humans, at least temporarily, Kritchevsky appeared to show that animal findings were relevant to the CHD problem, that the lipid hypothesis was a valid explanation for the new epidemic and that by reducing animal products in the diet Americans could avoid heart disease.

THE "EVIDENCE"

In the years that followed, a number of population studies demonstrated that the animal model—especially one derived from vegetarian animals—was not a valid approach for the problem of heart disease in human omnivores. A much publicized 1955 report on artery plaques in soldiers killed during the Korean War showed high levels of atherosclerosis, but another report—one that did not make it to the front pages—found that Japanese natives had almost as much pathogenic plaque—65 versus 75 percent—even though the Japanese diet at the time was lower in animal products and fat.[4] A 1957 study of the largely vegetarian Bantu found that they had as much atheroma—occlusions or plaque buildup in the arteries—as other races from South Africa who ate more meat.[5] A 1958 report noted that Jamaican Blacks showed a degree of atherosclerosis comparable to that found in the United States, although they suffered from lower rates of heart disease.[6] A 1960 report noted that the severity of atherosclerotic lesions in Japan approached that of the United States.[7] The 1968 International Atherosclerosis Project, in which over 22,000 corpses in 14 nations were cut open and examined for plaques in the arteries, showed the same degree of atheroma in all parts of the world—in populations that consumed large amounts of fatty animal products and those that were largely vegetarian, and in populations

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that suffered from a great deal of heart disease and in populations that had very little or none at all.[8] All of these studies pointed to the fact that the thickening of the arterial walls is a natural, unavoidable process. The lipid hypothesis did not hold up to these population studies, nor did it explain the tendency to fatal clots that caused myocardial infarction.

In 1956, an American Heart Association (AHA) fund-raiser aired on all three major networks. The MC interviewed, among others, Irving Page and Jeremiah Stamler of the AHA, and researcher Ancel Keys. Panelists presented the lipid hypothesis as the cause of the heart disease epidemic and launched the Prudent Diet, one in which corn oil, margarine, chicken and cold cereal replaced butter, lard, beef and eggs. But the television campaign was not an unqualified success because one of the panelists, Dr. Dudley White, disputed his colleagues at the AHA. Dr. White noted that heart disease in the form of myocardial infarction was nonexistent in 1900 when egg consumption was three times what it was in 1956 and when corn oil was unavailable. When pressed to support the Prudent Diet, Dr. White replied: "See here, I began my practice as a cardiologist in 1921 and I never saw an MI patent until 1928. Back in the MI free days before 1920, the fats were butter and lard and I think that we would all benefit from the kind of diet that we had at a time when no one had ever heard the word corn oil."

But the lipid hypothesis had already gained enough momentum to keep it rolling, in spite of Dr. White's nationally televised plea for common sense in matters of diet and in spite of the contradictory studies that were showing up in the scientific literature. In 1957, Dr. Norman Jolliffe, Director of the Nutrition Bureau of the New York Health Department initiated the Anti-Coronary Club, in which a group of businessmen, ranging in age from 40 to 59 years, were placed on the Prudent Diet. Club members used corn oil and margarine instead of butter, cold breakfast cereals instead of eggs and chicken and fish instead of beef. Anti-Coronary Club members were to be compared with a "matched" group of the same age who ate eggs for breakfast and had meat three times a day. Jolliffe, an overweight diabetic confined to a wheel chair, was confident that the Prudent Diet would save lives, including his own.

In the same year, the food industry initiated advertising campaigns that touted the health benefits of their products—low in fat or made with vegetable oils. A typical ad read: "Wheaties may help you live longer." Wesson recommended its cooking oil "for your heart's sake" a *Journal of the American Medical Association* ad described Wesson oil as a "cholesterol depressant." Mazola advertisements assured the public that "science finds corn oil important to your health." Medical journal ads recommended Fleishmann's unsalted margarine for patients with high blood pressure.

Dr. Frederick Stare, head of Harvard University's Nutrition Department, encouraged the consumption of corn oil—up to one cup a day—in his syndicated column. In a promotional piece specifically for Procter and Gamble's Puritan oil, he cited two experiments and one clinical trial as showing that high blood cholesterol is associated with CHD. However, both experiments had nothing to do with CHD, and the clinical trial did not find that reducing blood cholesterol had any effect on CHD events. Later, Dr. William Castelli, Director of the Framingham Study was one of several specialists to endorse Puritan. Dr. Antonio Gotto, Jr., former AHA president, sent a letter promoting Puritan Oil to practicing physicians—printed on Baylor College of Medicine, The De Bakey Heart Center letterhead.[9] The irony of Gotto's letter is that De Bakey, the famous heart surgeon, coauthored a 1964 study involving 1700 patients which also showed no definite correlation between serum cholesterol levels and the nature and extent of coronary artery disease.[10] In other words, those with low cholesterol levels were just as likely to have blocked arteries as those with high cholesterol levels. But while studies like De Bakey's moldered in the basements of university libraries, the vegetable oil campaign took on increased bravado and audacity.

The American Medical Association at first opposed the commercialization of the lipid hypothesis and warned that "the anti-fat, anti-cholesterol fad is not just foolish and futile. . . it also carries some risk." The American Heart Association, however, was committed. In 1961 the AHA published its first dietary guidelines aimed

at the public. The authors, Irving Page, Ancel Keys, Jeremiah Stamler and Frederick Stare, called for the substitution of polyunsaturates for saturated fat, even though Keys, Stare and Page had all previously noted in published papers that the increase in CHD was paralleled by increasing consumption of vegetable oils. In fact, in a 1956 paper, Keys had suggested that the increasing use of hydrogenated vegetable oils might be the underlying cause of the CHD epidemic.[11]

Stamler shows up again in 1966 as an author of *Your Heart Has Nine Lives*, a little self-help book advocating the substitution of vegetable oils for butter and other so-called "artery clogging" saturated fats. The book was sponsored by makers of Mazola Corn Oil and Mazola Margarine. Stamler did not believe that lack of evidence should deter Americans from changing their eating habits. The evidence, he stated, " . . was compelling enough to call for altering some habits even before the final proof is nailed down. . . the definitive proof that middle-aged men who reduce their blood cholesterol will actually have far fewer heart attacks waits upon diet studies now in progress." His version of the Prudent Diet called for substituting low-fat milk products such as skim milk and low-fat cheeses for cream, butter and whole cheeses, reducing egg consumption and cutting the fat off red meats. Heart disease, he lectured, was a disease of rich countries, striking rich people who ate rich food. . . including "hard" fats like butter.

It was in the same year, 1966, that the results of Dr. Jolliffe's Anti-Coronary Club experiment were published in the *Journal of the American Medical Association*.[12] Those on the Prudent Diet of corn oil, margarine, fish, chicken and cold cereal had an average serum cholesterol of 220, compared to 250 in the meat-and-potatoes control group. However, the study authors were obliged to note in the fine print that there were eight deaths from heart disease among Dr. Jolliffe's Prudent Diet group, and none among those who ate meat three times a day. Dr. Jolliffe was dead by this time. He succumbed in 1961 from a vascular thrombosis, although the obituaries listed the cause of death as complications from diabetes. The "compelling proof" that Stamler and others were sure would vindicate wholesale tampering with American eating habits had not yet been "nailed down."

The problem, said the insiders promoting the lipid hypothesis, was that the numbers involved in the Anti-Coronary Club experiment were too small. Dr. Irving Page urged a National Diet-Heart Study involving one million men, in which the results of the Prudent Diet could be compared on a large scale with the those on a diet high in meat and fat. With great media attention, the National Heart Lung and Blood Institute organized the stocking of food warehouses in six major cities, where men on the Prudent Diet could get tasty polyunsaturated donuts and other fabricated food items free of charge. But a pilot study involving 2,000 men resulted in exactly the same number of deaths in both the Prudent Diet and the control group. A brief report in *Circulation*, March 1968, stated that the study was a milestone "in mass environmental experimentation" that would have "an important effect on the food industry and the attitude of the public toward its eating habits." But the million-man Diet Heart Study was abandoned in utter silence "for reasons of cost." Its chairman, Dr. Irving Page, died of a heart attack.

HYDROGENATION AND *TRANS* FATS

Most animal fats—like butter, lard and tallow—have a large proportion of saturated fatty acids. Saturated fats are straight chains of carbon and hydrogen that pack together easily so that they are relatively solid at room temperature. Oils from seeds are composed mostly of polyunsaturated fatty acids. These molecules have kinks in them at the point of the unsaturated double bonds. They do not pack together easily and therefore tend to be liquid at room temperature. Judging from both food data and turn-of-the-century cookbooks, the American diet in 1900 was a rich one—with at least 35 to 40 percent of calories coming from fats, mostly dairy fats in the form of butter, cream, whole milk and eggs. Salad dressing recipes usually called for egg yolks or cream; only occasionally for olive oil. Lard or tallow served for frying; rich dishes like head cheese and scrapple contributed additional saturated fats during an era when cancer and heart disease were rare. Butter substitutes made up only a small portion of the American diet, and these margarines were blended from coconut oil, animal tallow

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and lard, all rich in natural saturates.

The technology by which liquid vegetable oils could be hardened to make margarine was first discovered by a French chemist named Sabatier. He found that a nickel catalyst would cause the hydrogenation—the addition of hydrogen to unsaturated bonds to make them saturated—of ethylene gas to ethane. Subsequently the British chemist Norman developed the first application of hydrogenation to food oils and took out a patent. In 1909, Procter & Gamble acquired the US rights to the British patent that made liquid vegetable oils solid at room temperature. The process was used on both cottonseed oil and lard to give "better physical properties"—to create shortenings that did not melt as easily on hot days.

The hydrogenation process transforms unsaturated oils into straight "packable" molecules, by rearranging the hydrogen atoms at the double bonds. In nature, most double bonds occur in the *cis* configuration, that is with both hydrogen atoms on the same side of the carbon chain at the point of the double bond. It is the *cis* isomers of fatty acids that have a bend or kink at the double bond, preventing them from packing together easily. Hydrogenation creates *trans* double bonds by moving one hydrogen atom across to the other side of the carbon chain at the point of the double bond. In effect, the two hydrogen atoms then balance each other and the fatty acid straightens, creating a packable "plastic" fat with a much higher melting temperature. Although *trans* fatty acids are technically unsaturated, they are configured in such a way that the benefits of unsaturation are lost. The presence of several unpaired electrons presented by contiguous hydrogen atoms in their *cis* form allows many vital chemical reactions to occur at the site of the double bond. When one hydrogen atom is moved to the other side of the fatty acid molecule during hydrogenation, the ability of living cells to make reactions at the site is compromised or altogether lost. *Trans* fatty acids are sufficiently similar to natural fats that the body readily incorporates them into the cell membrane; once there their altered chemical structure creates havoc with thousands of necessary chemical reactions—everything from energy provision to prostaglandin production.

After the second world war, "improvements" made it possible to plasticize highly unsaturated oils from corn and soybeans. New catalysts allowed processors to "selectively hydrogenate" the kinds of fatty acids with three double bonds found in soy and canola oils. Called "partial hydrogenation," the new method allowed processors to replace cottonseed oil with more unsaturated corn and soy bean oils in margarines and shortenings. This spurred a meteoric rise in soybean production, from virtually nothing in 1900 to 70 million tons in 1970, surpassing corn production. Today soy oil dominates the market and is used in almost eighty percent of all hydrogenated oils.

The particular mix of fatty acids in soy oil results in shortenings containing about 40 percent *trans* fats, an increase of about 5 percent over cottonseed oil, and 15 percent over corn oil. Canola oil, processed from a hybrid form of rape seed, is particularly rich in fatty acids containing three double bonds and the shortening can contain as much as 50 percent *trans* fats. *Trans* fats of a particularly problematical form are also formed during the deodorization of canola oil, although they are not indicated on labels for the liquid oil.[13]

Certain forms of *trans* fatty acids occur naturally in dairy fats. *Trans*-vaccenic acid makes up about 4 percent of the fatty acids in butter. It is an interim product which the ruminant animal then converts to conjugated linoleic acid, a highly beneficial anti-carcinogenic component of animal fat. Humans seem to utilize the small amounts of *trans*-vaccenic acid in butter fat without ill effects.

But most of the *trans* isomers in modern hydrogenated fats are new to the human physiology and by the early 1970s a number of researchers had expressed concern about their presence in the American diet, noting that their increasing use had paralleled the increase in both heart disease and cancer. The unstated solution was one that could be easily presented to the public: Eat natural, traditional fats; avoid newfangled foods made from vegetable oils; use butter, not margarine. But medical research and public consciousness took a different tack, one that accelerated the decline of traditional foods like meat, eggs and butter, and fueled continued dramatic increases in vegetable oil consumption.

SHENANIGANS AT THE AHA

Although the AHA had committed itself to the lipid hypothesis and the unproven theory that polyunsaturated oils afforded protection against heart disease, concerns about hydrogenated vegetable oils were sufficiently great to warrant the inclusion of the following statement in the organization's 1968 diet heart statement: "Partial hydrogenation of polyunsaturated fats results in the formation of *trans* forms which are less effective than *cis*, *cis* forms in lowering cholesterol concentrations. It should be noted that many currently available shortening and margarines are partially hydrogenated and may contain little polyunsaturated fat of the natural *cis*, *cis* form." The AHA printed 150,000 copies of the statement but never distributed them. The shortening industry objected strongly and a researcher named Fred Mattson of Procter and Gamble convinced Campbell Moses, medical director of the AHA, to destroy the statements.[14] The final recommendations for the public contained three major points—restrict calories, substitute polyunsaturates for saturates and reduce cholesterol in the diet.

Other organizations fell in behind the AHA in pushing vegetable oils instead of animal fats. By the early 1970s the National Heart Lung and Blood Institute, the AMA, the American Dietetic Association and the National Academy of Science had all endorsed the lipid hypothesis and the avoidance of animal fats for those Americans in the "at risk" category.

Since Kritchevsky's early studies, many other trials had shown that serum cholesterol can be lowered by increasing ingestion of polyunsaturates. The physiological explanation for this is that when excess polyunsaturates are built into the cell membranes, resulting in reduced structural integrity or "limpness," cholesterol is sequestered from the blood into the cell membranes to give them "stiffness." The problem was that there was no proof that lowering serum cholesterol levels could stave off CHD. That did not prevent the American Heart Association from calling for "modified and ordinary foods" useful for the purpose of facilitating dietary changes to newfangled oils and away from traditional fats. These foods, said the AHA literature, should be made available to the consumer, "reasonably priced and easily identified by appropriate labeling. Any existing legal and regulatory barriers to the marketing of such foods should be removed."

SHENANIGANS AT THE FDA

The man who made it possible to remove any "existing legal and regulatory barriers" was Peter Barton Hutt, a food lawyer for the prestigious Washington, DC law firm of Covington and Burling. Hutt once stated that "Food law is the most wonderful field of law that you can possibly enter." After representing the edible oil industry, he temporarily left his law firm to become the FDA's general council in 1971. The regulatory barrier to foods useful to the purpose of changing American consumption patterns was the Food, Drug and Cosmetic Act of 1938, which stated that ". . . there are certain traditional foods that everyone knows, such as bread, milk and cheese, and that when consumers buy these foods, they should get the foods that they are expecting. . . [and] if a food resembles a standardized food but does not comply with the standard, that food must be labeled as an 'imitation.'"[15]

The 1938 Food, Drug and Cosmetic Act became law partly in response to consumer concerns about the adulteration of ordinary foodstuffs. Chief among the products with a tradition of suffering competition from imitation products were fats and oils. In *Life on the Mississippi*, Mark Twain reports on a conversation overheard between a New Orleans cottonseed oil purveyor and a Cincinnati margarine drummer. New Orleans boasts of selling deodorized cottonseed oil as olive oil in bottles with European labels. "We turn out the whole thing—clean from the word go—in our factory in New Orleans. . . We are doing a ripping trade, too." The man from Cincinnati reports that his factories are turning out oleomargarine by the thousands of tons, an imitation that "you can't tell from butter." He gloats at the thought of market domination. "You are going to see the day, pretty soon, when you won't find an ounce of butter to bless yourself with, in any hotel in the Mississippi and Ohio Valleys, outside of the biggest cities. . . And we can sell it so dirt cheap that the whole country has got to take it. . . butter don't stand any show—there ain't any chance for competition. Butter's had its day—and

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These new regulations were adopted without the consent of Congress, continuing the trend instituted under Nixon in which the White House would use the FDA to promote certain social agendas through government food policies.

from this day out, butter goes to the wall. There's more money in oleomargarine than, why, you can't imagine the business we do."

In the tradition of Mark Twain's riverboat hucksters, Peter Barton Hutt guided the FDA through the legal and congressional hoops to the establishment of the FDA "Imitation" policy in 1973, which attempted to provide for "advances in food technology" and give "manufacturers relief from the dilemma of either complying with an outdated standard or having to label their new products as 'imitation' . . . [since]. . . such products are not necessarily inferior to the traditional foods for which they may be substituted."[15] Hutt considered the word "imitation" to be over simplified and inaccurate—"potentially misleading to consumers." The new regulations defined "inferiority" as any reduction in content of an essential nutrient that is present at a level of two percent or more of the US Recommended Daily Allowance (RDA). The new imitation policy meant that imitation sour cream, made with vegetable oil and fillers like guar gum and carrageenan, need not be labelled imitation as long as artificial vitamins were added to bring macro nutrient levels up to the same amounts as those in real sour cream. Coffee creamers, imitation egg mixes, processed cheeses and imitation whipped cream no longer required the imitation label, but could be sold as real and beneficial foods, low in cholesterol and rich in polyunsaturates.

These new regulations were adopted without the consent of Congress, continuing the trend instituted under Nixon in which the White House would use the FDA to promote certain social agendas through government food policies. They had the effect of increasing the lobbying clout of special interest groups, such as the edible oil industry, and short circuiting public participation in the regulatory process. They allowed food processing innovations regarded as "technological improvements" by manufacturers to enter the market place without the onus of economic fraud that might be engendered by greater consumer awareness and congressional supervision. They ushered in the era of ersatz foodstuffs, convenient counterfeit products—weary, stale, flat and immensely profitable.

SHENANIGANS IN CONGRESS

Congress did not voice any objection to this usurpation of its powers, but entered the contest on the side of the lipid hypothesis. The Senate Select Committee on Nutrition and Human Needs, chaired by George McGovern during the years 1973 to 1977, actively promoted the use of vegetable oils. "Dietary Goals for the United States," published by the committee, cited U.S. Department of Agriculture data on fat consumption, and stated categorically that "the overconsumption of fat, generally, and saturated fat in particular. . . have been related to six of the ten leading causes of death. . ." in the United States. The report urged the American populace to reduce overall fat intake and to substitute polyunsaturates for saturated fat from animal sources—margarine and corn oil for butter, lard and tallow. Opposing testimony included a moving letter—buried in the voluminous report—by Dr. Fred Kummerow of the University of Illinois, urging a return to traditional whole foods and warning against the use of soft drinks. In the early 1970s, Kummerow had shown that *trans* fatty acids caused increased rates of heart disease in pigs. A private endowment allowed him to continue his research—government funding agencies such as National Institutes of Health refused to give him further grants.

One unpublished study that was known to McGovern Committee members but not mentioned in its final report compared calves fed saturated fat from tallow and lard with those fed unsaturated fat from soybean oil. The calves fed tallow and lard did indeed show higher plasma cholesterol levels than the soybean oil-fed calves, and fat streaking was found in their aortas. Atherosclerosis was also enhanced. But the calves fed soybean oil showed a decline in calcium and magnesium levels in the blood, possibly due to inefficient absorption. They utilized vitamins and minerals inefficiently, showed poor growth, poor bone development and had abnormal hearts. More cholesterol per unit of dry matter was found in the aorta, liver, muscle, fat and coronary arteries, a finding which led the investigators to the conclusion the lower blood cholesterol levels in the soybean-oil fed calves may have been the result of cholesterol being transferred from the blood to other tissues. The calves in the soybean

oil group also collapsed when they were forced to move around and they were unaware of their surroundings for short periods. They also had rickets and diarrhea.

The McGovern Committee report continued dietary trends already in progress—the increased use of vegetables oils, especially in the form of partially hydrogenated margarines and shortenings. In 1976, the FDA established GRAS (Generally Recognized as Safe) status for hydrogenated soybean oil. A report prepared by the Life Sciences Research Office of the Federation of American Scientists for Experimental Biology (LSRO-FASEB) concluded that "There is no evidence in the available information on hydrogenated soybean oil that demonstrates or suggests reasonable ground to suspect a hazard to the public when it is used as a direct or indirect food ingredient at levels that are now current or that might reasonably be expected in the future."

ENIG SPEAKS OUT

When Mary Enig, a graduate student at the University of Maryland, read the McGovern committee report, she was puzzled. Enig was familiar with Kummerow's research and she knew that the consumption of animal fats in America was not on the increase—quite the contrary, use of animal fats had been declining steadily since the turn of the century. A report in the Journal of American Oil Chemists—which the McGovern Committee did not use—showed that animal fat consumption had declined from 104 grams per person per day in 1909 to 97 grams per day in 1972, while vegetable fat intake had increased from a mere 21 grams to almost 60.[16] Total per capita fat consumption had increased over the period, but this increase was mostly due to an increase in unsaturated fats from vegetable oils—with 50 percent of the increase coming from liquid vegetable oils and about 41 percent from margarines made from vegetable oils. She noted a number of studies that directly contradicted the McGovern Committee's conclusions that "there is . . . a strong correlation between dietary fat intake and the incidence of breast cancer and colon cancer," two of the most common cancers in America. Greece, for example, had less than one-fourth the rate of breast cancer compared to Israel but the same dietary fat intake. Spain had only one-third the breast cancer mortality of France and Italy but the total dietary fat intake was slightly greater. Puerto Rico, with a high animal fat intake, had a very low rate of breast and colon cancer. The Netherlands and Finland both used approximately 100 grams of animal fat per capita per day but breast and colon cancer rates were almost twice in the Netherlands what they were in Finland. The Netherlands consumed 53 grams of vegetable fat per person compared to 13 in Finland. A study from Cali, Columbia found a fourfold excess risk for colon cancer in the higher economic classes, which used less animal fat than the lower economic classes. A study on Seventh-Day Adventist physicians, who avoid meat, especially red meat, found they had a significantly higher rate of colon cancer than non-Seventh Day Adventist physicians. Enig analyzed the USDA data that the McGovern Committee had used and concluded that it showed a strong positive correlation with total fat and vegetable fat and an essentially strong negative correlation or no correlation with animal fat to total cancer deaths, breast and colon cancer mortality and breast and colon cancer incidence—in other words, use of vegetable oils seemed to predispose to cancer and animal fats seemed to protect against cancer. She noted that the analysts for the committee had manipulated the data in inappropriate ways in order to obtain mendacious results.

Enig submitted her findings to the journal of the Federation of American Societies for Experimental Biology (FASEB), in May, 1978, and her article was published in the FASEB's *Federation Proceedings*[17] in July of the same year—an unusually quick turnaround. The assistant editor, responsible for accepting the article, died of a heart attack shortly thereafter. Enig's paper noted that the correlations pointed a finger at the *trans* fatty acids and called for further investigation. Only two years earlier, the Life Sciences Research office, which is the arm of FASEB that does scientific investigations, had published the whitewash that had ushered partially hydrogenated soybean oil onto the GRAS list and removed any lingering constraints against the number one ingredient in factory-produced food.

Use of vegetable oils seemed to predispose to cancer and animal fats seemed to protect against cancer.

THE FOOD GIANTS FIGHT BACK

Enig's paper sent alarm bells through the industry. In early 1979, she received a visit from S. F. Reipma of the National Association of Margarine Manufacturers. Reipma was visibly annoyed. He explained that both his association and the Institute for Shortening and Edible Oils (ISEO) kept careful watch to prevent articles like Enig's from appearing in the literature. Enig's paper should never have been published, he said. He thought that ISEO was "watching out."

"We left the barn door open," he said, "and the horse got out."

Reipma also challenged Enig's use of the USDA data, claiming that it was in error. He knew it was in error, he said, "because we give it to them."

A few weeks later, Reipma paid a second visit, this time in the company of Thomas Applewhite, an advisor to the ISEO and representative of Kraft Foods, Ronald Simpson with Central Soya and an unnamed representative from Lever Brothers. They carried with them—in fact, waved them in the air in indignation—a two-inch stack of newspaper articles, including one that appeared in the *National Enquirer*, reporting on Enig's Federation Proceedings article. Applewhite's face flushed red with anger when Enig repeated Reipma's statement that "they had left the barn door open and a horse got out," and his admission that Department of Agriculture food data had been sabotaged by the margarine lobby.

The other thing Reipma told Enig during his unguarded visit was that he had called in on the FASEB offices in an attempt to coerce them into publishing letters to refute her paper, without allowing Enig to submit any counter refutation as was normally customary in scientific journals. He told Enig that he was "thrown out of the office"—an admission later confirmed by one of the FASEB editors. Nevertheless, a series of letters did follow the July 1978 article.[18] On behalf of the ISEO, Applewhite and Walter Meyer of Procter and Gamble criticized Enig's use of the data; Applewhite accused Enig of extrapolating from two data points, when in fact she had used seven. In the same issue, John Bailar, Editor-in-Chief of the *Journal of the National Cancer Institute*, pointed out that the correlations between vegetable oil consumption and cancer were not the same as evidence of causation and warned against changing current dietary components in the hopes of preventing cancer in the future—which is of course exactly what the McGovern Committee did.

In reply, Enig and her colleagues noted that although the NCI had provided them with faulty cancer data, this had no bearing on the statistics relating to *trans* consumption, and did not affect the gist of their argument—that the correlation between vegetable fat consumption, especially *trans* fat consumption, was sufficient to warrant a more thorough investigation. The problem was that very little investigation was being done.

University of Maryland researchers recognized the need for more research in two areas. One concerned the effects of *trans* fats on cellular processes once they are built into the cell membrane. Studies with rats, including one conducted by Fred Mattson in 1960, indicated that the *trans* fatty acids were built into the cell membrane in proportion to their presence in the diet, and that the turnover of *trans* in the cells was similar to that of other fatty acids. These studies, according to J. Edward Hunter of the ISEO, were proof that "*trans* fatty acids do not pose any hazard to man in a normal diet." Enig and her associates were not so sure. Kummerow's research indicated that the *trans* fats contributed to heart disease, and Kritchevsky—whose early experiments with vegetarian rabbits were now seen to be totally irrelevant to the human model—had found that *trans* fatty acids raise cholesterol in humans.[19] Enig's own research, published in her 1984 doctoral dissertation, indicated that *trans* fats interfered with enzyme systems that neutralized carcinogens and increased enzymes that potentiated carcinogens.[20]

TRANS FAT LEVELS

The other area needing further investigation concerned just how much *trans* fat there was in a "normal diet" of the typical American. What had hampered any thorough research into the correlation of *trans* fatty acid consumption and disease was the fact that these altered fats were not considered as a separate category in any of the data bases then available to researchers. A 1970 FDA internal memo stated that a market basket survey was needed to determine *trans* levels in commonly used foods. The memo remained buried in the FDA files. The massive Health and Human Services NHANES II (National Health and Nutrition Examination Survey) survey, conducted during the years 1976 to 1980, noted the increasing US consumption of margarine, french fried potatoes, cookies and snack chips—all made with vegetable shortenings—without listing the proportion of *trans*.

Enig first looked at the NHANES II data base in 1987 and when she did, she had a sinking feeling. Not only were *trans* fats conspicuously absent from the fatty acid analyses, data on other lipids made no sense at all. Even foods containing no *trans* fats were listed with faulty fatty acid profiles. For example, safflower oil was listed as containing 14 percent linoleic acid (a double bond fatty acid of the omega-6 family) when in fact it contained 80 percent; a sample of butter crackers was listed as containing 34 percent saturated fat when in fact it contained 78 percent. In general, the NHANES II data base tended to minimize the amount of saturated fats in common foods.

Over the years, Joseph Sampagna

and Mark Keeney, both highly qualified lipid biochemists at the University of Maryland, applied to the National Science Foundation, the National Institutes of Health, the US Department of Agriculture, the National Dairy Council and the National Livestock and Meat Board for funds to look into the *trans* content of common American foods. Only the National Livestock and Meat Board came through with a small grant for equipment; the others turned them down. The pink slip from National Institutes of Health criticized items that weren't even relevant to the proposal. The turndown by the National Dairy Council was not a surprise. Enig had earlier learned that Phil Lofgren, then head of research at the Dairy Council, had philosophical ties to the lipid hypothesis. Enig tried to alert Senator Mettzanbaum from Ohio, who was involved in the dietary recommendations debate, but got nowhere.

A USDA official confided to the Maryland research group that they "would never get money as long as they pursued the *trans* work." Nevertheless they did pursue it. Sampagna, Keeney and a few graduate students, funded jointly by the USDA and the university, spent thousands of hours in the laboratory analyzing the *trans* fat content of hundreds of commercially available foods. Enig worked as a graduate student, at times with a small stipend, at times without pay, to help direct the process of tedious analysis. The long arm of the food industry did its best to put a stop to the group's work by pressuring the USDA to pull its financial support of the graduates students doing the lipid analyses, which the University of Maryland received due to its status as a land grant college.

In December of 1982, *Food Processing* carried a brief preview of the University of Maryland research[21] and five months later the same journal printed a blistering letter from Edward Hunter on behalf of the Institute of Shortening and Edible Oils.[22] The University of Maryland studies on *trans* fat content in common foods had obviously struck a nerve. Hunter stated that the Bailar, Applewhite and Meyer letters that had appeared in Federation Proceedings five years earlier, "severely criticized and discredited" the conclusions reached by Enig and her colleagues. Hunter was concerned that Enig's group would exaggerate the amount of *trans* found in common foods. He cited ISEO data indicating that most margarines and shortenings contain no more than 35 percent and 25 percent *trans* respectively, and that most contain considerably less.

What Enig and her colleagues actually found was that many margarines indeed contained about 31 percent *trans* fat—later surveys by others revealed that Parkay margarine contained up to 45 percent *trans*—while many shortenings found ubiquitously in cookies, chips and baked goods contained more than 35 percent. She also discovered that many baked goods and processed foods contained considerably more fat from partially hydrogenated vegetable oils than was listed on the label. The finding of higher levels of fat in products made with partially hydrogenated oils was confirmed by Canadian government researchers many years later, in 1993.[23]

Final results of Enig's groundbreaking compilation were published in the October 1983 edition of the *Journal of the American Oil Chemists Society*.[24] Her analyses of more than 220 food items, coupled with food disappearance data, allowed University of Maryland researchers to confirm earlier estimates that the average American consumed at least 12 grams of *trans* fat per day, directly contradicting ISEO assertions that most Americans consumed no more than six to eight grams of *trans* fat per day. Those who consciously avoided animal fats typically consumed far more than 12 grams of *trans* fat per day.

CAT AND MOUSE GAMES

The ensuing debate between Enig and her colleagues at the University of Maryland, and Hunter and Applewhite of the ISEO, took the form of a cat and mouse game running through several scientific journals. *Food Processing* declined to publish Enig's reply to Hunter's attack. *Science Magazine* published another critical letter by Hunter in 1984,[25] in which he misquoted Enig, but refused to print her rebuttal. Hunter continued to object to assertions that average consumption of *trans* fat in partially hydrogenated margarines and shortenings could exceed six to eight grams per day, a concern that Enig found puzzling when coupled with the official ISEO position that *trans* fatty acids were innocuous and posed no threat to public health.

The ISEO did not want the American public to hear about the debate on hydrogenated vegetable oils—for Enig this translated into the sound of doors closing. A poster presentation she organized for a campus health fair caught the eye of the dietetics department chairman who suggested she submit an abstract to the Society for Nutrition Education, many of whose members are registered dietitians. Her abstract concluded that ". . . meal plans and recipes developed for nutritionists and dieticians to use when designing diets to meet the Dietary Guidelines, the dietary recommendation of the American Heart Association or the Prudent Diet have been examined for *trans* fatty acid content. Some diet plans are found to contain approximately 7 percent or more of calories as *trans* fatty acids." The Abstract Review Committee rejected the submission, calling it "of limited interest."

Early in 1985 the Federation of American Societies for Experimental Biology (FASEB) heard more testimony on the *trans* fat issue. Enig alone represented the alarmist point of view, while Hunter and Applewhite of the ISEO, and Ronald Simpson, then with the National

Association of Margarine Manufacturers, assured the panel that *trans* fats in the food supply posed no danger. Enig reported on University of Maryland research that delineated the differences in small amounts of naturally occurring *trans* fats in butter, which do not inhibit enzyme function at the cellular level, and man-made *trans* fats in margarines and vegetable shortenings which do. She also noted a 1981 feeding trial in which swine fed *trans* fatty acid developed higher parameters for heart disease than those fed saturated fats, especially when *trans* fatty acids were combined with added polyunsaturates.[26] Her testimony was omitted from the final report, although her name in the bibliography created the impression that her research supported the FASEB whitewash.[27]

In the following year, 1986, Hunter and Applewhite published an article exonerating *trans* fats as a cause of atherosclerosis in the prestigious *American Journal of Clinical Nutrition*,[28] whose sponsors, by the way, include companies like Procter and Gamble, General Foods, General Mills, Nabisco and Quaker Oats. The authors once again stressed that the average per capita consumption of *trans* fatty acids did not exceed six to eight grams. Many subsequent government and quasi government reports minimizing the dangers of *trans* fats used the 1986 Hunter and Applewhite article as a reference.

Enig testified again in 1988 before the Expert Panel on the National Nutrition Monitoring System (NNMS). In fact she was the only witness before a panel, which began its meeting by confirming that the cause of America's health problems was the overconsumption of "fat, saturated fatty acids, cholesterol and sodium." Her testimony pointed out that the 1985 FASEB report exonerating *trans* fatty acids as safe was based on flawed data.

Behind the scenes, in a private letter to Dr. Kenneth Fischer, Director of the Life Sciences Research Office (LSRO), Hunter and Applewhite charged that "the University of Maryland group continues to raise unwarranted and unsubstantiated concerns about the intake of and imagined physiological effects of *trans* fatty acids and . . . they continue to overestimate greatly the intake of *trans* acids by typical Americans." "No one other than Enig," they said, "has raised questions about the validity of the food fatty acid composition data used in NHANES II and. . . she has not presented sufficiently compelling arguments to justify a major reevaluating."

The letter contained numerous innuendos that Enig had mischaracterized the work of other researchers and had been less than scientific in her research. It was widely circulated among National Nutrition Monitoring System agencies. John Weihrauch, a USDA scientist, not an industry representative, slipped it surreptitiously to Dr. Enig. She and her colleagues replied by asking, "If the trade association truly believes 'that *trans* fatty acids do not pose any harm to humans and animals'. . . why are they so concerned about any levels of consumption and why do they so vehemently and so frequently attack researchers whose finding suggest that the consumption of *trans* fatty acids is greater than the values the industry reports?"

Maryland researchers argued that *trans* fats should be included in food nutrition labels; the Hunter and Applewhite letter asserted that "there is no documented justification for including *trans* acids . . . as part of nutrition labeling."

During her testimony Enig also brought up her concerns about other national food databases, citing their lack of information on *trans*. The Food Consumption Survey contained glaring errors—reporting, for example, consumption of butter in amounts nearly twice as great as what exists in the US food supply, and of margarine in quantities nearly half those known to exist in the food supply. "The fact that the data base is in error should compel the Congress to require correction of the data base and reevaluation of policy flowing from erroneous data," she argued, "especially since the congressional charter for NHANES was to compare dietary intake and health status and since this data base is widely used to do just that." Rather than "correction of the data base," [The] National Nutritional Monitoring System officials responded to Enig's criticism by dropping the whole section pertaining to butter and margarine from the 1980 tables.

Enig's testimony was not totally left out of the National Nutritional Monitoring System final report, as it had been from the FASEB report three years earlier. A summary of the proceedings and listing of panelists released in July of 1989 by Director Kenneth Fischer announced that a transcript of Enig's testimony could be obtained from Ace Federal Reporter in Washington DC.[29] Unfortunately, his report wrongly listed the date of her testimony as January 20, 1988, rather than January 21, making her comments more difficult to retrieve.

The Enig-ISEO debate was covered by the prestigious *Food Chemical News* and *Nutrition Week*[30]—both widely read by Congress and the food industry, but virtually unknown to the general public. National media coverage of dietary fat issues focused on the proceedings of the National Heart, Lung and Blood Institute as this enormous bureaucracy plowed relentlessly forward with the lipid hypothesis. In June of 1984, for example, the press diligently reported on the proceedings of the NHLBI's Lipid Research Clinics Conference, which was organized to wrap up almost 40 years of research on lipids, cholesterol and heart disease.

The problem with the 40 years of NHLBI-sponsored research on lipids, cholesterol and heart disease was that

it had not produced many answers—at least not many answers that the NHLBI was pleased with. The ongoing Framingham Study found that there was virtually no difference in coronary heart disease "events" for individuals with cholesterol levels between 205 mg/dL and 294 mg/dL—the vast majority of the US population. Even for those with extremely high cholesterol levels—up to almost 1200 mg/dL, the difference in CHD events compared to those in the normal range was trivial.[31] This did not prevent Dr. William Kannel, then Framingham Study Director, from making claims about the Framingham results. "Total plasma cholesterol" he said, "is a powerful predictor of death related to CHD." It wasn't until more than a decade later that the real findings at Framingham were published—without fanfare—in the *Archives of Internal Medicine*, an obscure journal. "In Framingham, Massachusetts," admitted Dr. William Castelli, Kannel's successor "the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower people's serum cholesterol. . . . 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."[32]

NHLBI's Multiple Risk Factor Intervention Trial (MRFIT) studied the relationship between heart disease and serum cholesterol levels in 362,000 men and found that annual deaths from CHD varied from slightly less than one per thousand at serum cholesterol levels below 140 mg/dL, to about two per thousand for serum cholesterol levels above 300 mg/dL, once again a trivial difference. Dr. John LaRosa of the American Heart Association claimed that the curve for CHD deaths began to "inflect" after 200 mg/dL, when in fact the "curve" was a very gradually sloping straight line that could not be used to predict whether serum cholesterol above certain levels posed a significantly greater risk for heart disease. One unexpected MRFIT finding the media did not report was that deaths from all causes—cancer, heart disease, accidents, infectious disease, kidney failure, etc.—were substantially greater for those men with cholesterol levels below 160 mg/dL.[33]

LIPID RESEARCH CLINICS TRIAL

What was needed to resolve the validity of the lipid hypothesis once and for all was a well-designed, long-term diet study that compared coronary heart disease events in those on traditional foods with those whose diets contained high levels of vegetable oils—but the proposed Diet-Heart study designed to test just that had been cancelled without fanfare years earlier. In view of the fact that orthodox medical agencies were united in their promotion of margarine and vegetable oils over animal foods containing cholesterol and animal fats, it is surprising that the official literature can cite only a handful of experiments indicating that dietary cholesterol has "a major role in determining blood cholesterol levels." One of these was a study involving 70 male prisoners directed by Fred Mattson[34]—the same Fred Mattson who had pressured the American Heart Association into removing any reference to hydrogenated fats from their diet-heart statement a decade earlier. Funded in part by Procter and Gamble, the research contained a number of serious flaws: selection of subjects for the four groups studied was not randomized; the experiment inexcusably eliminated "an equal number of subjects with the highest and lowest cholesterol values;" twelve additional subjects dropped out, leaving some of the groups too small to provide valid conclusions; and statistical manipulation of the results was shoddy. But the biggest flaw was that the subjects receiving cholesterol did so in the form of reconstituted powder—a totally artificial diet. Mattson's discussion did not even address the possibility that the liquid formula diet he used might affect blood cholesterol differently than would a whole foods diet when, in fact, many other studies indicated that this is the case. The culprit, in fact, in liquid protein diets appears to be oxidized cholesterol, formed during the high-temperature drying process, which seems to initiate the buildup of plaque in the arteries.[35] Powdered milk containing oxidized cholesterol is added to reduced fat milk—to give it body—which the American public has accepted as a healthier choice than whole milk. It was purified, oxidized cholesterol that Kritchevsky and others used in their experiments on vegetarian rabbits.

The NHLBI argued that a diet study using whole foods and involving the whole population would be too difficult to design and too expensive to carry out. But the NHLBI did have funds available to sponsor the massive Lipid Research Clinics Coronary Primary Prevention Trial in which all subjects were placed on a diet low in cholesterol and saturated fat. Subjects were divided into two groups, one of which took a cholesterol-lowering drug and the other a placebo. Working behind the scenes, but playing a key role in both the design and implementation of the trials, was Dr. Fred Mattson, formerly of Procter and Gamble.

An interesting feature of the study was the fact that a good part of the trial's one-hundred-and-fifty-million-dollar budget was devoted to group sessions in which trained dieticians taught both groups of study participants how to choose "heart-friendly" foods—margarine, egg replacements, processed cheese, baked goods made with vegetable shortenings, in short the vast array of manufactured foods awaiting consumer acceptance. As both groups received dietary indoctrination, study results could support no claims about the relation of diet to heart disease. Never-

theless, when the results were released, both the popular press and medical journals portrayed the Lipid Research Clinics trials as the long-sought proof that animal fats were the cause of heart disease. Rarely mentioned in the press was the ominous fact that the group taking the cholesterol-lowering drugs had an increase in deaths from cancer, stroke, violence and suicide.[36]

LRC researchers claimed that the group taking the cholesterol-lowering drug had a 17 percent reduction in the rate of CHD, with an average cholesterol reduction of 8.5 percent. This allowed LRC trials Director Basil Rifkind to claim that "for each 1% reduction in cholesterol, we can expect a 2% reduction in CHD events." The statement was widely circulated even though it represented a completely invalid representation of the data, especially in light of the fact that when the lipid group at the University of Maryland analyzed the LRC data, they found no difference in CHD events between the group taking the drug and those on the placebo.

A number of clinicians and statisticians participating in a 1984 Lipid Research Clinics Conference workshop, including Michael Oliver and Richard Krommel, were highly critical of the manner in which the LRC results had been tabulated and manipulated. The conference, in fact, went very badly for the NHLBI, with critics of the lipid hypothesis almost outnumbering supporters. One participant, Dr. Beverly Teter of the University of Maryland's lipid group, was delighted with the state of affairs. "It's wonderful'" she remarked to Basil Rifkind, study coordinator, "to finally hear both sides of the debate. We need more meetings like this" His reply was terse and sour: "No we don't."

NATIONAL CHOLESTEROL CONSENSUS CONFERENCE

Dissenters were again invited to speak briefly at the NHLBI-sponsored National Cholesterol Consensus Conference held later that year, but their views were not included in the panel's report, for the simple reason that the report was generated by NHLBI staff before the conference convened. Dr. Teter discovered this when she picked up some papers by mistake just before the conference began, and found they contained the consensus report, already written, with just a few numbers left blank. Kritchevsky represented the lipid hypothesis camp with a humorous five-minute presentation, full of ditties. Edward Ahrens, a respected researcher, raised strenuous objections about the "consensus," only to be told that he had misinterpreted his own data, and that if he wanted a conference to come up with different conclusions, he should pay for it himself.

The 1984 Cholesterol Consensus Conference final report was a whitewash, containing no mention of the large body of evidence that conflicted with the lipid hypothesis. One of the blanks was filled with the number 200. The document defined all those with cholesterol levels above 200 mg/dL as "at risk" even though the most ardent supporters of the lipid hypothesis had surmised in print that 240 should be the magic cutoff point. The choice of 200 had nothing to do with science and everything to do with the procurement of future funding. As the Conference began, Enig and several others overheard a discussion on the cutoff number between James Cleeman (National Cholesterol Education Program coordinator), Claude Lenfant (NHLBI director) and Basil Rifkin (director of the Lipid Research Clinics trial). Rifkin said to the other two: "But we can't have the cutoff at 240; it has to be at 200 or we won't have enough people to test."

The final report of the Cholesterol Consensus Conference called for mass cholesterol screening, Such screening would, in fact, need to be carried out on a massive scale as the federal medical bureaucracy, by picking the number 200, had defined the vast majority of the American adult population as "at risk." The report resurrected the ghost of Norman Jolliffe and his Prudent Diet by suggesting the avoidance of saturated fat and cholesterol for all Americans now defined as "at risk," and specifically advised the replacement of butter with margarine.

The Consensus Conference also provided a launching pad for the nationwide National Cholesterol Education Program, which had the stated goal of "changing physicians' attitudes." NHLBI-funded studies had determined that while the general population had bought into the lipid hypothesis, and was dutifully using margarine and buying low-cholesterol foods, the medical profession remained skeptical. A large "Physicians Kit" was sent to all doctors in America, compiled in part by the American Pharmaceutical Association, whose representatives served on the NCEP coordinating committee. Doctors were taught the importance of cholesterol screening, the advantages of cholesterol-lowering drugs and the unique benefits of the Prudent Diet. NCEP materials told every doctor in America to recommend the use of margarine rather than butter.

SCREENING FOR EVERYONE

In November of 1986, the *Journal of the American Medical Association* published a series on the Lipid Research Clinics trials, including "Cholesterol and Coronary Heart Disease: A New Era" by longtime American Heart Association member Scott Grundy, MD, PhD.[37] The article is a disturbing combination of euphoria and agony—euphoria at the forward movement of the lipid hypothesis juggernaut, and agony over the elusive nature of real proof. "The recent consensus conference on cholesterol. . . implied that levels between 200 and 240. . carry at least a mild increase in

risk, which they obviously do. . .” said Grundy, directly contradicting an earlier statement that “Evidence relating plasma cholesterol levels to atherosclerosis and CHD has become so strong as to leave little doubt of the etiologic connection.” Grundy called for “. . . the simple step of measuring the plasma cholesterol level in all adults. . . those found to have elevated cholesterol levels can be designated as at high risk and thereby can enter the medical care system. . . an enormous number of patients will be included.” Who benefits from “the simple step of measuring the plasma cholesterol level in all adults?” Why, hospitals, laboratories, pharmaceutical companies, the vegetable oil industry, margarine manufacturers, food processors and, of course, medical doctors. “Many physicians will see the advantages of using drugs for cholesterol lowering. . .” said Grundy, even though “a positive benefit/risk ratio for cholesterol-lowering drugs will be difficult to prove.” The cost in the US of cholesterol screening and cholesterol-lowering drugs alone now stands at sixty billion dollars per year, even though a positive risk/benefit ratio for such treatment has never been established. Physicians, however, have “seen the advantages of using drugs for cholesterol lowering” as a way of creating patients out of healthy people.

Grundy was equally schizophrenic about the benefits of dietary modification. “Whether diet has a long term effect on cholesterol remains to be proved,” he stated, but “Public health advocates furthermore can play an important role by urging the food industry to provide palatable choices of foods that are low in cholesterol, saturated fatty acids and total calories.” Such foods, almost by definition, contain partially hydrogenated vegetable oils that imitate the advantages of animal fats. Grundy knew that the *trans* fats were a problem, that they raised serum cholesterol and contributed to the etiology of many diseases—he knew because a year earlier, at his request, Mary Enig had sent him a package of data detailing numerous studies that gave reason for concern, which he acknowledged in a signed letter as “an important contribution to the ongoing debate.”

Other mouthpieces of the medical establishment fell in line after the Consensus Conference. In 1987 the National Academy of Science (NAS) published an overview in the form of a handout booklet containing a whitewash of the *trans* problem and a pejorative description of palm oil—a natural fat high in beneficial saturates and monounsaturates that, like butter, has nourished healthy population groups for thousands of years, and, also like butter, competes with hydrogenated fats because it can be used as a shortening. The following year the Surgeon General’s Report on Nutrition and Health emphasized the importance of making low-fat foods more widely available. Project LEAN (Low-Fat Eating for America Now) sponsored by the J. Kaiser Family Foundation and a host of establishment groups such as the America Heart Association, the American Dietetic Association, the American Medical Association, the USDA, the National Cancer Institute, Centers for Disease Control and the National Heart, Lung and Blood Institute announced a publicity campaign to “aggressively promote foods low in saturated fat and cholesterol in order to reduce the risk of heart disease and cancer.”

NATIONAL FOOD PROCESSORS ASSOCIATION CONFERENCE

The following year, Enig joined Frank McLaughlin, Director of the Center for Business and Public Policy at the University of Maryland, in testimony before the National Food Processors Association. It was a closed conference, for NFPA members only. Enig and McLaughlin had been invited to give “a view from academia.” Enig presented a number of slides and warned against singling out classes of fats and oils for special pejorative labeling. A representative from Frito-Lay took umbrage at Enig’s slides, which listed amounts of *trans* fats in Frito-Lay products. Enig offered to redo the analyses if Frito-Lay would fund the research. “If you’d talk different, you’d get money,” he said.

Enig urged the association to endorse accurate labeling of *trans* fats in all food items but conference participants—including representatives from most of the major food processing giants—preferred a policy of “voluntary labeling” that did not unnecessarily alert the public to the presence of *trans* fats in their foods. To date they have prevailed in preventing the inclusion of *trans* fats on nutrition labels.

Enig’s cat and mouse game with Hunter and Applewhite of the Institute of Shortening and Edible Oils continued throughout the later years of the 1980s. Their modus operandi was to pepper the literature with articles that downplayed the dangers of *trans* fats, to use their influence to prevent opposing points of view from appearing in print and to follow-up the few alarmist articles that did squeak through with “definitive rebuttals.” In 1987 Enig submitted a paper on *trans* fatty acids in the US diet to the American Journal of Clinical Nutrition, as a reply to the erroneous 1985 FASEB report as well as to Hunter and Applewhite’s influential 1986 article, which by even the most conservative analysis underestimated the average American consumption of partially hydrogenated fats. Editor-in-chief Albert Mendeloff, MD rejected Enig’s rebuttal as “inappropriate for the journal’s readership.” His rejection letter invited her to resubmit her paper if she could come up with “new evidence.” In 1991, the article finally came out in a less prestigious publication, the *Journal of the American College of Nutrition*,[38] although Applewhite did his best to

coerce editor Mildred Seelig into removing it at the last minute. Hunter and Applewhite submitted letters and then an article of rebuttal to the *American Journal of Clinical Nutrition*,[39] which were published shortly thereafter. In the article, entitled "Reassessment of *trans* fatty acid availability in the US diet," Hunter and Applewhite argued that the amount of *trans* in the American diet had actually declined since 1984, due to the introduction of soft margarines and tub spreads. The media fell in line with their pronouncements, with numerous articles by food writers recommending low-*trans* tub spreads, made from polyunsaturated vegetable oils, as the sensible alternative to saturated fat from animal sources—not surprising as most newspapers rely on the International Food Information Council, an arm of the food processing industry, for their nutrition information.

OTHER RESEARCH ON *TRANS*

Enig and the University of Maryland group were not alone in their efforts to bring their concerns about the effect of partially hydrogenated fats before the public. Fred Kummerow at the University of Illinois, blessed with independent funding and an abundance of patience, carried out a number of studies that indicated that the *trans* fats increased risk factors associated with heart disease, and that vegetable oil-based fabricated foods such as Egg Beaters cannot support life.[40] George Mann, formerly with the Framingham project, possessed neither funding nor patience—he was, in fact, very angry with what he called the Diet-Heart scam. His independent studies of the Masai in Africa,[41] whose diet is extremely rich in cholesterol and saturated fat, and who are virtually free of heart disease, had convinced him that the lipid hypothesis was "the public health diversion of this century. . . the greatest scam in the history of medicine."[42] He resolved to bring the issue before the public by organizing a conference in Washington DC in November of 1991.

"Hundreds of millions of tax dollars are wasted by the bureaucracy and the self-interested Heart Association," he wrote in his invitation to participants. "Segments of the food industry play the game for profits. Research on the true causes and prevention is stifled by denying funding to the 'unbelievers.' This meeting will review the data and expose the rascals."

The rascals did their best to prevent the meeting from taking place. Funding promised by the Greenwall Foundation of New York City was later withdrawn, so Mann paid most of the bills. A press release sent as a dirty trick to speakers and participants wrongly announced that the conference had been cancelled. Several speakers did in fact renege at the last minute on their commitment to attend, including the prestigious Dr. Roslyn Alfin-Slater and Dr. Peter Nixon of London. Dr. Eliot Corday of Los Angeles cancelled after being told that his attendance would jeopardize future funding.

The final pared-down roster included Dr. George Mann, Dr. Mary Enig, Dr. Victor Herbert, Dr. Petr Skrabenek, William B. Parsons, Jr., Dr. James McCormick, a physician from Dublin, Dr. William Stehbens from New Zealand, who described the normal protective process of arterial thickening at points of greatest stress and pressure, and Dr. Meyer Texon an expert in the dynamics of blood flow. Mann, in his presentation, blasted the system that had foisted the lipid hypothesis on a gullible public. "You will see," he said, "that many of our contributors are senior scientists. They are so for a reason that has become painfully conspicuous as we organized this meeting. Scientists who must go before review panels for their research funding know well that to speak out, to disagree with this false dogma of Diet/Heart, is a fatal error. They must comply or go unfunded. I could show a list of scientists who said to me, in effect, when I invited them to participate: 'I believe you are right, that the Diet-Heart hypothesis is wrong, but I cannot join you because that would jeopardize my perks and funding.' For me, that kind of hypocritical response separates the scientists from the operators—the men from the boys."

THE NATION WELL OILED

By the nineties the operators had succeeded, by slick manipulation of the press and of scientific research, in transforming America into a nation that was well and truly oiled. Consumption of butter had bottomed out at about five grams per person per day, down from almost 18 at the turn of the century. Use of lard and tallow had been reduced by two-thirds. Margarine consumption had jumped from less than two grams per person per day in 1909 to about 11 in 1960. Since then consumption figures had changed little, remaining at about 11 grams per person per day—perhaps because knowledge of margarine's dangers had been slowly seeping out to the public. However, most of the *trans* fats in the current American diet come not from margarine but from shortening used in fried and fabricated foods. American shortening consumption of 10 grams per person per day held steady until the 1960s, although the content of that shortening had changed from mostly lard, tallow and coconut oil—all natural fats—to partially hydrogenated soybean oil. Then shortening consumption shot up and by 1993 had tripled to over 30 grams per person per day.

But the most dramatic overall change in the American diet was the huge increase in the consumption of liquid vegetable oils, from slightly less than two grams per person per day in 1909 to over 30 in 1993—a fifteen-fold increase.

POLYUNSATURATES DANGERS

The irony is that these trends have persisted concurrently with revelations about the dangers of polyunsaturates. Because polyunsaturates are highly subject to rancidity, they increase the body's need for vitamin E and other antioxidants. Excess consumption of vegetable oils is especially damaging to the reproductive organs and the lungs—both of which are sites for huge increases in cancer in the US. In test animals, diets high in polyunsaturates from vegetable oils inhibit the ability to learn, especially under conditions of stress; they are toxic to the liver; they compromise the integrity of the immune system; they depress the mental and physical growth of infants; they increase levels of uric acid in the blood; they cause abnormal fatty acid profiles in the adipose tissues; they have been linked to mental decline and chromosomal damage; they accelerate aging. Excess consumption of polyunsaturates is associated with increasing rates of cancer, heart disease and weight gain; excess use of commercial vegetable oils interferes with the production of prostaglandins leading to an array of complaints ranging from autoimmune disease to PMS. Disruption of prostaglandin production leads to an increased tendency to form blood clots, and hence myocardial infarction, which has reached epidemic levels in America.[43]

Vegetable oils are more toxic when heated. One study reported that polyunsaturates turn to varnish in the intestines. A study by a plastic surgeon found that women who consumed mostly vegetable oils had far more wrinkles than those who used traditional animal fats. A 1994 study appearing in *The Lancet* showed that almost three quarters of the fat in artery clogs is unsaturated. The "artery clogging" fats are not animal fats but vegetable oils.[44]

Those who have most actively promoted the use of polyunsaturated vegetable oils as part of a Prudent Diet are well aware of their dangers. In 1971, William B. Kannel, former director of the Framingham study, warned against including too many polyunsaturates in the diet. A year earlier, Dr. William Connor of the American Heart Association issued a similar warning, and Frederick Stare reviewed an article which reported that the use of polyunsaturated oils caused an increase in breast tumors. And Kritchevsky, way back in 1969, discovered that the use of corn oil caused an increase in atherosclerosis.[45]

As for the *trans* fats, produced in vegetable oils when they are partially hydrogenated, the results that are now in the literature more than justify concerns of early investigators about the relation between *trans* fats and both heart disease and cancer. The research group at the University of Maryland found that *trans* fatty acids not only alter enzymes that neutralize carcinogens, and increase enzymes that potentiate carcinogens, but also depress milk fat production in nursing mothers and decrease insulin binding.[46] In other words, *trans* fatty acids in the diet interfere with the ability of new mothers to nurse successfully and increase the likelihood of developing diabetes.

Unpublished work indicates that *trans* fats contribute to osteoporosis. Hanis, a Czechoslovakian researcher, found that *trans* consumption decreased testosterone, caused the production of abnormal sperm and altered gestation.[47] Koletzko, a German pediatric researcher found that excess *trans* consumption in pregnant mothers predisposed them to low birth weight babies.[48] *Trans* consumption interferes with the body's use of omega-3 fatty acids found in fish oils, grains and green vegetables, leading to impaired prostaglandin production.[49] George Mann confirmed that *trans* consumption increases the incidence of heart disease.[50] In 1995, European researchers found a positive correlation between breast cancer rates and *trans* consumption.[51]

Until the 1995 study, only the disturbing revelations of Dutch researchers Mensink and Katan, in 1990, received front page coverage. Mensink and Katan found that margarine consumption increased coronary heart disease risk factors.[52] The industry—and the press—responded by promoting tub spreads, which contain reduced amounts of *trans* compared to stick margarine. For the general population, these *trans* reductions have been more than offset by changes in the types of fat used by the fast food industry. In the early 1980s, Center for Science in the Public Interest campaigned against the use of beef tallow for frying potatoes. Before that they campaigned against the use of tallow for frying chicken and fish. Most fast food companies switched to partially hydrogenated soybean oil for all fried foods. Some deep fried foods have been tested at almost 50 percent *trans*.[53]

Epidemiologist Walter Willett at Harvard worked for many years with flawed data bases which did not identify *trans* fats as a dietary component. He found a correlation with dietary fat consumption and both heart disease and cancer. After his researchers contacted Enig about the *trans* data, they developed a more valid data base that was used in the analysis of the massive Nurses Study. When Willett's group separated out the *trans* component in their analyses, they were able to confirm greater rates of cancer in those consuming margarine and vegetable shortenings—not butter, eggs, cheese and meat.[54] The correlation of *trans* fat consumption and cancer was never published, but was reported at the Baltimore Data Bank Conference in 1992.

In 1993 Willett's research group at Harvard found that *trans* contributed to heart disease,[55] and this study was not ignored, but received much fanfare in the press. Willett's first reference in

his report was Enig's work on the *trans* content of common foods.

The industry continues to argue that American *trans* consumption is a low six to eight grams per person per day, not enough to contribute to today's epidemic of chronic disease. Total per capita consumption of margarine and shortening hovers around 40 grams per person per day. If these products contain 30 percent *trans* (many shortenings contain more) then average consumption is about 12 grams per person per day. In reality, consumption figures can be dramatically higher for some individuals. A 1989 Washington Post article documented the diet of a teenage girl who ate 12 donuts and 24 cookies over a three-day period. Total *trans* worked out to at least 30 grams per day, and possibly much more. The fat in the chips that teenagers consume in abundance may contain up to 48 percent *trans* which translates into 45.6 grams of *trans* fat in a small ten-ounce bag of snack chips—which a hungry teenager can gobble up in a few minutes. High school sex education classes do not teach American teenagers that the altered fats in their snack foods may severely compromise their ability to have normal sex, conceive, give birth to healthy babies and successfully nurse their infants.

BENEFITS OF ANIMAL FATS

Foods containing *trans* fat sell because the American public is afraid of the alternative—saturated fats found in tallow, lard, butter, palm and coconut oil, fats traditionally used for frying and baking. Yet the scientific literature delineates a number of vital roles for dietary saturated fats—they enhance the immune system,[56] are necessary for healthy bones,[57] provide energy and structural integrity to the cells,[58] protect the liver[58] and enhance the body's use of essential fatty acids.[60] Stearic acid, found in beef tallow and butter, has cholesterol lowering properties and is a preferred food for the heart.[61] As saturated fats are stable, they do not become rancid easily, do not call upon the body's reserves of antioxidants, do not initiate cancer, do not irritate the artery walls.

Your body makes saturated fats, and your body makes cholesterol—about 2000 mg per day. In general, cholesterol that the average American absorbs from food amounts to about 100 mg per day. So, in theory, even reducing animal foods to zero will result in a mere 5 percent decrease in the total amount of cholesterol available to the blood and tissues. In practice, such a diet is likely to deprive the body of the substrates it needs to manufacture enough of this vital substance; for cholesterol, like saturated fats, stands unfairly accused. It acts as a precursor to vital corticosteroids, hormones that help us deal with stress and protect the body against heart disease and cancer; and to the sex hormones like androgen, testosterone, estrogen and progesterone; it is a precursor to vitamin D, a vital fat-soluble vitamin needed for healthy bones and nervous system, proper growth, mineral metabolism, muscle tone, insulin production, reproduction and immune system function; it is the precursor to bile salts, which are vital for digestion and assimilation of fats in the diet. Recent research shows that cholesterol acts as an antioxidant.[62] This is the likely explanation for the fact that cholesterol levels go up with age. As an antioxidant, cholesterol protects us against free radical damage that leads to heart disease and cancer. Cholesterol is the body's repair substance, manufactured in large amounts when the arteries are irritated or weak. Blaming heart disease on high serum cholesterol levels is like blaming firemen who have come to put out a fire for starting the blaze.

Cholesterol is needed for proper function of serotonin receptors in the brain.[63] Serotonin is the body's natural "feel-good" chemical. This explains why low cholesterol levels have been linked to aggressive and violent behavior, depression and suicidal tendencies.

Mother's milk is especially rich in cholesterol and contains a special enzyme that helps the baby utilize this nutrient. Babies and children need cholesterol-rich foods throughout their growing years to ensure proper development of the brain and nervous system. Dietary cholesterol plays an important role in maintaining the health of the intestinal wall,[64] which is why low-cholesterol vegetarian diets can lead to leaky gut syndrome and other intestinal disorders.

Animal foods containing saturated fat and cholesterol provide vital nutrients necessary for growth, energy and protection from degenerative disease. Like sex, animal fats are necessary for reproduction. Humans are drawn to both by powerful instincts. Suppression of natural appetites leads to weird nocturnal habits, fantasies, fetishes, bingeing and splurging.

Animal fats are nutritious, satisfying and they taste good. "Whatever is the cause of heart disease," said the eminent biochemist Michael Gurr in a recent article, "it is not primarily the consumption of saturated fats."[65] And yet the high priests of the lipid hypothesis continue to lay their curse on the fairest of culinary pleasures—butter and Bernaise, whipped cream, souffles and omelets, full-bodied cheeses, juicy steaks and pork sausage.

COMING FULL CIRCLE, YET LEARNING NOTHING

On April 30, 1996 a senior researcher named David Kritchevsky received the American Oil Chemists' Society's Research Award in recognition of his accomplishments as a "researcher on cancer and atherosclerosis as well as cholesterol metabolism." His accomplishments include co-authorship of more than 370 research papers, one of which appeared a month later in the

American Journal of Clinical Nutrition.[66] "Position paper on *trans* fatty acids" continued the debate on *trans* fats that began in the same journal with Hunter and Applewhite's 1986 attack on Enig's research. "A controversy has arisen about the potential health hazards of *trans* unsaturated fatty acids in the American diet," wrote Kritchevsky and his coauthors.

Actually the controversy dates back to 1954. In the rabbit studies that launched Kritchevsky on his career, the researcher actually found that cholesterol fed with Wesson oil "markedly accelerated" the development of cholesterol-containing low-density lipoproteins; and cholesterol fed with shortening gave cholesterol levels twice as high as cholesterol fed alone.[67] Enig's work—and that of Kummerow and Mann and several others—merely confirmed what Kritchevsky ascertained decades ago but declined to publicize, that vegetable oils, and particularly partially hydrogenated vegetable oils, are bad news.

But the "Position paper on *trans* fatty acids" took no position at all. Studies have given contradictory results, said the authors, and the amount of *trans* in the average American diet is very difficult to determine. As for labeling, "There is no clear choice of how to include *trans* fatty acids on the nutrition label. The database is insufficient to establish a classification scheme for these fats." There may be problems with *trans*, says the senior researcher, but their use "helps to reduce the intake of dietary fats higher in saturated fatty acids. Also, vegetable fats are not a source of dietary cholesterol, unlike saturated animal fats." Kritchevsky and his coauthors conclude that physicians and nutritionists should "focus on a further decrease in total fat intake and especially the intake of saturated fat. . . A reduction in total fat intake simplifies the problem, because all fats in the diet decrease and choices are unnecessary." However, even senior scientists find that fence straddling is necessary. "We may conclude," wrote Kritchevsky and his colleagues, "that consumption of liquid vegetable oils is preferable to solid fats."

FOOTNOTE:

Early this year, 1998, a symposium entitled "Evolution of Ideas about the Nutritional Value of Dietary Fat" reviewed the many flaws in the lipid hypothesis and highlighted a study in which mice fed purified diets died within 20 days but whole milk kept the mice alive for several months.[68] One of the participants was David Kritchevsky who noted that the use of low-fat diets and drugs in intervention trials, "did not affect overall CHD mortality." Ever with a finger in the wind, this influential Founding Father of the lipid hypothesis concluded thus: "Research continues apace and, as new findings appear, it may be necessary to reevaluate our conclusions and preventive medicine policies."

Mary G. Enig, PhD is an expert of international renown in the field of lipid biochemistry. She has headed a number of studies on the content and effects of trans *fatty acids in America and Israel. She is a licensed nutritionist, certified by the Certification Board for Nutrition Specialists, a qualified expert witness, nutrition consultant to individuals, industry and state and federal governments, contributing editor to a number of scientific publications, Fellow of the American College of Nutrition and President of the Maryland Nutritionists Association. She is the author of over 60 technical papers and presentations. Dr. Enig is currently working on the exploratory development of an adjunct therapy for AIDS using complete medium chain saturated fatty acids from whole foods. She is Vice President of the Weston A Price Foundation and Scientific Editor of* Wise Traditions *as well as the author of* Know Your Fats: The Complete Primer for Understanding the Nutrition of Fats, Oils, and Cholesterol, Bethesda Press. *She is the mother of three healthy children brought up on whole foods including butter, cream, eggs and meat.*

Sally Fallon is the author of Nourishing Traditions: The Cookbook that Challenges Politically Correct Nutrition and the Diet Dictocrats *(with Mary G. Enig, PhD), a thought-provoking guide to traditional foods with a startling message: Animal fats and cholesterol are not villains but vital factors in the diet, necessary for normal growth, proper function of the brain and nervous system, protection from disease and optimum energy levels. She joined forces with Enig again to write* Eat Fat, Lose Fat*, and has published numerous articles on the subject of diet and health. She serves as President of the Weston A. Price Foundation and is the founder of A Campaign for Real Milk. Her four healthy children were raised on whole foods including butter, cream, eggs and meat.*

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Know Your Fats

CHOLESTEROL LOWERING AND LOWFAT DIETS FOR CHILDREN

By Mary G. Enig, PhD

The American Academy of Pediatrics recommends that children two years old and older should eat a diet of fruits, vegetables, whole grains, lowfat and non-fat dairy products, beans, fish and lean meats. The guidelines also recommend very low amounts of saturated and *trans* fats. The "experts" are increasingly urging strict adherence to this diet in children. "The idea that heart disease starts in the 50s has been substantially discounted," says Dr. Robert Eckel of the University of Colorado and former president of the American Heart Association. "Saturated fat is always an enemy to the arteries, at any age."[1]

I have described some of the harmful consequences of lowfat diets for growing children in previous columns (see westonaprice.org/knowyourfats/diet_children.html). The purported rationale for putting children on fat-restricted diets involves preventing future obesity and heart disease. Yet one study indicates that children put on lowfat diets actually develop markers for heart disease. Children on lowfat diets whose genes would normally have been producing the desirable light and fluffy form of LDL-cholesterol started to make the dangerous small and dense form of LDL.[2]

LOWFAT DIETS FOR CHILDREN?

Promoters of lowfat diets have seen to it that whole milk has been virtually eliminated in schools. This new policy is predicated on the assumption that the fat in whole milk will make children become fat. Yet a recent study on children in Sweden revealed that lower fat intake was associated with higher body mass index and greater insulin resistance.[3] Children on lowfat diets also consumed more sugar. Since the beverage choice for American children in schools today is either reduced-fat milk or chocolate milk, greater sugar consumption will no doubt be a consequence of the fat-restriction policy.

More cause for alarm comes from another recent study, this one published in *Human Reproduction*.[4] The risk of anovulatory infertility was found to be 27 percent lower in women who ate at least one portion of high-fat dairy food per day compared with women who had one high-fat serving of dairy per week, or even less. Women who ate two or more portions of lowfat dairy foods a day increased their risk of ovulation-related infertility by 85 percent.

The researchers concluded that women who want to get pregnant should consume high-fat dairy products but, once pregnant, switch back to lowfat foods. The assumption is that ovulation can be restored in adult women by switching from lowfat to full-fat dairy products. But what happens in girls who are denied healthy dairy fats throughout childhood, even, it seems, in the womb? Will they be able to become pregnant by

But what happens in girls who are denied healthy dairy fats throughout childhood, even, it seems, in the womb?

DIET FOODS MAY CAUSE WEIGHT GAIN

As health officials continue to harp on the dangers of weight gain, parents are increasingly likely to give low-calorie products to their children. But studies with rats provide further evidence of the folly of this policy. Young animals given low-calorie version of foods ended up overeating, whether they were lean or obese; however, older adolescent rats fed diet foods did not show the same tendency to overeat.[9] "Diet foods are probably not a good idea for growing youngsters," said Professor David Pierce, head of the study. But that is exactly what the "experts" recommend—lowfat versions of dairy products and meat, and a restriction of animal fats like butter.

Not content with statin therapy for the small percentage of children with hereditary familial hypercholesterolemia, the American Heart Association now endorses statin treatment in children for a variety of risk factors.

consuming full-fat dairy products for the first time when they are adults? This is a dangerous assumption to make since the vitamins in dairy fats are essential for the development of the reproductive system throughout the growing years.

Some researchers are urging caution. Dr. John Kostyak and a team from Pennsylvania State University recently warned in the online magazine *Nutrition Journal* that so-called "muesli mothers" are taking adult dietary messages to extremes and inflicting them on their children. "Sufficient fat must be included in the diet for children to support normal growth and development," says Kostyak. Unfortunately, the fats he recommends are the "good fats," such as olive oil and sunflower oil. However, some commentators are urging full-fat dairy products for children under five—contradicting US government policy that urges restriction of dairy fats after the age of two.[5]

CHOLESTEROL LOWERING

The pharmaceutical industry seems intent on putting growing children on cholesterol-lowering drugs, with a carefully orchestrated campaign that first targets children with a condition called hereditary familial hypercholesterolemia, chronic "high" cholesterol levels of genetic origin. In a recently published meta-analysis, researchers reported no side effects in children given cholesterol-lowering statin drugs compared to a placebo.[6] With the drugs, they were able to reduce total cholesterol by 25 percent and LDL-cholesterol by 30 percent. The report contains a number of caveats—namely that longer-term studies are needed, as current studies have only examined possible side effects such as growth problems and retarded sexual development for a period of one or two years. Nevertheless, the author, Dr. Barbara A. Hutten from Academic Medical Center, Amsterdam, Netherlands, stated with confidence, "When a child has been diagnosed with heterozygous familial hypercholesterolemia, statin treatment should be considered for all children older than eight years."

Not content with statin therapy for the small percentage of children with hereditary familial hypercholesterolemia, the American Heart Association now endorses statin treatment in children for a variety of risk factors: ". . . just as with adults, there are certain risk factors in children that may call for more aggressive treatment."[7] The National Cholesterol Education Program (NCEP) has drawn up new guidelines that would include diabetes, overweight and a family history of cardiovascular disease, as well as familial hypercholesterolemia, as risk factors in children that could be treated with statins. Even "male gender" has been singled out as a risk factor!

NCEP estimates that under these guidelines, from 36 to 46 percent of children and adolescents would be targeted for cholesterol-lowering measures, which could include statin treatment. Although the American Heart Association statement notes that "There is a real need for ongoing research regarding drug therapy of high risk lipid abnormalities in children, *particularly regarding long-term efficacy and safety* [emphasis mine]," it seems clear that plans to lower cholesterol in children are going forward.

A NOTE OF CAUTION

A note of caution comes from the expert committee of the US Preventive Services Task Force.[8] The committee examined randomized, control clinical trials and all available evidence, carefully considering ten key questions concerning the effectiveness, risks and accuracy of screening; the effectiveness and risks of diet, drug and exercise interventions for managing cholesterol and preventing cardiovascular disease; and whether reducing blood cholesterol

CAN CHOLESTEROL PROTECT AGAINST FETAL ALCOHOL SYNDROME?

Fetal Alcohol Spectrum Defects (FASD) includes numerous abnormalities, such as neurological, craniofacial and cardiac malformations. In studies with zebra fish, researchers found that alcohol interferes with embryonic development by disrupting cholesterol-dependent activation of a critical signaling molecule. But with cholesterol supplementation of the alcohol-exposed embryos, FASD-like defects were prevented. The defects resulted from minimal alcohol exposure, equivalent to a 120-pound woman drinking one 12-ounce beer.[10] This study raises the specter of increased FASD in human children as a result of lowfat diets for pregnant women.

levels in youth reduces the risk of high cholesterol and cardiovascular disease in adulthood. Their conclusion: "The evidence is insufficient to recommend for or against routine screening for lipid disorders in infants, children, adolescents, or young adults (up to age 20). Evidence for effectiveness is lacking, of poor quality, or conflicting, and the balance of benefits and harms can not be determined."

The committee raised serious concerns about side effects. They found evidence of growth failure, nutritional dwarfing and inhibited progression of puberty in children on fat-restricted diets. They also reported that "lower fat intake was associated with lower levels of calcium, zinc, magnesium, phosphorus, vitamin E, vitamin B_{12}, thiamine, niacin and riboflavin."

As for prescribing statin drugs to children, the committee noted that these drugs have FDA approval for use in children only in cases of familial hypercholesterolemia. "There is no evidence that diet or exercise interventions in childhood lead to improved lipid profiles or better health outcomes in adulthood." Furthermore, statin drugs also have side effects, including liver damage, gastrointestinal problems and decreased absorption of vitamins and minerals.

Unfortunately, this report received scant publicity and will probably do little to stop the anti-cholesterol campaign in children. As a result, well-meaning parents and health officials will continue to apply cholesterol-lowering measures to young people—including starving them of nutrients—in the mistaken belief that they are protecting their health.

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Soy Alert!

NOT SO SOY HEALTHY FOR THE HEART

By Kaayla T. Daniel, PhD, CCN

Soy does not lower cholesterol, does not prevent heart disease and does not deserve an FDA-approved soy heart health claim. This amazing announcement comes from none other than the American Heart Association (AHA) published in the January 17, 2006 issue of its journal *Circulation*.

ATHLETES AT RISK

Not long before, University of Colorado researchers reported in the January issue of the *Journal of Clinical Investigation* that soy worsens cardiomyopathy, a form of heart disease that is very much on the rise, afflicting one in 500 Americans. Cardiomyopathy, defined as a weakening of the heart muscle or change in structure of the heart, is the leading cause of death among young athletes, a group that may consume a lot of soy in the form of protein powders and energy bars.

High levels of soy isoflavones—plant estrogens found in products like soy milk and soy nuts as well as many menopausal supplements—put women at risk for cardiovascular disease.

WOMEN AT RISK

Now investigators have found more damning evidence against soy. High levels of soy isoflavones—plant estrogens found in products like soy milk and soy nuts as well as many menopausal supplements—put women at risk for cardiovascular disease. The study—reported in the May, 2007 issue of *Journal of Women's Health*—began when Carl J. Pepine, MD, chief of cardiology at the University of Florida College of Medicine in Gainesville, along with ten other researchers from his own and five other medical institutions, aimed to find out whether women who have high concentrations of isoflavones in their blood had better vascular health. Subjects were participants in the Women's Ischemia Syndrome Evaluation (WISE) who had reported chest pain and were thus suspected to suffer from myocardial ischemia (defined as pathological loss of or reduction in blood flow—ischemia—to a part of the muscular tissue of the heart—myocardium).

More than 900 women have participated in the WISE project, which was founded a decade ago by the National Institutes of Health to study whether heart disease develops differently in women than in men. Because heart disease is more likely to occur after menopause, scientists have blamed waning estrogen levels. Dr. Pepine and his colleagues had expected that women with high levels of genistein (the primary isoflavone found in soybeans) would show improved vascular health, but found the opposite to be true. Speaking to a reporter for *Science News*, Dr. Pepine said: "There are a lot of women taking these things (isoflavone-rich products), without any direct evidence that they're beneficial." He warned that there is a "small but growing body of research suggesting there could be a down side to overindulging in them."

INDUSTRY RESPONSE

Industry response to mounting evidence for soy's lack of benefit has been entirely predictable: endless references to soy being both low in saturated fat and free of cholesterol (twin evils that "everyone knows" cause heart disease) combined with chipper reports of hot, new evidence "proving" that soy is the best thing for the heart since love. Although some of this hype has made it into the news—particularly in magazines where soy foods and soy milk are heavily advertised—a shift has definitely taken place. Health magazines are increasingly leaving soy off lists of healthy foods. These days they aren't yet reporting risks from soy, but they aren't singing its praises either.

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