

HEALTH AND STRESS

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CONFUSING CHOLESTEROL CONTROVERSY CONTINUES

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An elevated cholesterol level has long been considered to be the culprit responsible for coronary artery disease, stroke, and other manifestations of accelerated atherosclerosis. Over the past two decades, increased concentrations of other lipids have been incriminated, including triglycerides, low density lipoproteins (LDL), or specific apolipoprotein components.

Despite good evidence that such abnormalities are not present in at least half of heart attack victims, and that high homocysteine may be an even more important predictor, the cholesterol juggernaut continues to roll on. It is perpetuated by public health campaign illustrations showing how excess lipids precipitate out to form obstructive atherosclerotic lesions. Advertisements question consumers "Do you know your cholesterol count?", as if it were just as important as knowing your blood pressure.

The low cholesterol steamroller is fueled by a variety of vested commercial interests with huge fortunes at stake. While pharmaceutical companies who promote cholesterol lowering drugs in a fiercely competitive market immediately come to mind, the food industry has an even larger investment. Substitute or low fat butter and other dairy products have been around for years, but it seems that almost every supermarket food item now comes in a low fat version, e.g., soups, stews, sodas, salad dressings, cookies, potato chips, bread, and other bakery goods. Some of these, such as margarine and trans-fat cooking oils, may be more likely to contribute to atherosclerosis than the products they were designed to replace.

The manufacturers of all sorts of cholesterol and lipid measuring devices and the chemicals required to perform the tests are anxious to keep everyone cholesterol conscious. There are also the distributors of herbal and nutritional products that claim to lower cholesterol "naturally" without any of the side effects of drugs. Others promise to provide protection from free radical bombardment that oxidizes LDL "bad cholesterol", and thus makes it more damaging.

The public is so brainwashed, that many people believe that the lower your cholesterol, the healthier you will be or the longer you will live. Nothing could be further from the truth.

ALSO INCLUDED IN THIS ISSUE

The Hazards Of Low Cholesterol.....	2
Dietary Fat - How Much, What Kind?.....	3
The Egg And You.....	3
Oxidized Trans-Fats And Margarines.....	4
Deciphering The French Paradox - Alcohol, Red Wine, Fiber, Folate, Foie Gras, And More.....	5
The Americanization Of France.....	6
Stress, Cholesterol, And Longevity.....	6
The Role Of Physical Stress.....	7
The Commercialization Of Cholesterol And Contemporary Medicine.....	8

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The Hazards Of Low Cholesterol

It would seem reasonable to assume that eating lots of fatty foods would cause an increase in levels of blood lipids and cholesterol. It's also easy to visualize how excess amounts of these fat components could precipitate out as atherosclerotic deposits to clog up arteries in the brain and heart. The logical conclusion from the above would be that strictly limiting fat intake, and/or lowering cholesterol with medications, should significantly lower the likelihood of those heart attacks and strokes due to obstructive atherosclerosis.

Unfortunately, it's not that simple. A high fat diet does not necessarily mean that your cholesterol will soar or that you will be loaded with atherosclerosis. More importantly, the results of rigid restriction of fat intake in patients with high blood cholesterol and lipid levels are generally disappointing. In addition, the majority of patients admitted to the hospital for an acute heart attack or stroke have cholesterol and allied measurements that are well within normal limits.

Finally, although French and other diets tend to be high in foods that are rich in saturated animal fat, death rates due to coronary heart disease are much lower than those reported in the U.S and Great Britain.

Cholesterol is a basic building block for steroid hormones and other vital compounds, and there have been concerns that attempts to lower it by stringent dietary restriction and especially drugs with unknown long term side effects could boomerang. While obviously true for growing children, there is evidence that a low cholesterol is also associated with increased hazards and deaths in the elderly.

In a study of 937 healthy people aged 70 to 79, those with lower than normal blood levels of cholesterol were much more likely than their peers to suffer poor health and to die when followed over the next three years. Those who also had a low serum albumin were at even greater risk for functional decline, such as the ability to do normal housework chores or walk up a flight of stairs. The combination of low serum cholesterol and low albumin was associated with a 3.6 times greater risk of dying within the next 36 months.

Low cholesterol (under 160) has been linked with higher rates of depression, suicide, and other violent behaviors; about 10-15 percent of Americans fall into this classification. In a recent report, researchers measured depression and anxiety levels in healthy young women with naturally occurring low cholesterol levels. They found that two thirds scored high on depression or anxiety scales, compared to only one in four controls with normal or high values. With respect to specific types of lipids, those with low levels of triglycerides and LDL cholesterol, both of which are associated with less heart disease, had higher anxiety and depression ratings.

Depression can cause a severe loss in appetite which could eventually result in a reduction in cholesterol levels. However, a more plausible explanation is that low cholesterol levels reduce brain levels of serotonin. Low serotonin is a risk factor for violent behavior, suicide, depression and anxiety. The study concluded that women in their late teens or twenties with low cholesterol were at increased risk for depression and anxiety. Whether efforts to increase cholesterol and serotonin with diet and drugs could correct this is not clear.

Dietary Fat - How Much, What Kind?

The dietary fat taboo probably started 100 years ago, when the Russian physician Nikolai Anichkov demonstrated that he could produce atherosclerotic lesions in rabbits by feeding them a high-cholesterol diet. Little attention was paid to this until the Korean War, when autopsy studies performed on healthy young soldiers killed in battle revealed that more than three out of four showed significant coronary atherosclerosis, despite an average age of twenty two. It was presumed that this was due to their relatively high saturated fat diet, since Greek and Japanese populations, who consume much less animal fat, had heart attack rates that were almost 90 percent lower. On the other hand, autopsy studies of healthy, young American civilians killed in automobile and other accidents rarely revealed this significant increase in coronary atherosclerosis. What could explain this marked difference?

One suggestion was that the soldiers were under increased and chronic stress due to the persistent perils of combat, since stress is known to elevate serum cholesterol and accelerate atherosclerosis via increased free radical damage. However, it is far more likely that the lesions seen in Korean War casualties resulted from their diet of powdered eggs and other K rations rich in oxidized cholesterol products. In addition, a review of Anichkov's original experiments revealed that it was not pure cholesterol that he fed his rabbits but rather oxidized cholesterol. Cholesterol is a large, inert molecule, and in the event that excesses in the blood precipitate out, they do not result in the inflammatory type of atherosclerotic plaque that causes obstructive disease. Even low density lipoproteins, which cause the most injury tend not to invade the arterial wall unless they are oxidized.

The high cholesterol → atherosclerosis hypothesis is also refuted by numerous other observations. Coronary heart disease is relatively uncommon in the Massai natives in Africa or Eskimos in the Arctic, both of whom have high-fat diets. It is not the amount of fat but rather its composition that is important.

Eskimos have a 33 percent lower cardiovascular death rate than non-native residents. It has been suggested that this may result from their high fish diet, which is rich in omega-3 fatty acids that retard the development of atherosclerosis. The relative freedom from heart disease seen in Mediterranean peoples is thought to be due to their high consumption of olive oil, a monounsaturated fat with similar properties. At the height of the heart attack epidemic fifty years ago, mortality rates were lowest in mid-west dairy farming areas where fat intake was the highest. In other surveys, a decreased incidence of coronary heart disease was associated with increased consumption of fresh dairy products, probably because of their high antioxidant content.

The Egg And You

We are advised to restrict cholesterol consumption to 300 mg. per day, and since an egg contains well over 200 mg., two or three would quickly put you well over the daily limit. Several years ago, The New England Journal of Medicine reported a well-documented case of a healthy 86 year old man **who had consumed two dozen eggs every day for the past 15 years!** He had a special arrangement with the Nursing Home where he resided to purchase two dozen fresh eggs daily because he was convinced of their health benefits. Careful records confirmed that all of the eggs were eaten in various forms, and although this had probably been going on closer to twenty years, he had a **normal cholesterol and electrocardiogram and no indication of heart disease.**

Fresh eggs may actually be quite healthy because they contain antioxidants, folate, other B-vitamins and unsaturated fats that reduce the risk of heart attack. Unfortunately, they are often eaten with bacon, sausage and other fried foods that can more than offset their benefits. A National Institutes of Health study of over 100,000 men and women recently concluded that eating an egg a day had no effect on their cardiovascular health. However, that would not apply to powdered eggs, and other artificial dairy products like margarine that have high concentrations of trans-fats.

(Continued on Page 4)

Oxidized Trans-Fats And Margarines

The American diet is particularly high in oxidized fats, most of which are in the form of trans-fats or trans-fatty acids. These products were developed by food processing companies in the present century by a production process called hydrogenation. This "transforms" vegetable oils into more solid substances that resist spoiling, and behave more like butter or lard during baking and frying. The resultant trans-fat compounds are quite different in chemical composition than the fatty acids naturally present in food, and have far more serious health effects. They decrease HDL "good cholesterol", increase LDL "bad cholesterol", and can cause diabetes by interfering with insulin receptors, so that the pancreas has to constantly manufacture more insulin, and eventually becomes exhausted. Trans-fat chemicals alter the composition, size, and number of fat cells, thus promoting the development of obesity as well as diabetes. They can also contribute to cancer by accelerating the malignant changes initiated by various carcinogens.

Trans-fats are found in almost all commercially made donuts, crackers, cookies, pastries, deep-fat fried foods, potato and corn chips, confectionery, frosting and candy products, and margarine. For most Americans, fried foods are the biggest source of trans-fats. A medium order of fast food french fries contains four to five times as much trans-fat as one teaspoon of stick margarine. One survey of 85,000 nurses confirmed that those who consumed more of these oxidized trans-fat foods, and especially margarine, had higher rates of both heart disease and cancer.

Margarine was originally touted as a substitute that would taste the same as butter, but reduce the risk for heart attacks and other atherosclerotic complications. However, it now appears that margarine may be just as harmful or even more so. Commercial interests with a lot at stake have waged a huge public campaign to offset these reports and have also been scurrying to develop products that would be less damaging.

Not all of these margarines are the same. Hard stick margarines have undergone more hydrogenation, and contain over twice as much trans-fats as softer products that come in tubs. In general, the easier it is to spread, the lower the trans-fat concentration is likely to be. Margarines that can be sprayed or squeezed on foods or cooking utensils may be the best option. These versions also allow you to make your own blend with olive or other vegetable oils that contain healthier unsaturated fats.

Foods usually list "partially hydrogenated" oil on the label, but many authorities feel that the FDA should also require that trans-fatty acid content should be included. In addition, since trans fat is the largest amount of artificial chemical in our food supply, similar information should be available on the menu items of fast food chains and restaurants serving fried foods and commercially baked products. The National Association of Margarine Manufacturers has indicated it would not oppose such mandatory labeling, claiming that newly developed products are significantly lower in saturated fat as well as trans-fatty acids.

Indeed, there is a new campaign to promote margarine as medicine. The FDA recently approved two brands of margarine, Benecol and Take Control, as cholesterol lowering agents when eaten in sufficient quantities daily. Benecol, which was developed in Finland, has received the most publicity. It has a canola and soybean base and contains cholesterol lowering plant stanol esters derived from pine tree sterols. Take Control has a similar base plus sunflower oil, and lowers cholesterol because of its content of sterols extracted from soy beans. Benecol spread can be used in cooking or baking, but the protective sterols in Take Control break down with heat. Both can lower cholesterol up to 10 percent if you consume 1.5 to two tablespoons every day, at a cost of about \$260.00 a year.

As indicated previously, it is not the total amount of fat but its composition that may be most important. Some, like olive oil, may be very healthy, and certain saturated fats seem to paradoxically protect against heart attacks.

Deciphering The French Paradox

Although the French eat foods that are rich in artery clogging saturated animal fat, heart attacks and deaths due to coronary artery disease are much lower compared to the United States and Great Britain. Numerous explanations have been offered for this "French Paradox", including increased intake of alcohol (particularly red wine), a diet high in fiber, folate, nuts, monounsaturated fats, complex carbohydrates, and much less stressful eating habits. Many feel it is only a matter of time for this protection from coronary heart disease to disappear, and the reasons for this deserve further discussion.

Alcohol And Wine

Alcohol has been shown to increase blood levels of protective high density lipoproteins (HDL cholesterol). Red wine, which is the most popular beverage in France, contains antioxidants and chemicals that decrease platelet stickiness and aggregation, thus decreasing the tendency to clot formation that can contribute to heart attacks and strokes. The French also tend to drink alcohol (especially wine) on a leisurely, moderate and regular basis, usually with meals. There is some suggestion that this consumption pattern provides much more protection from heart attacks, compared to drinking alcohol without eating, or primarily during weekend binges.

Fiber, Folate, And Nuts

Another factor might be that the French consume two or three times more fiber from whole grains and its by-products than do Americans and the British. Cereal fibers in particular have been shown in several studies to have cardioprotective effects.

Low levels of serum folate have also been shown to be linked to an increased risk for heart attack and stroke. Folic acid lowers levels of homocysteine, which acts as an irritant to the inner lining of the arterial wall and accelerates the development of atherosclerotic plaque. The average French diet consists of lots of dark green leafy vegetables containing high concentrations of folate, other protective B complex vitamins, as well as antioxidants.

Increased nut intake can also provide similar benefits, and is believed to contribute to the lower incidence of coronary events in Seventh Day Adventists. The French eat about twice as much nuts as American. The type of fat contained in nuts is of the unsaturated variety that lowers levels of harmful, low density lipoproteins (LDL Cholesterol).

Foie Gras and Fluctuating Blood Sugars

The French also have a much higher consumption of foie gras, which is very high in fat. However, the fats in duck and goose liver are also more likely to reduce coronary artery disease than contribute to it. Their chemical composition is much closer to the cardioprotective compounds found in olive oil, rather than those in butter and lard. The Gascon region of Southwest France has the highest saturated fat intake in the world. Inhabitants slather goose and duck fat onto bread instead of butter, snack on fried duck skin, eat twice as much foie gras as the rest of France and fifty times as much as we do. Yet, 315 out of every 100,000 middle-aged American men die of heart attacks each year, compared to 145 in France, and only 80 in Toulouse and the surrounding Gascon area, despite their higher rates of dietary indiscretion and smoking.

Fluctuations in blood sugar may be another factor. In frequent consumption of foods that quickly elevate blood sugar stimulates a sudden release of insulin to restore normalcy. This in turn triggers an increase in triglycerides; repeated episodes of this can accelerate the appearance of clinical diabetes, which is a risk factor for heart disease. Americans tend to have a diet high in sweets and refined carbohydrates which results in rapid and repeated rises and fluctuations in blood sugar. The French are more apt to eat cheeses and complex carbohydrates like pasta and minimally processed grains, which increase blood sugar much more gradually and do not trigger repeated insulin responses. Some of the chemicals developed during the fermentation process for cheese also tend to bind the type of calcium compounds that are components of arteriosclerotic lesions, so that less is absorbed.

(Continued on Page 6)

The Americanization Of France

Another distinction may be that the French tend to dine in a leisurely fashion with friends and family, as opposed to Americans who eat fast foods on the run and often alone. The French are also more apt to be gourmets than gluttons. Food portion sizes tend to be half to a third of what is put on the plate in the U.S. and there is little snacking in between meals. As a result, only eight percent of the population are classified as being obese compared to 30 percent in the U.S., another risk factor for coronary artery disease.

All of this has started to change dramatically in recent years. An increasing time crunch has resulted in fewer regular, relaxed meals. In addition, over the last 20 years the French diet has become more "Americanized", and more family meals are now coming from pizza parlors and MacDonald's. Children are given soda rather than wine to drink, and overall wine consumption has now dropped to about half of former levels. Snacking has now become quite common, and as might be predicted, the incidence of obesity is rising, and cholesterol levels are starting to approach those in Great Britain.

The French are also exercising less than in the past, as more of them drive to large supermarkets to shop for prepared foods, instead of walking or bicycling to the local grocery or bakery to purchase fresh produce. One very recent study showed that simply walking 20 minutes five days a week could significantly lower the incidence of hypertension and heart attacks in elderly men. In another, men who walked 1.5 miles or more daily were half as likely to have coronary disease compared to controls walking a quarter of a mile or less.

At present, only about one percent of French males die of heart disease before age 50, and it may take several decades for all of the above changes to translate into higher heart attack rates. Many authorities believe that it may be only a matter of time before the French paradox of longevity despite presumed dietary indiscretions becomes a thing of the past. Increased stress may also play a role.

Stress, Cholesterol, And Longevity

Numerous studies suggest that stress can contribute to cardiovascular disease, and several possible mechanisms of action have been proposed. Stress related ventricular fibrillation is a common cause of sudden death, and severe emotional stress can result in myocardial necrosis in the absence of coronary occlusion. Stress also contributes to smoking, hypertension, elevated blood sugar, and cholesterol, the major risk factors for heart attacks.

Stress has a far more powerful influence on cholesterol than dietary fat intake. In one classical study, cholesterol soared in medical students just before final examinations, dropped after the test was over, but quickly jumped up again as the students were told they had failed the exam, even though they had passed. Cholesterol levels in tax accountants rise progressively as April 15 approaches despite any change in diet, and promptly return to normal after the tax season is over. In a more recent study, healthy young men, one of whose parents had suffered a heart attack, showed significantly higher blood cholesterol rises in response to stress than controls with no such family history.

On the other hand, since it does not seem likely that an elevated cholesterol per se causes coronary heart disease, there must be some other connection. Atherosclerosis and almost all of the other consequences of aging appear to be due to free radical damage to cells as we grow older. Stress increases the production of free radicals, as well as homocysteine, which is emerging as a far more important factor in the development of atherosclerotic plaque than cholesterol.

People who are more resistant to stress than others seem to live longer, presumably because they have developed better adaptive and coping skills. But the reverse may also be true, since mutant strains of long lived experimental animals also show much more resistance to toxic stressors. So which comes first? There is not much you can do about heredity, and if cholesterol is not the culprit that causes atherosclerosis and coronary occlusion, then what is responsible?

The Role Of Physical Stress

The conviction that cholesterol causes atherosclerosis is based on findings from the forced feeding of cholesterol to rabbits and other experimental animals. In this model, the first visible evidence of atherosclerosis was the appearance of one or more fatty streaks in the aorta or other arteries. Microscopic examination of these deposits showed scavenger cells that were loaded with cholesterol and other lipids. However the obstructive atherosclerotic plaque seen in humans is quite different. It has more of the appearance of an inflammatory response, and the principal finding is a proliferation and piling up of smooth-muscle cells. Similar lesions can be produced in animals by injury to the inner lining of the arterial wall even when cholesterol and other lipid levels are well within normal limits.

There are several lines of investigation to support the notion that most human atherosclerosis similarly starts as a response to physical trauma rather than some primary chemical process. When polyethylene catheters are inserted into the aortas of experimental animals to obtain diagnostic information, it has been observed that atherosclerotic lesions subsequently start to form wherever the catheter has touched the inner lining of the vessels, rather than in more customary locations. Their microscopic appearance also resembles atherosclerotic plaque in humans, rather than those induced by elevated cholesterol in animals. The rapid restenosis of coronary and other bypass grafts due to occlusive plaque is also believed to result from similar trauma during the operative procedure, and similarly is independent of cholesterol levels. Some people seem to be more susceptible to this than others. One possibility is that the endothelial inner lining of their arteries is more reactive to physical irritation, resulting in the release of chemicals from injured cells that cause increased local spasm or prevent dilatation. If this were true, then drugs designed to dampen this heightened responsiveness might reduce ischemic symptoms due to diminished blood flow. They might also retard the progression of atherosclerosis, and research studies strongly support both of these predictions.

The prevention of vessel spasm and promotion of widening in response to trauma is due to local release of nitric oxide. Repeated insults can rapidly deplete the ability to produce nitric oxide, but this can be restored by the administration of its precursor L-arginine. In animals, a single injection of L-arginine prevents restenosis following balloon angioplasty and dietary supplementation inhibits atherosclerosis due to elevated cholesterol. In humans, L-arginine has been shown to improve symptoms of intermittent claudication due to obstructive disease in the legs. A recent Mayo Clinic study in patients with coronary heart disease reported that administering one pill daily resulted in a marked reduction of symptoms within one week. These benefits persisted throughout the six months of the study, but no improvement was seen in controls taking a sugar pill. In all of these studies, L-arginine was shown to act on the endothelial lining of blood vessels to relax them, and it also prevented platelet clumping, clot formation and the proliferation of smooth muscle cells.

In the absence direct physical trauma, how is the process of atherosclerosis initiated in humans? Atherosclerotic lesions tend to localize at curvatures and branches of the arterial system where the local flow is more rapid, turbulent and irregular. One of the curious and predictable things about atherosclerosis is that it tends to occur in certain anatomical locations, such as the inner sides of the bifurcation of the aorta or the sharper curves of coronary vessels where blood flow is more rapid and turbulent. In other enclosed hydraulic system models with similar curves and Y shaped connections, this results in a greater negative pressure at these locations. In cities subjected to frequent high winds and hurricanes, the palm trees lining an avenue will always be seen to be bending in. Similarly, in the arterial system, negative pressure on the inner endothelial lining of vessels is greatest at such sites, and repetitive insults would result in sufficient injury to initiate the atherosclerotic process.

The Commercialization Of Cholesterol And Contemporary Medicine

The high fat-heart disease hypothesis is one of the worst frauds perpetrated on the American public. That's the opinion of George Mann, a respected researcher who was one of the first to point out the role of trans-fats and homocysteine in accelerating atherosclerosis. None of the numerous clinical trials of low cholesterol diets have shown any benefits, and some which included lipid lowering drugs have been harmful. However, the cholesterol cartel of drug companies, manufacturers of low fat foods, blood testing devices, and others with huge vested financial interests have waged a highly successful promotional campaign. Their power is so great that they have infiltrated medical and governmental regulatory agencies that would normally protect us from such unsubstantiated dogma. They fund many of the research studies reported in medical journals and the media, and conferences offering Continuing Medical Education credits. Practicing physicians also get much of their information and samples from drug company representatives who are obviously biased. Direct TV advertising to consumers, unheard of years ago, puts additional increased pressure on physicians, as do the managed care formularies that tell them what drugs to use based on bottom line concerns. Compared to their peers a half century ago, most doctors don't have the time or skills to critically evaluate reports, very few know anything about research, nor did the generation that taught them. The Center for Health Futures is sponsoring a "Renaissance In Medicine" Colloquium to see what can be done to restore the education, training and authority of physicians to its previous high standards.

What we call "risk factors" for coronary artery disease are merely statistical associations or "markers". About 300 have been identified, but it is clear that many, like a deep earlobe crease or early vertex baldness have no real causal relationship. The possible role of others is supported by interventions showing that their absence or presence in varying degrees does have an effect. These range from family history, cholesterol, chlamydia, cytomegalovirus, homocysteine, diabetes, antioxidants like vitamin E, fiber, folate, red wine and other French paradox components, and aspirin, to psychosocial and emotional factors such as bereavement, social isolation, anxiety, and depression. The mechanisms of action involved for each of these are not known, but are obviously so different, that one must conclude that atherosclerosis is a complex multifactorial process. It appears to start with trauma to the vessel wall that allows other agencies like oxidized lipids, infectious agents, homocysteine and other irritants to promote an inflammatory reaction that results in obstructive plaque.

Theories don't have to be correct, only facts do. Lowering significantly elevated cholesterol levels is obviously beneficial for some patients, but there is growing evidence that this may have more to do with decreasing coagulation tendencies than reducing the deposition of fat in arterial walls. Recent trials with statin drugs do show that they reduce mortality. However, this is not necessarily correlated with cholesterol reduction, and it has now been shown that they reduce platelet clumping and clotting much like aspirin. Saturated fat is hardly healthy, but cholesterol is not the cause of atherosclerosis.

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