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AS WE SEE IT

The Cholesterol Controversy

By William Faloon

As we enter the year 2009, a heated debate continues about the role of cholesterol in the development of atherosclerosis and heart disease. Based on our analysis, both sides still have it wrong!

Almost comical is the role reversal this controversy has taken. When progressive individuals first proposed that high cholesterol increases heart attack risk, the medical establishment ridiculed the idea. The FDA went so far as to make it illegal for food companies to claim that diets low in saturated fat and cholesterol had any relationship to artery disease. (Note: High-*saturated* fat diets cause blood cholesterol to spike.)

The dispute raged for decades until the medical establishment (and the FDA) not only embraced the concept that high cholesterol causes heart attacks, but claimed this scientific area their exclusive domain.¹⁻⁵ Many of today's complementary medicine practitioners, who would have been early proponents of low-*saturated* fat diets, now question the association between cholesterol and vascular disease.

The fact that confusion still exists over this straightforward medical principle helps explain why atherosclerotic disease remains today's leading preventable killer.



William Faloon

EARLY HISTORY OF CHOLESTEROL AND ARTERIAL DISEASE

If we travel back to 1913, we would learn of an impressive study showing that rabbits fed a high-cholesterol diet develop atherosclerotic lesions that closely resemble those seen in humans.⁶ This research was initially criticized because rabbits are plant eaters whose normal diets are not the same as humans. When dogs and rats are fed high-cholesterol diets, they do not develop artery disease. It was later discovered that dogs and rats efficiently convert cholesterol to bile acids that are excreted. When these excretion mechanisms are blocked, however, dogs and rats do develop atherosclerosis in response to cholesterol feeding.

The cholesterol theory gained a bit more credibility when atherosclerotic lesions were readily induced in guinea pigs,^{7,8} goats,⁹ hens, parrots,¹⁰ and even primates¹¹ in response to cholesterol elevation.

Interestingly, the first hints that high cholesterol caused atherosclerosis in humans occurred back in 1889.^{2,12} A doctor reported a case of a child with a rare genetic disorder that caused massive overproduction of cholesterol. The child died suddenly at age 11. An autopsy revealed extensive atherosclerotic-like lesions in large arteries.

It was not until 1939 that a publication in the *Archives of Internal Medicine* pulled together the evidence linking this rare genetic disorder (*familial hyper-cholesterolemia*) to coronary artery disease.^{2,13} It was argued back then, however, that the extraordinarily high cholesterol levels in those with this genetic defect could not be extrapolated to people with only moderate cholesterol elevations.

By 1955, the cholesterol-heart attack connection was attracting some respectability. A study was done that measured cholesterol blood levels in people from seven different countries. Citizens of Finland, who ate the most *saturated fats*, had an average cholesterol reading of over 260 (mg/dL). The Japanese, who consumed the least saturated fat, had average cholesterol readings of a little over 160. Over a 10-year period, the number of fatal heart attacks per 1,000 men was about 70 in Finland and a little less than 5 in Japan. Saturated fat made up 20% of the Finnish diet, but only 2.5% of the Japanese diet.^{2,14}



These findings had a significant impact on the cholesterol debate, but the vast majority of physicians and the federal government still proclaimed that high cholesterol had nothing to do with heart attack risk.

WHERE SOME RESEARCHERS WENT WRONG

In trying to prove that diets high in *saturated fats* increase cholesterol levels and subsequent heart attack risk, scientists conducted studies that substituted omega-6-rich *polyunsaturated fats* (corn oil, safflower oil) in place of *saturated fats* (butter, lard, meat fats).¹⁵⁻¹⁷ While these studies showed reduced cholesterol levels and heart attack rates, the findings were not nearly as impressive as they could have been. This is because these kinds of *polyunsaturated fats* (corn oil, sunflower oil, safflower oil, etc.) rich in omega-6 fatty acids create inflammatory byproducts and induce LDL oxidation that damages arteries.

During this era of medical ignorance (the 1950s-1960s), the benefits of **monounsaturated fats** (such as those found in olive oil) and **omega-3 fats** (such as those found in cold-water fish, flaxseed, and walnut oils) in *preventing* heart attacks were not recognized. The best that doctors could provide in lieu of dangerous *saturated fats* was to substitute omega-6 polyunsaturated fats, which then created a dangerous proinflammatory state (due to an imbalance in omega-6 to omega-3 fats).¹⁸

THE 1960S... MORE LOST OPPORTUNITIES

I'll never forget going to my first funeral and seeing a 42-year-old neighbor lying in a coffin. He had died of a sudden heart attack, but no one talked about how unusual his early death was. That's because men over age 40 were routinely dying of heart attacks in those days.

Heart attacks and strokes claimed astronomical numbers of lives in the Western world throughout most of the past century. During the early 1960s, for example, the number of acute fatal heart attacks in younger men was substantially higher than today.¹⁹⁻²¹ A lot of this had to do with cigarette smoking and the high-saturated fat diet that was a staple in most households.

By the early 1960s, however, the theory that one could reduce their heart attack risk by avoiding *saturated fats* had taken hold at least in the alternative medicine community. Even the *American Heart Association* went on record as early as 1961 with the following closely guarded statement:

“Those people who have had one or more atherosclerotic heart attacks or strokes may reduce the possibility of recurrences by such a change in diet.”^{2,22}

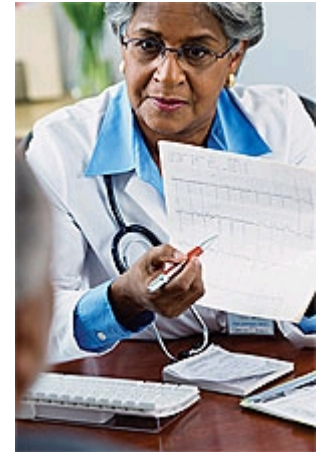
Based on a wave of studies showing reductions in heart attack incidence in those who reduced their *saturated fat* intake, nutritionists and alternative doctors were finally espousing heart-healthy diets. The FDA responded by criminalizing the commercial dissemination of this information by the food industry. Heart attacks claimed about seven million American lives in the 1960s.²⁰ The FDA's suppression of the link between poor diet and heart attack risk kept funeral businesses thriving.

THE GREAT CHOLESTEROL DEBATES

In 1969, an *American Heart Association* spokesperson stated that those with excess cholesterol levels should be treated medically. This doctor went on to state, **“in short, we have come ... to the point where we are probably preventing a disease that was considered to be an inevitable accompaniment of aging not very long ago.”²⁶**

The *American Heart Association* met with fierce criticism from prominent cardiologists who continued to proclaim there was no evidence that cholesterol reduction conferred protection against heart attacks. These attacks on the cholesterol theory of heart disease were published in the leading medical journals of the day.

Regrettably, it was not until 1984 that the medical establishment formally recognized the relationship between high cholesterol and heart attack incidence.²⁷



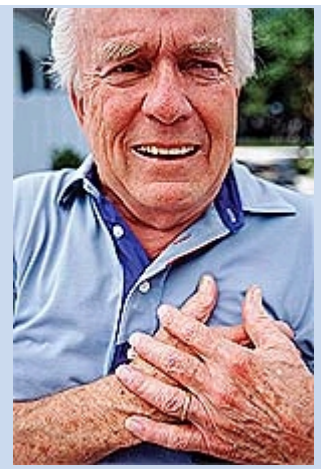
THE FIRST DIET-HEART COOKBOOK

Readers of Life Extension magazine may recall the name **John Gofman, MD, PhD**. This is the physicist turned medical doctor whose early work on radioactive isotopes resulted in him being recruited to work on The Manhattan Project to develop the first atomic bomb.

Dr. Gofman's expertise on the biological effects of radiation caused him to later take a very controversial position. He meticulously documented how medical diagnostic X-rays are a cause of cancer and artery disease, something most in the medical establishment still refuse to accept.

In **1947**, Dr. Gofman began research that would soon lead him to conclude that cholesterol is a cause of atherosclerosis.^{23,24} Dr. Gofman and his colleagues were the first to show that specific fractions of cholesterol such as LDL (low-density lipoprotein) are the most dangerous. Dr. Gofman was involved in the publication of possibly the first book in **1951** about how low-fat and low-cholesterol diets prevent heart disease.²⁵

I am always amazed at individuals who are able to contribute so much. In Dr. Gofman's case, he was instrumental in harnessing nuclear energy, warning of the dangers of low-level radiation, and then moved on to a completely different field to discover specific fractions of cholesterol that cause atherosclerosis.



NATHAN PRITIKIN... AN EARLY HERO OF MINE

I will never forget as a teenager seeing a televised debate between *Nathan Pritikin* and a mainstream cardiologist. Pritikin explained how very low-fat diets could reverse coronary atherosclerosis, whereas the cardiologist ridiculed the notion.

I knew little about heart attacks back then, other than the fact that my family members and neighbors were having them on a regular basis. I also witnessed the poor diets these heart attack victims ate, which made Nathan Pritikin's arguments all the more convincing to me.

What was so remarkable about Nathan Pritikin was that he had no medical training. In 1957, at age 40, Pritikin was diagnosed as having heart disease. Faced with a lifetime of ever-increasing disability, he pored over the scientific literature and formulated a diet and exercise program to treat his disease.²⁸ After nine years of trial and error, he had cured himself.



Long before the medical establishment acknowledged that something as simple as diet might be causing serious illnesses, Pritikin had created a scientifically sound program using food and exercise as medicine. This revolutionary departure from the flawed theories of the 1950s caused him to become a public enemy of the medical establishment. Nathan Pritikin's healthy diet program did more than reverse heart disease. Patients who came to his clinic often saw their type 2 diabetes, arthritis, and hypertension disappear.^{29,30}

Despite these clinical successes, Nathan Pritikin was ceaselessly attacked by doctors as being a charlatan. For much of the 1970s, Nathan Pritikin waged a public battle with government and private health agencies, as well as with the American Medical Association. The medical establishment doggedly refused to accept that what one ate had anything to do with their risk of heart disease.

Pritikin was so confident that he was *reversing* heart disease with healthy diets that he ordered his own body autopsied after his death. Almost 30 years after being diagnosed with *irreversible* coronary artery disease, the autopsy showed his arteries were akin to those of a young man and clear of any signs of heart disease.³¹

In 1987, two years after Nathan Pritikin's death, the *Journal of the American Medical Association* announced a study that showed regression of atherosclerosis in the coronary arteries of humans who reduced their blood cholesterol by a similar degree as were accomplished at the *Pritikin Longevity Centers*.³² Numerous subsequent studies confirmed that Nathan Pritikin was scientifically correct...and the medical establishment's position fatally flawed.³³⁻³⁶

Tens of millions of Americans needlessly perished because the role of cholesterol in causing heart disease was not recognized nearly as early as it should have been.

WHAT IS CHOLESTEROL?

Cholesterol is a lipid (fat) that is chemically classified as a *sterol*. It provides critically important functions in the body such as building and maintaining cell *membranes*. Cholesterol also functions as a precursor to hormones like *testosterone* and fat-soluble vitamins.

While cholesterol is essential to life, the *lipoprotein* it is bound to plays a role in whether it injures or protects the arterial wall. Since cholesterol is insoluble in blood, it is transported in the circulatory system by *lipoproteins*.

LDL (low-density lipoprotein) transports cholesterol to the cells, whereas HDL (high-density lipoprotein) transports cholesterol away from the cells.

When one has excess LDL, too much cholesterol can be deposited into the arterial wall. Insufficient HDL, on the other hand, impairs cholesterol transport away from the arterial wall (for disposal in the liver). Too much LDL and/or not enough HDL can thus set the stage for *atherosclerosis*. These simple facts, however, explain only part of the problem.



POMEGRANATE SUPPRESSES LDL OXIDATION

LDL Atherosclerosis Risk Factor	Effect of Pomegranate ⁶¹
LDL basal oxidative state	Reduced by 90%
LDL susceptibility to copper-induced oxidation	Reduced by 59%
Paraoxonase-1 (protects against LDL oxidation)	Improved by 83%
Total antioxidant status	Improved by 130%

DANGER OF OXIDIZED LDL

The over-promotion of “statin” drugs has resulted in today’s cardiologists focusing on getting their patients’ LDL and total cholesterol down as low as possible. Pharmaceutical company advertising has made it appear as if the only cause of atherosclerosis is excess LDL and cholesterol.

Beginning in 1979, however, researchers made discoveries indicating that it is the oxidation of LDL that results in the most arterial damage.³⁷⁻³⁹ Thousands of studies now reveal how oxidized LDL contributes to the atherosclerotic process from start to finish.

There are doctors who argue that atherosclerosis is all about *inflammation* and response to endothelial injury and has nothing to do with LDL cholesterol. What these doctors overlook is the fact that oxidized LDL injures endothelial cells and causes inflammation!⁴⁰⁻⁴⁶

Oxidized LDL causes endothelial cells to secrete “adhesion molecules” that allow white blood cells to penetrate the inner lining of the artery (the endothelium). This is where initial fatty streaks and atherosclerotic plaques develop.⁴⁷

Oxidized LDL turns on white blood cell gene expression that enables them to convert into foam cells, which results in continuous accumulation of oxidized LDL in the atherosclerotic plaque.⁴⁸

Oxidized LDL initiates an inflammatory process by causing foam cells to secrete molecules that attract proinflammatory cells.⁴⁷

Oxidized LDL enhances the process whereby immune cells, foam cells, smooth muscle cells, and endothelial cells degrade collagen, which leads to the rupture of the fibrous plaque.⁴⁹

The endothelium requires *nitric oxide* to function properly. A hallmark characteristic of *endothelial dysfunction* is a lack of nitric oxide. Oxidized LDL impairs the endothelial cells’ ability to produce nitric oxide.⁵⁰

As you may surmise by now, both absolute LDL level and LDL oxidation are involved in atherosclerotic processes and heart attack risk.



CORONARY RISK FOLLOWING TREATMENT WITH STATIN DRUGS ACCORDING TO ACHIEVED LDL LEVELS

Achieved LDL Level (mg/dL of blood)	Under 54	54-71	72-94	Over 94
	Lowest Risk	10%	30%	80%
		Greater Risk	Greater Risk	Greater Risk

The data above is a tabulation of the results from patients who had already suffered a coronary event who were then prescribed either 80 mg of Lipitor® (atorvastatin) or 40 mg of Zocor® (simvastatin) per day.

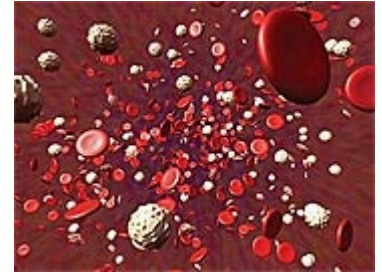
These findings show that the achieved level of LDL was strongly predictive of recurring coronary events. These relative risks are adjusted for age, smoking status, diabetes, hypertension, and body mass index (BMI).⁶⁴

COMMON SENSE APPROACHES TO HEART ATTACK PREVENTION

Financial bias, apathy, and scientific ignorance have resulted in most Americans failing to protect themselves against today's leadingcrippler and killer... atherosclerosis.

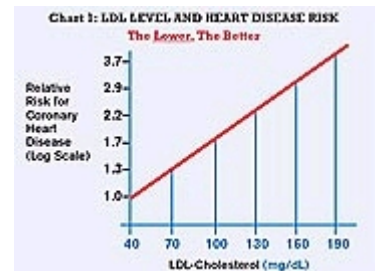
Some cardiologists erroneously believe that if all their patients took a statin drug and aspirin, coronary artery disease would disappear. Equally disturbing are doctors who claim that that aging people should not worry about their LDL levels.

For 29 years, we at *Life Extension* have emphasized that atherosclerosis has many underlying causes.⁵¹ Our findings have been validated in many subsequently published studies.⁵²⁻⁵⁴ At the end of this article, we have reprinted an updated version showing 17 daggers aimed at the heart, each dagger representing an *independent* risk factor for developing coronary artery disease. Fortunately, most *Life Extension* members are already taking nutrients, hormones, and sometimes drugs to protect against every one of these cardiac risk factors.



When it comes to inhibiting LDL oxidation, members should find comfort in knowing they have been taking supplements that have been confirmed to dramatically inhibit LDL oxidation. A number of studies document the ability of *ubiquinol* CoQ10 to protect against LDL oxidation better than lycopene, alpha tocopherol, and other lipid-soluble antioxidants.⁵⁵⁻⁵⁹ Some of these studies show that **alpha tocopherol** (vitamin E) can turn into an LDL pro-oxidant unless ubiquinol is also present.⁶⁰ These studies help explain the inability of the alpha form of vitamin E by itself to significantly reduce heart attack rates in certain populations.

Perhaps no other nutrient has demonstrated better *anti-LDL oxidation* effects than pomegranate. In a clinical study, human subjects taking pomegranate showed a beneficial 35% reduction in carotid intima-media thickness accompanied by a 45% improvement in carotid blood flow. As evidenced by the chart above, pomegranate improved markers related to LDL oxidation by up to 130%.⁶¹



These kinds of impressive study results, showing how LDL oxidation can be suppressed, might tempt some people to ignore dangerously high LDL blood levels. We at *Life Extension* strongly advise against this. The chart below vividly shows the sharp increase in **coronary artery disease** risk as LDL concentrations increase in the blood.⁶²

Relative risk for coronary heart disease based on level of low-density lipoprotein (LDL).⁶² As LDL rises, so does the risk of heart attack.

KEEP YOUR LDL LEVELS BELOW 100

Atherosclerosis remains the leading cause of death in the Western world.⁶³ Eastern populations who are switching to high-fat Western diets are seeing vascular disease rates spiral upwards. We cannot ignore almost 100 years of research showing that excess LDL-bound cholesterol is a coronary risk factor.

While there may someday be a definitive finding that something as simple as pomegranate provides complete protection against LDL oxidation, and therefore excess LDL itself, we don't have these data confirmed today.

You have entrusted us to provide you with an accurate analysis of the available scientific literature to keep you alive in good health. We therefore reiterate our 29-year recommendation that healthy members keep their LDL levels below 100 mg/dL.

Very high-risk groups (e.g., smokers or those with diabetes, abdominal obesity, a recently sustained heart attack, low HDL, high triglycerides, or known coronary artery disease) should strive for a 70-75 mg/dL LDL target level. (Note: Those who drive LDL

down to these very low levels should use blood tests to make sure they are not also suppressing critical hormones like testosterone and DHEA.)

The chart on top of this page clearly demonstrates that in people who have already suffered a coronary event, each progressive LDL elevation increases the risk of a second event. In this study, cardiac disease incidence was 80% greater in those with an LDL reading over 94 mg/dL compared with those whose LDL was under 54 mg/dL.⁶⁴

SEE HOW OUR PRESIDENTS DIED IN THE PAST CENTURY

To better understand the epidemic of heart disease that existed during the era of the cholesterol debates, look no further than the medical histories of the presidents of the United States.

President **Dwight Eisenhower** suffered his first heart attack in 1955. That same year, Senator **Lyndon Johnson** suffered his first heart attack. Back in those days, recovery from a heart attack was a slow and arduous process.

Dwight Eisenhower smoked four packs a day of cigarettes until he quit in 1949. Combined with his high-saturated fat intake, he was at great cardiac risk. Here is what President Eisenhower ate the day of his first heart attack:⁷⁷

- Breakfast: sausage, bacon, mush, hotcakes
- Lunch: hamburger with raw onion
- Dinner: roast lamb

In 1957, President Eisenhower suffered a stroke. By the time of this death in 1969, Eisenhower had suffered at least seven heart attacks, along with multiple other diseases that can be related to the unhealthy nature of the typical American diet of his era.⁷⁷

Three years later, former President **Harry Truman** died at age 88 from atherosclerotic coronary artery disease.

Lyndon Johnson was only 46 years old when he suffered his first coronary occlusion heart attack. He too had been a heavy smoker, but quit after his first heart attack. Shortly after leaving the presidency, Lyndon Johnson resumed cigarette smoking and continued eating foods that we know today damage arterial linings (the endothelium). Johnson developed severe angina pain that crippled him until he suffered his final heart attack in 1973 at age ^{65,78}

So while the FDA and the medical establishment were ridiculing the notion that high-saturated fat diets caused artery disease, the most famous political leaders of the day were keeling over from heart attacks right before the public's eyes.

Heart attack and/or stroke claimed the lives of most of the presidents in the past century including Theodore Roosevelt, William Taft, Woodrow Wilson, Calvin Coolidge, Franklin Roosevelt, and Richard Nixon.

TYING IT ALL TOGETHER

In reviewing the history of dietary fats and heart disease risk, a number of interesting facts emerge. Nathan Pritikin put a lot of the pieces together when he mandated that virtually all dietary fat should be eliminated (less than 10% total calories from fat). This protected his followers against the atherogenic effects of both saturated fat and dangerous omega-6-rich *polyunsaturated* fat sources like corn, soybean, safflower, and sunflower oils. The problem is that adhering to Pritikin's very strict diet is difficult for the vast majority of people.

Fortunately, we know today that following a low-saturated fat, *Mediterranean*-type diet with lots of natural polyphenol antioxidants provides huge cardioprotective benefits.⁶⁵ We have also acquired the knowledge that specific fats (omega-3s and certain monounsaturated fats) are extremely beneficial in reducing vascular disease risk.^{66,67}

As we report in this issue of *Life Extension*, a large number of studies continue to validate the ability of low-cost plant polyphenols to not only protect against LDL oxidation, but to also boost beneficial HDL and lower absolute LDL levels in the blood.⁶⁸⁻⁷⁶

It may thus be possible for many aging humans to achieve optimal blood lipid status using an integrative approach to support healthy cholesterol levels and reduce oxidant stress... without resorting to prescription drugs.

NATURAL PROTECTION AT THE LOWEST PRICES

For 21 consecutive years, *Life Extension* members have taken advantage of the annual **Super Sale** to stock up on a large supply of their favorite nutrient formulations.

During this annual winter event, every **Life Extension product** is discounted so that members can obtain our most up-to-date formulas at the lowest prices of the year.

We recently upgraded our most popular products to provide even more life-protecting nutrients. Until February 2, 2009, members can obtain extra discounts on every one of these formulas during our once-a-year Super Sale.



For longer life,



William Faloon

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