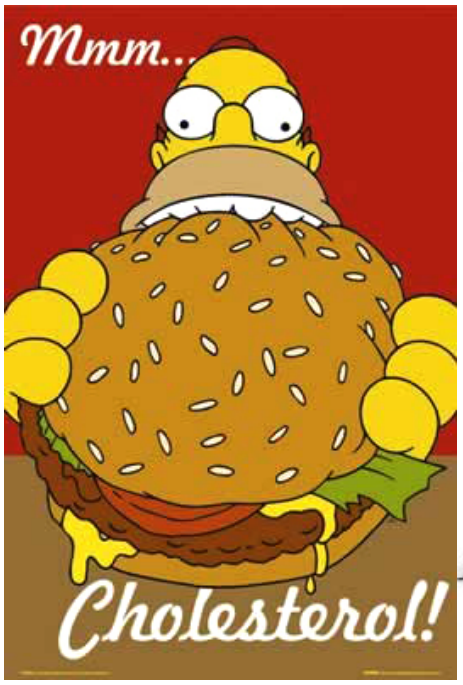
CHOLESTEROL AND THE BIG FAT LIE



"Let's do the good cholesterol, bad cholesterol bit."

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Books:

The Great Cholesterol Con **by Anthony Colpo**
The Cholesterol Myths: Exposing the Fallacy that Saturated Fat and Cholesterol Cause Heart Disease **by Dr. Uffe Ravnskov, MD, PhD**
The Truth About Saturated Fat **by Mary Enig and Sally Fallon**
The Heart Revolution **by Dr. Kilmer S. McCully, MD**

Articles:

Websites:

Cholesterol and Health Website
http://www.cholesterol-and-health.com

Audio/Video:

Cholesterol and Heart Disease
http://youtu.be/i8SSCNaaDcE
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Integral Nutrition:

Cholesterol Does Not Cause Heart Disease, Inflammation Does

Conventional:

Cholesterol Causes Heart Disease: Take Our Cholesterol-Lowering Drugs!!

Terms:

Statin Drugs

WHY IS CHOLESTEROL NEEDED?

Source: ***http://www.livestrong.com/article/28294-cholesterol-needed/***

Cholesterol is a necessary substance for the synthesis of some hormones in the body. It is also an essential element in cell membranes. This waxy substance is found in the bloodstream and in *every individual cell of the body*.

Hormone Synthesis

The body cannot synthesize hormones such as progesterone, estrogen, testosterone or cortisol, without a sufficient amount of cholesterol being present.

Semi-Permeable Membranes

The membranes of human cells allow some substances to pass back and forth. Cholesterol is necessary for this semi-permeable ability--letting nutrients in and wastes out.

Vitamin D Conversion

Vitamin D is manufactured when sunlight hits the skin. This can only occur if the proper amount of cholesterol is present.

Bile Salts

Bile salts are derived from cholesterol and play an essential role in the absorption of fats in the intestines by dissolving fat droplets.

Cholesterol Sources

There are two sources of cholesterol: the body and food. The American Heart Association states that about 75 percent of blood cholesterol is made by the body, with only 25 percent determined by diet.

Liver

The human liver is responsible for producing the cholesterol that is manufactured in the body. It can also eliminate some of the excess cholesterol caused by diet.

CHOLESTEROL IS THE PRECURSOR TO ALL STEROID HORMONES

Source: by Chris Masterjohn

http://www.cholesterol-and-health.com/Steroid-Hormones.html

Synthesis of Steroid Hormones From Cholesterol

Cholesterol is the precursor to all steroid hormones, including:

- **Glucocorticoids (blood sugar regulation)**
- **Mineralcorticoids (mineral balance and blood pressure regulation)**
- **Sex Hormones (many functions)**

Cholesterol is the precursor to a hormone called pregnenolone, which has important functions itself, but is also the precursor to all other steroid hormones.

Pregnenolone is converted to progesterone, a sex hormone, which in turn is converted into cortisol, which regulates inflammation and blood sugar, aldosterone, which regulates mineral balance and blood pressure, or testosterone, a type of sex hormone referred to as an androgen, which regulates libido, muscle mass, and plays other roles.

In females, and to a lesser degree in males, testosterone is further modified, undergoing conversion to estradiol, a different type of sex hormone called an estrogen.

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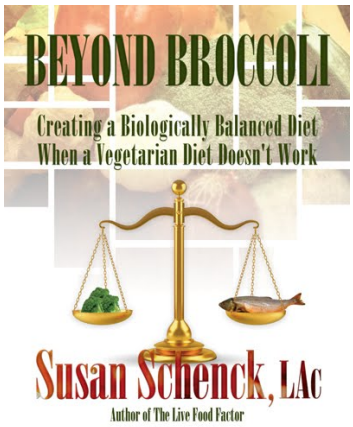
FALLING FOR THE BIG FAT LIE

Source: *Beyond Broccoli* by Susan Schenck, L.Ac. Chapter 11



For years, "heart-attack food" was synonymous with red meat, eggs, and butter. The evidence is in. That is utterly and completely wrong. Fast-acting, concentrated carbohydrate is the ultimate heart-attack food, particularly for those with a sedentary lifestyle.

—Richard K. Bernstein, MD, *The Diabetes Diet*

As early as the 1800s, long before the Atkins and Zone diets, doctors told people who wanted to lose weight that they had to reduce starches and sugary foods and replace them with protein foods, such as meat, dairy, and eggs instead. Conventional wisdom had it that bread, doughnuts, cakes, grains, and potatoes would fatten you up, as well as keep you hungry, while proteins and good fats (there weren't as many bad fats around until the 20th century) kept you full and satisfied.

So how did the fat-as-villain myth begin? How did this deadly lie go mainstream, now proven to be so blatantly false, not to mention contrary to the way man has eaten for millions of years? Investigative journalist Gary Taubes spent five years researching that question, and the extensive results are in his book Good Calories, Bad Calories, which reads like a political science text on the politics of nutrition.

It began with Ancel Keys, PhD, a University of Minnesota physiologist, who came up with the theory that dietary fat raises cholesterol levels and gives you heart disease. We now know that dietary fat influences only 15 to 20 percent of the cholesterol levels, and high blood cholesterol is not really a cause of heart disease.244

Furthermore, if you go on a low-cholesterol diet, your liver, which makes about 80 percent of your cholesterol, will simply compensate by making more. In other words, if you don't eat much cholesterol, your liver makes up for much of the difference. If you eat a lot of cholesterol, your liver backs off.

Dr. Keys made the cover of Time magazine in 1961 with his hypothesis and prompted the American Heart Association to publish its first official endorsement of a low-fat, low-cholesterol diet as a means to prevent heart disease. Saturated fat is higher in cholesterol, and most saturated fat comes from animal products: meat, dairy, and eggs.

Thus began the myth that animal fats, which our ancestors lived on for 2.6 million years, are bad for you. This was merely a hypothesis, mind you, not a proven fact.

Whenever an epidemiological study contradicted this theory, it was called a "paradox." For example, when the French had a low rate of heart disease yet indulged in diets rich in saturated fats, it was the "French paradox." **When the Italians of Rosetto ate copious amounts of animal fats and even cooked with lard, yet had a strikingly low percentage of deaths from heart disease**, this was thought to be another exception.

The Masai of Africa lived on very little besides milk, blood, and beef yet were free of heart disease and had low cholesterol levels. Eskimos eating their traditional diets, which often consisted of 100 percent animal meat for long stretches of time, were so healthy that they had no medicine men.

There is even a study that demonstrates an American paradox.

> ***These effects include the paradox that a high-fat, highly saturated-fat diet is associated with diminished coronary artery disease progression in women with the metabolic syndrome, a condition that is epidemic in the United States.245***

There were so many more paradoxes — the Greek Paradox, the East African Paradox, the Swiss Paradox, the Pacific Island Paradox, the Japanese Paradox — that Dr. Malcolm Kendrick, MD, author of *The Great Cholesterol Con*, queried, "How many paradoxes do you need before the only paradox left is the diet-heart hypothesis itself?"

Yet Dr. Keys' theory received a tremendous amount of publicity because there were profits to be made from it. Interestingly, Keys himself recanted the theory decades later, stating, "There's no connection whatsoever between cholesterol in food and cholesterol in the blood. None. And we've known that all along" (Eating Well, March/April 1997).

He also admitted that his lipid hypothesis, originally based on rabbits, herbivores by nature, applied only to rabbits. His retraction came after a man who ate 20 to 30 eggs a day for 15 years was found to have healthy plasma cholesterol! 246

Still, the lipid hypothesis grew because it was such a cash cow for the vegetable oil and other food-processing companies, as well as for the pharmaceutical industry, the main beneficiaries.

This theory became instilled into mainstream consciousness in 1977 after Senator George McGovern announced the publication of the first *Dietary Goals for the United States*. Investigator Gary Taubes takes us behind the scenes, to show us that this dubious theory was decided by academic and corporate politics, not real science. Yes, you read that right. Government health guidelines — a dietary disaster for so many of us! — were basically the result of some closed-door wheeling and dealing from behind the scenes.

It turns out that this established "wisdom" was determined by a handful of men in a good ol' boys' club of academia, none of whom even worked with obese patients! These men included Jean Mayer, Fred Stare, Jules Hirsch, George Bray, Theodore Van Itallie, Albert Stunkard, George Cahill, and Philip White.

Meanwhile, Dr. Robert Atkins, MD, was writing about his tremendous results with patients who were able to shed the shackles of obesity without getting hungry, feeling deprived, or even "going low calorie." One of the good ol' boys, Van Itallie, had a personal grudge against Atkins and wrote that his work "lacked scientific merit" — despite the endocrinological science behind the fact that insulin, stimulated on high-carb diets, causes the body to store fat.

Don't get me wrong. I am not an Atkins Diet fan! That diet is usually too high in protein and mostly cooked, but Atkins was a modern-day popularizer of the need to lower insulin levels.

Other MDs — William Banting, Alfred Pennington, and Herman Taller, author of *Calories Don't Count* — had likewise been getting astounding results in weight loss with their obese patients by using carbohydrate-restricted diets.

The late Dr. Frederick Stare, PhD, former head of Harvard University's nutrition department, began his career with articles warning about the nutritional deficiencies created by diets that included white flour. He even wrote about a study that found that diets high in vegetable oils correlated with heart disease.

But after becoming the department head, he sold out to the food industries in exchange for grants. His articles soon assured the public that white flour and processed foods were harmless. He even advocated eating refined sugar!

Stare and his colleagues at Harvard ensured that anyone who claimed that carbohydrates were uniquely fattening would be labeled a quack. Stare and colleagues White and Mayer publicly condemned Taller's low-carb book.

When did they denounce it? They badmouthed the low-carb diet one year after Havard University's nutrition department broke ground on a new $5 million building, a lot of money back in the 1970s. Where did most of the donations for the building come from? As you might guess, the lead "gift" came from a corporation vested in carbs, General Food Corporation, maker of very carb-rich cereals.

Over the next decade, Harvard's nutrition department continued to receive funding from the sugar industry. Stare "coincidentally" became a staunch advocate of sugar and carbs.

So there you have it folks. Our health was placed on the sacrificial altar of corporate profits. Our health was sold when the lie was told. Harvard University professors profited from the lie.

The food companies were delighted with the lie because calorie for calorie, starches and sugars are the cheapest nutrients for them to produce. They yield the highest profit.

Grains can be stored, so having longer shelf lives makes them cheap and abundant. This is why grains have been called "the staff of life" — because they can be stored and used during times of famine.

Finally, the public was happy with the lie because carbs taste sweet and are like narcotics to the brain. Most people prefer pizza, crackers, doughnuts, cakes, pretzels, chips, and cookies (even fat-free ones) to yogurt, eggs, and meat. People get addicted to carbs, not protein.

Grain Merchants behind the Scenes

Who else may have been behind this great low-fat conspiracy to lead the public to purchase more grains and vegetable oils? According to researcher Dan Morgan, author of Merchants of Grain: The Power and Profits of the Five Giant Companies at the Center of the World's Food Supply, there are seven secretive families that monopolize the world's grain supplies.

According to Morgan, since these grain companies had been privately owned for centuries and mostly still are, there were no public stockholders and therefore no financial statements that had to be openly published, despite the fact that they received billions of dollars in government subsidies — at least those operating in the US.

These grain companies make money whether grain prices rise or fall. Farmers and commodities speculators alone shoulder the risks of bad weather, changes in governmental policies, and falling prices. (Since Morgan's book was written, at least one of these companies has gone public.)

These seven reclusive families own or control the five big grain companies: the Fribourgs at Continental Grain Company (New York City, US); the Hirsches and Borns at Bunge (no nationality); the Cargills and MacMillans at Cargill, Inc. (Minneapolis, US); and the Louis-Dreyfuses (Paris, France) and Andres (Switzerland) at the companies with those names.

Few people even know the names of most of these companies and fewer still have ever heard of the oligopolists who own them. Their families like to keep it that way.

They are likely behind the entire low-fat craze, since they are the ones who profit most. They are likely behind the soy craze, since soybeans are one of the farm crops they monopolize.
No doubt they are behind the "eat grains for fiber" propaganda (now proven bogus), as well as the food pyramid, which suggests we eat 6 to 11 servings of grain a day! Grains — generally the worst food for humans — are at the base of the food pyramid.

The Great Cholesterol Con

Heart attacks and heart disease used to be very rare, even though people indulged in pork lard, beef tallow, eggs, and steaks. When Dr. Paul Dudley White, personal physician to President Eisenhower and lead author of a text on coronary disease, decided to specialize in coronary heart disease, he was recommended to find a more profitable specialty, because heart problems were so rare.

By the 1950s and 1960s, heart disease became the leading killer. What had changed in just a few decades? Factory farming practices had begun in the 1920s, degrading the quality of our meats and resulting in damaged arteries a few decades later. Animals were fed **grains** to fatten them up, for example, which skewed their omega-6:3 ratios. Also to blame was the widespread dietary replacement of animal fats with **toxic, rancid vegetable oils** high in omega-6 fats. Worthy of note as well was the **increase in carb consumption, especially refined sugar and other highly processed foods.**

Yet the simplistic dietary fat theory was promoted as the chief culprit.

Despite a huge brainwashing of the masses to eat less saturated fat and lower their cholesterol levels, heart disease only increased. A reduced death rate has resulted from improved emergency care, but progression of the disease itself is relentless.

Despite masses of people giving up smoking and taking up exercise, heart disease continues. The ulterior motive here from corporations? To sell the food companies' vegetable oils, to sell the high-carb foods, and especially to sell the drugs that lower cholesterol.

Dr. George V. Mann, MD, a Johns Hopkins-educated biochemist and physician who was the associate director in the Framingham study of cholesterol, later studied and wrote about the Masai cattle-herding people in Africa. Their unique diet was nearly all meat, blood, and fatty milk. During some periods of their lives, that was all they ate. He found these people to be completely free of heart disease.

Although there was significant atherosclerosis, raised plaques were rare, and cholesterol levels were very low, as his study states.

> *The intake of animal fat exceeds that of American men. Measurements of the aorta showed extensive atherosclerosis with lipid infiltration and fibrous changes but very few complicated lesions.247*

He also found that the Pygmies living in the African rain forest had low levels of cholesterol despite eating large amounts of meat.248

Dr. Mann called the lipid hypothesis the greatest scam in the history of medicine.

> *When we find the real cause and prevention of the cholesterol problem, it will seem to many that there was an unwholesome conspiracy. 249*

In an editorial published in the New England Journal of Medicine in 1977, Dr. Mann *called the cholesterol theory of heart disease "the greatest scam in the history of medicine."

A world-renowned heart surgeon from Houston, Dr. Michael DeBakey, MD, devoted extensive research to the cholesterol theory of heart disease. He found that out of every ten people in the US who have atherosclerotic heart disease, only three or four (30 to 40 percent) had high cholesterol levels. That 30 to 40 percent is the same percentage of the general population that has elevated cholesterol!

In other words, 30 to 40 percent of people with heart disease have high cholesterol levels, but so do 30 to 40 percent of healthy people with no risk of heart attack. A study he undertook proved that there was no correlation between heart disease and high cholesterol.250

Other studies have found such correlations, but not a simplistic cause-effect one. There are other more important factors.

Author Michael Murray, ND, calls the fixation on cholesterol by the drug companies and doctors "one of the greatest medical injustices of all time."

Half the people who die of a heart attack or a stroke have low to normal cholesterol levels. How do the drug companies and the government respond to this fact? They simply recommend making the suggested target cholesterol levels even lower, thereby effectively casting an even wider net for potential customers. . .

Six of the nine expert members of the government panel that drafted the new cholesterol guidelines had either received grants from, or were paid consulting or speakers' fees by, the companies that make some of the most popular statin drugs. . .

These new guidelines should dramatically increase the number of patients on statin drugs. Keep in mind that statins are already the biggest moneymakers in the drug industry.251

Cholesterol Is Absolutely Necessary!

Cholesterol is one of the most important substances in our bodies. Without it you would die. Cholesterol gives our cell membranes their necessary stiffness and stability. Without it, you would "melt down like the Wicked Witch in *The Wizard of Oz*," as Drs. Michael and Mary Eades say.

Cholesterol is used to make our hormones, including estrogen, testosterone, progesterone, cortisol, aldosterone, and DHEA. Cholesterol is a precursor to corticosteroids, hormones used to deal with stress.

It is a precursor to vitamin D, which is needed for healthy bones. Sunlight on the skin interacts with cholesterol to produce vitamin D. If your cholesterol levels are too low, you risk the degenerative diseases that come with vitamin D deficiencies.

Cholesterol is needed in maintaining the health of the intestinal wall, which is replaced every four or five days. It prevents leaky gut syndrome and other intestinal disorders.

Cholesterol is needed to repair wear and tear on the skeleton and muscles, repair injured tissues, and renew hair, skin, and nails.

The brain is rich in cholesterol and needs cholesterol for serotonin receptors to function. Without sufficient cholesterol, people and animals become depressed. Low levels of cholesterol are linked to depression, suicide, homicide, and violence, because a serotonin deficiency is often behind all these conditions. **Low cholesterol levels over time cause the brain to shrink and may eventually result in Alzheimer's disease.**

Depression, Suicide, Violence, and Homicide Worse with Low Cholesterol

Lowering serum cholesterol levels by diet or drugs has been shown in numerous studies to increase the rate of death by suicide or violence.252

Nutritionist Nathan Pritikin was among those who popularized low-fat diets. After many years on his diet, while still relatively young in his early 60s, he was diagnosed with cancer and depression. This led to his committing suicide. **Cancer, depression, and suicide are all now well-researched and proven consequences of insufficient cholesterol.**

My own life has been a continuous experiment in diets. When I look back at my deepest, darkest night of the soul, the period during which I was severely depressed, it coincides with the late '80s and early '90s when I was on a low-fat diet! (Two of those years I was also a cooked food vegetarian.) I was dysfunctional in both relationships and my professional life.

I had been duped like everyone else into eating a lot of grains and fat-free products, such as no-fat salad dressings and fat-free cheeses. Like everyone else, I threw out the egg yolks and ate the whites only. So depressed was I that I took Prozac, which made me gain 40 pounds within six months!

I snapped out of this depression only when I moved on to the Atkins Diet and later the Zone Diet. Even then, I was not eating healthful raw fats, but at least I could dump the Prozac. I became hap.pier on a raw diet full of good plant fats, but I am now happier than ever since I added fish oils, raw egg yolks, and cod liver oil with its DHA and EPA.

Saturated Fats Are Critical for Health

We know that *overdoing* saturated fats can lead to strokes, Alzheimer's disease, and even diabetes, but completely eliminating saturated fats from the diet can lead to other problems in those people who are unable to synthesize enough in their bodies.

Saturated fats have also been maligned by the entire low-fat industry. They have been blamed for clogging arteries when in reality ***only 26 percent of the fat clogging the arteries is saturated. Most, 74 percent, is unsaturated, of which 41 percent is polyunsaturated, the kind found in vegetable oils that were supposed to make our arteries healthy***.253 In fact in one study, the progression of atherosclerosis was shown to be greater in those who took the least saturated fat.254

Saturated fats constitute at least 50 percent of our cell membranes, giving them stiffness and integrity. Sally Fallon and Mary Enig, PhD, cite a study showing that 50 percent of our dietary fats need to be saturated in order for calcium to be effectively incorporated into the skeletal structure.255

Vitamins A and D, found only in animal fats, help with protein and mineral absorption. Animal products are the richest sources of dietary saturated fats, with the exceptions of coconut, cacao butter,

and palm oil. Protein cannot be utilized without dietary fats. In nature, protein and fats usually come together, as in eggs, milk, fish, and meat from land animals.

Other fat-soluble vitamins found in some animal fats include vitamins E and K. Dr. Price re.ferred to these fat-soluble vitamins as "activators" or "catalysts" and observed that without them we cannot absorb the water-soluble vitamins and cannot utilize the minerals, no matter how much of them we ingest. This is why the whole "low-fat milk" and "low or zero animal fat" craze is crazy! People who promote that have clearly not studied Dr. Price's research.

I recall that when I grew up, everyone suddenly switched from butter to margarine, believing the propaganda that it was "better for the heart." Now we know the opposite is true: margarine is often made from soy, which is toxic to humans, and contains trans fats — even worse, as nutritionist Mary Enig, PhD, and collaborator Sally Fallon explain.

Saturated fats maintain cellular integrity everywhere in the body. Why? Because every cell membrane is ideally made up of about 50 percent saturated fat. When we eat too much polyunsaturated oil and not enough saturated fat (or carbohydrates that the body turns into saturated fat), our cells don't function correctly. Those cell membrane fatty acids need to be saturated for the cell to have the necessary "stiffness," or integrity, and to work properly. When the cell walls do not contain enough saturated fat, they actually become "floppy" and cannot work properly.256

Sally Fallon discusses the benefits of butter and other animal fats in her classic book Nourishing Traditions. These fats contain the "Wulzen factor," named after researcher Dr. Rosalind Wulzen, which gets rid of stiffness in the body: arthritis, hardening of the arteries, cataracts, and calcification of the pineal gland.

Butter also contains conjugated linoleic acids, but only if the cows were completely grass fed. Animal fats (from animals fed their proper diets, not grains) are rich in omega-3 fats and have the right ratio to omega-6 fats, unlike the vegetable oils that are too high in omega-6s. Even lard, which we used when I was a kid, is better than the rancid omega-6 vegetable oils that replaced it! Lard is an excellent source of vitamin D.

Though it is best to eat everything raw, if you must cook, animal fats are more stable when heated than vegetable oils are. Saturated fats are more stable and safe to use at high temperatures in cooking. So when we used to fry things in lard, it was actually much less harmful than using vegetable oils, which are unstable, toxic, and rancid when heated.

Even coconut oil was badmouthed by the food industries eager to sell us their vegetable oils. Coconut oil, though it doesn't contain the crucial vitamins found in animal fats, is stable when used for cooking and helps in weight loss.

One study found that a reduction in dietary saturated fat resulted in lower levels of the "good" HDL cholesterol.257 Even more powerful, researchers Ronald P. Mensink and Martijn B. Katan reviewed 27 studies and found that saturated fat elevated HDL whenever it was used instead of carbohydrates!This effect diminished with increasing use of unsaturated fats.258

Saturated fats also protect the liver from foreign substances, assist in bone growth, protect against infection, influence hormone function, and protect against heart disease.259

Nutritionist and weight-loss coach Dr. Jonny Bowden, PhD, CNS, outlines the reasons saturated fats were blamed for heart disease:

- A diet high in saturated fats can be a problem when they are eaten as part of a high-carb diet.
- Saturated fats were demonized because much of the research on diet and disease lumped saturated fats in with dangerous trans fats.
- Modern saturated fats usually come from poor sources: corn fed, hormone-treated cows, with their unhealthful dairy products, deli meats, etc.260

So What Causes Heart Disease?

Studies show that the percentage of dietary cholesterol has little effect on the risk for cardiovascular disease.261 There is great controversy in the idea that eating a high-cholesterol diet increases blood cholesterol levels. Even if it did, there is no proof that high cholesterol levels alone result in heart disease. Neither is there proof that lowering cholesterol levels will eliminate heart disease, unless they get so low one's mental or emotional well-being might be endangered.

The only concern for cholesterol is that HDL should be high relative to LDL, and the ratio of triglycerides to HDL should be low.262 As far as LDL, the "bad" cholesterol, goes, there are two types, A and B, indicating whether the LDL particles are light and cottony (A) or small and dense (B). Hav.ing a type A LDL pattern is healthier.263

According to Robb Wolf, author of The Paleo Solution, the type B LDL pattern is "born from the VLDL [very low-density lipoprotein] that is the product of high-carbohydrate intake" [emphasis his].264 Gary Taubes concurs.

> *Carbohydrate-rich diets not only lower HDL and raise triglycerides, they also make LDL small and dense. 265*

Furthermore, cholesterol abnormalities are considered symptoms, not causes, of heart disease. As Taubes explains, "The cholesterol seems to be an innocent bystander."266

So now that we have found that animal fats, dietary cholesterol and saturated fats are not as le.thal as previously thought, what has been shown to be the real cause of heart disease?

One theory is a high blood level of the amino acid homocysteine, a metabolite of the amino acid methionine, is to blame. Methionine is a sulfur- containing amino acid found chiefly in flesh foods, nuts, and seeds. Consumption of vitamin B12, as well as vitamins B6 and folate (found abundantly in organ meats and chicken liver, but also in green, leafy vegetables), helps lower excess serum homocysteine levels. A high homocysteine level is also considered to be a risk factor for dementia.

The less B12 one has, the higher blood homocysteine levels rise. The richest source of B12 is meat. Studies show that vegans and vegetarians tend to have higher levels of homocysteine than do nonvegetarians because of consuming less B12.267 On the other hand, too much methionine-containing protein (common in meat) can increase homocysteine beyond acceptable levels, so balance is key. Eat enough animal foods to get adequate B12, but not enough to overdose on methionine.

Another factor in heart disease is high blood levels of insulin. To get one's insulin levels down, one must eat a diet low in carbs. In such a diet, it is usually necessary to include meat, fish, or eggs, since they are the only foods that contain no carbs. The American Heart Association agrees that high-glycemic diets rich in low-fiber carbs are related to heart disease.268

Another factor in heart disease is a blood level high in triglycerides, which increase when insulin levels are high.269 The ratio of triglycerides to HDL should be low. In one study, those people with the highest triglyceride ratio had a 16 times greater risk of heart attack than those with the lowest ratio.270

HDL should be high, the higher the better. An article published in the *New England Journal of Medicine* explained that HDL is "a biomarker for dietary carbohydrate." In other words, a healthfully high level of HDL indicates that one is eating few carbs. If it's low, it is likely you are eating a lot of carbs.271

Consider what Dr. Bowden has to say.

> *Triglyceride levels always come down on a low carb diet. Always. Not "sometimes": all the time. (Which makes sense — the body takes all that excess sugar and packages it into triglycerides; so the less sugar in the diet, the fewer triglycerides in the blood.)272*

Yet another factor in heart disease is inflammation, the C-reactive protein (CRP) theory. Inflammation is made worse by vegetable oils that replaced animal fats, because these oils are very high in omega-6 fats. Many of these fats, such as margarine, are hydrogenated trans fats, which increase the risk of heart disease and many other diseases.273

A high-carb diet, which is the norm with many vegetarians, also increases inflammation unless the carbs are mostly raw fruits and vegetables.274

All of those factors are exacerbated when one ceases to eat healthful meat from free-range farmed animals that were fed their proper, species-specific diets.

The total amount of meat consumed by Americans has risen dramatically in the past century. This increase, from toxic, factory-farmed animals no less, also plays a huge role in heart disease. It gets even worse when one overindulges carbs, so typical on vegetarian and vegan diets.

Ironically, getting rid of healthful animal fats has led to an increased risk of heart disease.

Factors in Heart Disease

- **high homocysteine levels**
- **high insulin levels**
- **high triglyceride levels**
- **high CRP levels (inflammation)275**
- **high LDL cholesterol levels with type B**
- **high VLDL levels**

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THE CHOLESTEROL MYTHS THAT MAY BE HARMING YOUR HEALTH

Source: By Dr. Mercola
http://articles.mercola.com/sites/articles/archive/2011/10/22/debunking-the-science-behind-lowering-cholesterol-levels.aspx

Could it be possible that nearly everything your doctor and the media is telling you about high cholesterol and how it relates to heart disease and strokes is wrong?

Absolutely!

The media and health experts have been giving out massive misinformation about cholesterol. In a thought-provoking two-part series, Dr. Ernest N. Curtis, a doctor of internal medicine and cardiology, puts to rest several decades-old studies that supposedly "proved" the cholesterol-heart disease link.

Debunking the Cholesterol "Science" and Unveiling the Truth

If high cholesterol and high-fat diets are really NOT the cause of heart disease, then how did this massive misinformation campaign start? It actually started more than 100 years ago when the Lipid Hypothesis or the Cholesterol Theory was developed by a German pathologist named Rudolph Virchow. After studying arterial plaques from corpses, he theorized that cholesterol in your blood led to the development of plaques in your arteries.

Meanwhile, in 1913 in St. Petersburg, Russia, Nikolaj Nikolajewitsch Anitschkow fed rabbits cholesterol and determined that it led to atherosclerotic changes (apparently no one questioned the fact that rabbits are herbivores and do not naturally consume cholesterol!). This started the notion that eating

cholesterol leads to plaque deposits in your arteries, and at that time it was believed that all cholesterol in your blood was due to dietary sources.

This, of course, is not true, as it's now known that your liver makes about 75 percent of your body's cholesterol. That's right! Even if you didn't eat *any* cholesterol, you would still have cholesterol in your body, which is a good thing considering it's needed by every one of your cells to produce cell membranes.

Your diet is actually an afterthought when it comes to what your cholesterol levels will be, but this simple truth is largely ignored or unrealized even by many physicians.

In the early 1900s, the Cholesterol Theory was already taking root, but it received even more completely flawed support in the 1950s and subsequent years thereafter. The string of research that effectively solidified the cholesterol myth we know all too well today.

The Seven Countries' Study Incorrectly Links Dietary Fat to Heart Disease

In 1953, Dr. Ancel Keys published a seminal paper that serves as the basis for nearly all of the initial scientific support for the Cholesterol Theory. The study is known as the Seven Countries Study, that linked the consumption of dietary fat to coronary heart disease. What you may not know is that when Keys published his analysis that claimed to prove the link between dietary fats and coronary heart disease (CHD), he selectively analyzed information from only seven countries to prove his correlation, rather than comparing all the data available at the time -- from 22 countries.

As you might suspect, the studies he excluded were those that did not fit with his hypothesis, namely those that showed a low percentage fat in their diet and a high incidence of death from CHD as well as those with a high-fat diet and low incidence of CHD. If all 22 countries had been analyzed, there would have been no correlation found whatsoever; it should have been called the 22 Countries Study!

The nutrition community of that time completely accepted the hypothesis, and encouraged the public to cut out butter, red meat, animal fats, eggs, dairy and other "artery clogging" fats from their diets -- a radical change at that time that is still very much in force today.

Most of the experts I know believe that Dr. Keys' research was pivotal for perpetuating the low-fat approach to health. This is a major part of the solid science you will need to know if anyone seeks to disagree with you when you share this information; this study is really the foundation that triggered the massive emphasis on low-fat diets and the flawed belief that cholesterol is so pernicious.

More Flawed "Proof": The Framingham Study

The next major support for the cholesterol theory came from a study you have likely heard of called the Framingham Heart Study, which is often cited as proof of the lipid hypothesis. This study began in 1948 and involved some 6,000 people from the town of Framingham, Massachusetts who filled out detailed questionnaires about their lifestyle habits and diets. The study is credited with identifying heart disease risk factors, such as smoking, high blood pressure, lack of exercise and, yes, high cholesterol.

The cholesterol link was weak, as researchers noted those who weighed more and had abnormally high blood cholesterol levels were slightly more at risk for future heart disease, but widely publicized. **What you *don't* hear about is the fact that the more cholesterol and saturated fat people ate, the *lower* their cholesterol levels.**

In a 1992 editorial published in the Archives of Internal Medicine, Dr. William Castelli, a former director of the Framingham Heart study, stated:

"In Framingham, Mass., the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower the person's serum cholesterol. The opposite of what… Keys et al would predict…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."

The "MrFit" Study: Hypothesis Proven by Omission

The U.S. Multiple Risk Factor Intervention Trial (MRFIT), sponsored by the National Heart, Lung and Blood Institute, is another study that is highly misleading. It compared mortality rates and eating habits of over 12,000 men, and the finding that was widely publicized was that people who ate a low-saturated fat and low-cholesterol diet had a marginal reduction in coronary heart disease.

What did they leave out?

Their mortality from all causes was higher! As Mary Enig and Sally Fallon stated in *The Truth About Saturated Fat*:

"The few studies that indicate a correlation between fat reduction and a decrease in coronary heart disease mortality also document a concurrent increase in deaths from cancer, brain hemorrhage, suicide and violent death. After 10 years of lowering fat intake and not smoking, they found no significant difference in death from heart disease or total death compared to the control group of smokers, poor diet etc."

Statistical Lies: The Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT)

Around the same time as the MRFIT study was the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT), which cost $150 million and is often cited to justify a low-fat diet, even though dietary factors were not tested in the study at all. Instead, the study tested the effects of cholestyramine, a cholesterol-lowering drug.

As Enig and Fallon wrote:

> ***"Their statistical analysis of the results implied a 24% reduction in the rate of coronary heart disease in the group taking the drug compared with the placebo group; however, non-heart disease deaths in the drug group increased --***

deaths from cancer, stroke, violence and suicide. Even the conclusion that lowering cholesterol reduces heart disease is suspect.

Independent researchers who tabulated the results of this study found no significant statistical difference in coronary heart disease death rates between the two groups. However, both the popular press and medical journals touted the LRC-CPPT as the long-sought proof that animal fats are the cause of heart disease ..."

What really happened, and how LRC-CPPT came to lend further support to the lipid hypothesis was nothing more than another masterful case of statistical manipulation. As Dr. Curtis stated:

"After 10 years the number dying from coronary heart disease (CHD) plus those suffering a non-fatal myocardial infarction (NFMI) were totaled for both groups. The total incidence in the cholestyramine group was 7.0% and the control group 8.6%.

This small difference of 1.6% was reported as a 19% reduction in death and heart attack by using relative risk reduction (the difference of 1.6% is roughly 19% of 8.6) in place of the less misleading absolute risk reduction (1.6%). Furthermore, this tiny difference was given the designation of "statistically significant" by changing the criteria originally given for determination of significance after the data was in."

It is often the case that leaders who want to use the cholesterol agenda use statistics to "prove" their point.

Cholesterol Drug Benefits Perpetuated by Statistical Myths

The LRC-CPPT study was only able to show a meaningful benefit because it focused on *relative* risk reduction rather than *absolute* risk reduction. What's the difference? You can find a very simple explanation of relative risk vs. absolute risk at the Annie Appleseed Project web site, but let me sum it up here.

- Relative risk reduction is calculated by dividing the absolute risk reduction by the control event rate
- Absolute risk reduction is the decrease in risk of a treatment in relation to a control treatment

In plain English, here's what that means: let's say you have a study of 200 women, half of whom take a drug and half take a placebo, to examine the effect on breast cancer risk. After five years, two women in the drug group develop breast cancer, compared to four who took the placebo. This data could lead to either of the following headlines, and *both* would be correct:

"New Miracle Drug Cuts Breast Cancer Risk by 50%!"

"New Drug Results in 2% Drop in Breast Cancer Risk!"

How can this be?

The Annie Appleseed Project explains:

"The headlines represent two different ways to express the same data. The first headline expresses the relative risk reduction — the two women who took the drug (subjects) and developed breast cancer equal half the number (50%) of the four women who took the placebo (controls) and developed breast cancer.

The second headline expresses the absolute risk reduction — 2% of the subjects (2 out of 100) who took the drug developed breast cancer and 4% of the controls (4 out of 100) who took the placebo developed breast cancer — an absolute difference of 2% (4% minus 2%)."

You can now see why clinical trials, especially those funded by drug companies, will cite relative risk reductions rather than absolute risk reductions, and as a patient you need to be aware that statistics can be easily manipulated.

As STATS at George Mason University explains:

"An important feature of relative risk is that it tells you nothing about the actual risk."

How Statins Really Work Explains *Why* They Don't Really Work

A new look at statin cholesterol-lowering drugs from the Massachusetts Institute of Technology claims that no study has ever proven that statins improve all-cause mortality -- in other words, they don't prolong your life any longer than if you'd not taken them at all. And rather than improving your life, they actually contribute to a deterioration in the quality of your life, destroying muscles and endangering liver, kidney and heart function.

According to Stephanie Seneff, author of this stunning revelation:

"Statin drugs inhibit the action of an enzyme, HMG coenzyme A reductase, that catalyses an early step in the 25-step process that produces cholesterol. This step is also an early step in the synthesis of a number of other powerful biological substances that are involved in cellular regulation processes and antioxidant effects.

One of these is coenzyme Q10, present in the greatest concentration in the heart, which plays an important role in mitochondrial energy production and acts as a potent antioxidant …

Statins also interfere with cell-signaling mechanisms mediated by so-called G-proteins, which orchestrate complex metabolic responses to stressed conditions. Another crucial substance whose synthesis is blocked is dolichol, which plays a crucial role in the endoplasmic reticulum. We can't begin to imagine what diverse effects all of this disruption, due to interference with HMG coenzyme A reductase, might have on the cell's ability to function …

There can be no doubt that statins will make your remaining days on earth a lot less pleasant than they would otherwise be … "

It's widely known that statins lower your CoQ10 levels by blocking the pathway involved in cholesterol production -- the same pathway by which Q10 is produced. Statins also reduce the blood cholesterol that transports CoQ10 and other fat-soluble antioxidants.

The loss of CoQ10 leads to loss of cell energy and increased free radicals which, in turn, can further damage your mitochondrial DNA, effectively setting into motion an evil circle of increasing free radicals and mitochondrial damage.

There are no official warnings in the U.S. regarding CoQ10 depletion from taking statin drugs, and many physicians fail to inform you about this problem as well. Labeling in Canada, however, clearly warns of CoQ10 depletion and even notes that this nutrient deficiency "could lead to impaired cardiac function in patients with borderline congestive heart failure."

As your body gets more and more depleted of CoQ10, you may suffer from fatigue, muscle weakness and soreness, and eventually heart failure, so it is imperative if you take statin drugs that you take CoQ10 or, if you are over the age of 40, the reduced version called ubiquinol.

Statins May Even Cause Diabetes!

Statins carry other side effects as well, including diabetes. A meta-analysis, published in JAMA in June, concluded that those taking higher doses of statins were at increased risk of diabetes compared to those taking moderate doses. What this means is that the higher your dose, the higher your risk of developing diabetes.

The "number needed to harm" for intensive-dose statin therapy was 498 for new-onset diabetes—that's the number of people who need to take the drug in order for one person to develop diabetes. In even simpler terms, one out of every 498 people who are on a high-dose statin regimen will develop diabetes. (The lower the "number needed to harm," the greater the risk factor is.)

(As a side note, the "number needed to treat" per year for intensive-dose statins was 155 for cardiovascular events. This means that 155 people have to take the drug in order to prevent *one* person from having a cardiovascular event.)

Aside from what I've already covered above, statin drugs are associated with a rather extensive list of harmful side effects, including:

Weakness	Polyneuropathy (nerve damage in the hands and feet)	Acidosis	Dysfunction of the pancreas
Muscle aches and pains	Anemia	Sexual dysfunction	Cataracts
Rhabdomyolysis, a serious degenerative muscle tissue condition	Potential increase in liver enzymes so patients must be monitored for normal liver function	Suppressed immune function	Increased cancer risk

So How Can You Optimize Your Cholesterol Levels?

The most effective way to optimize your cholesterol profile and prevent heart disease is via diet and exercise. It's actually quite simple too. Remember that 75 percent of your cholesterol is produced by your liver, which is influenced by your insulin levels.

Therefore, if you optimize your insulin level, you will automatically optimize your cholesterol and reduce your risk of both diabetes and heart disease. There is NO magic pill to cure heart disease, as the underlying cause is insulin resistance caused by eating too many sugars, grains and especially fructose.

So, my primary recommendations for safely regulating your cholesterol and reducing your risk of heart disease include:

- **Reduce, with the plan of eliminating grains and fructose from your diet.** This is one of the best ways to optimize your insulin levels, which will have a positive effect on not just your cholesterol, but also reduces your risk of diabetes and heart disease, and most other chronic diseases. Use my Nutrition Plan to help you determine the ideal diet for you, and consume a good portion of your food raw.

- **Get plenty of high quality, animal-based omega 3 fats, such as krill oil**, and reduce your consumption of damaged omega-6 fats (trans fats, vegetable oils) to balance out your omega-3 to omega-6 ratio.

- **Include heart-healthy foods in your diet, such as olive oil, coconut and coconut oil, organic raw dairy products and eggs, avocados, raw nuts and seeds, and organic grass-fed meats.**

- **Optimize your vitamin D levels by getting proper sun exposure** or using a safe tanning bed.

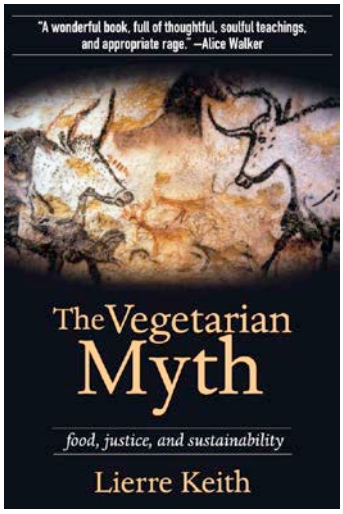
- **Exercise daily.** Make sure you incorporate peak fitness exercises, which also optimizes your human growth hormone (HGH) production.

- Avoid smoking or drinking alcohol excessively.

- Be sure to get plenty of good, **restorative sleep.**

CHOLESTEROL AND THE LIPID HYPOTHESIS: EXPOSED

Source: *The Vegetarian Myth* by Lierre Keith, Chapter 4: Nutritional Vegetarians, pgs 160-176



Cholesterol is, of course, the bulwark that the nutritional vegetarians will stand behind. The Lipid Hypothesis-the theory that ingested fat causes heart disease-is the stone tablet that the Prophets of Nutrition have brought down from the mountain. We have been shown the one, true way: cholesterol is the demon of the age, the dietary Black Plague, a judgment from an angry God, condemning those who stray into the Valley of Animal Products with disease. That at least is what the priests of the Lipid Hypothesis declared, having looked into the entrails of ... rabbits.

Rabbits?

Yes, it all began when researchers fed protein and cholesterol to rabbits and their blood cholesterol shot up. And it reached numbers never seen in humans. The cholesterol was in the rabbits' arteries, but it produced a different kind of lesion than in humans, and the animals never developed advanced plaques in their blood vessels. Instead, cholesterol accumulated in their organs, resulting in fatty buildup in their kidneys and livers, discolored eyes, and loss of fur. These force-fed rabbits didn't die from coronary disease; they died from starvation because they lost their appetites. Which is about what you'd expect when you take an herbivore designed for cellulose and stuff her full of fat and protein.

This haruspicy has also been done on "chickens, guinea pigs, pigeons, parrots, goats, rats and mice" with similar arterial deposits developing.[44]

When these experiments are done on carnivores-cats, dogs, foxesno damage results. In dogs, cholesterol feeding had no effect at all unless the poor creatures had their thyroids removed or chemically suppressed.[45]

Writes Anthony Colpo, "High amounts of cholesterol appeared to be readily metabolized by carnivorous animals, whereas herbivorous animals may not be equipped to metabolize large amounts of dietary cholesterol or animal fat, both of which are absent from plant foods."46
Not to put too fine a point on it, but duh?

Remember that 80 percent of the cholesterol in your blood was made by your body. Only 20 percent was put there by your food choices. Your body knows where it wants that cholesterol level. It may have been misled-by insulin, for instance, but it will adjust its production based on what you ingest. If you eat more cholesterol, it will produce less.

A meta-analysis of *one hundred sixty seven-yes,* that's 167-cholesterol-feeding experiments found that raising dietary cholesterol had a negligible effect on

blood cholesterol, and no link to CHD (coronary heart disease) risk. 47

Before we go any further, do you even know what cholesterol is?

This benign, maligned substance is needed by every cell in your body, and most of all by the ones that make you human. Cholesterol is technically a sterol, not a fat**. One of the main functions of the liver is to make cholesterol, not because your liver wants you dead, but because life isn't possible without cholesterol.** Low levels of cholesterol may very well kill people. The increased mortality due to *low* cholesterol is serious enough that the National Heart, Lung and Blood Institute of the National Institutes of Health held a conference to explore researchers' findings on the subject.[48] **Evidence from a multitude of sources was presented linking low blood cholesterol levels to an increase in various cancers, hemorrhagic stroke, respiratory and digestive diseases, and violent death," Colpo sums Up. 49**

In France, a study of 6,000 men over seventeen years showed that those whose cholesterol declined the most had the *highest* risk for cancer.[50]

Or how about the heart failure patients whose risk of death was twice as high for those with the *lowest* cholesterol levels?51

There's a lot more, but none of it will make sense until you understand that **cholesterol is a life-sustaining substance, not a murderer inside your blood.**

Cholesterol has a special trick that plays a crucial role in animal bodies: it doesn't dissolve in water. Our internal environment is liquid. Hence, cell membranes need to be structurally stable. Without cholesterol, you would be a puddle, not an animal. Your cell membranes also need to be waterproof. This is especially true for the cells of your nervous system, including your brain, which is one reason why more cholesterol is found there than anywhere else.

Cholesterol is also the body's basic repair substance. The integrity of your intestinal wall especially depends on it. And cholesterol has antioxidant powers, keeping cancer-causing free radicals from doing their damage. Finally, all of your hormones, including your sex hormones, are made from cholesterol.
Does that sound so awful?
As a culture, we've been collectively sitting around the campfire and, while night takes hold, listening to the big kids like the American Heart Association and the USDA. They've been telling us a story about an escapee from a mental hospital with an alias of Cholesterol and a hook for a hand ... The grownups are there in the background, telling us it's not true, but when do we listen to them?

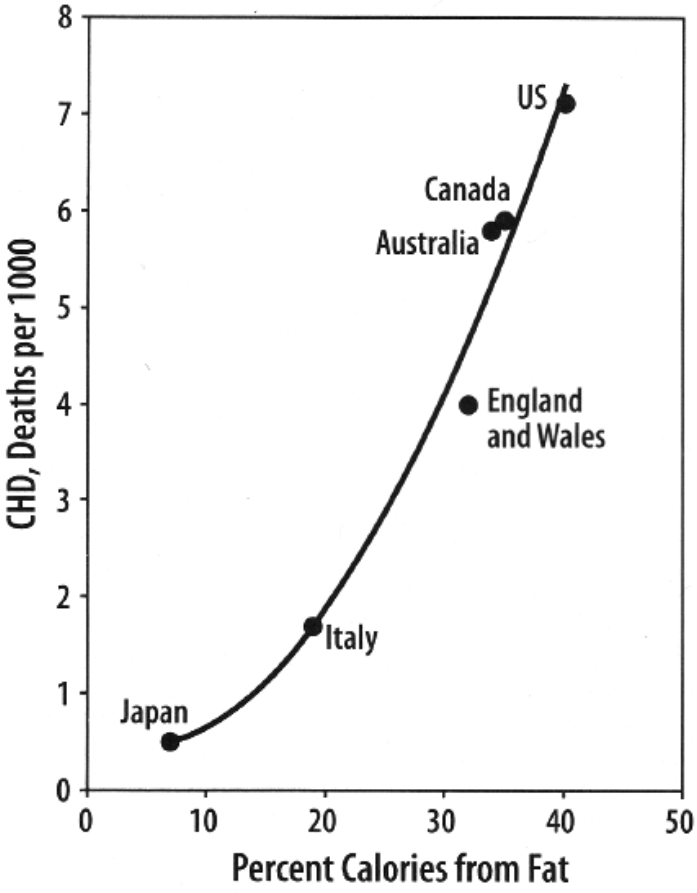


Figure 4A. Correlation between the total fat consumption as a percent of total calorie consumption, and mortality from coronary heart disease in six countries. Redrawn from *The Cholesterol Myths* by Uffe Ravnskov.

One of the big kids was Ancel Keys, who assembled the famous Six Countries Study. **Figure 4A shows what *he* wanted you to know.**

This "study" is absurd for two reasons. To understand them, you need the basic science education that the public school system failed to provide. The whole point of an experiment is to test a hypothesis. You do that by eliminating as many variables as possible. With epidemiological evidence like the Keys study, it's impossible. That's why epidemiological studies can only prove correlation. They cannot prove causality. They may suggest intriguing areas for exploration but until all the variables are controlled and the results are reproducible, no conclusions can be drawn. The kind of cross-country comparison that Keys did "involves comparing apples with oranges-that is countries with widely varying cultural, social, political and physical environments."52 With such an infinite number of variables, a finding of definitive causation would be ridiculous.

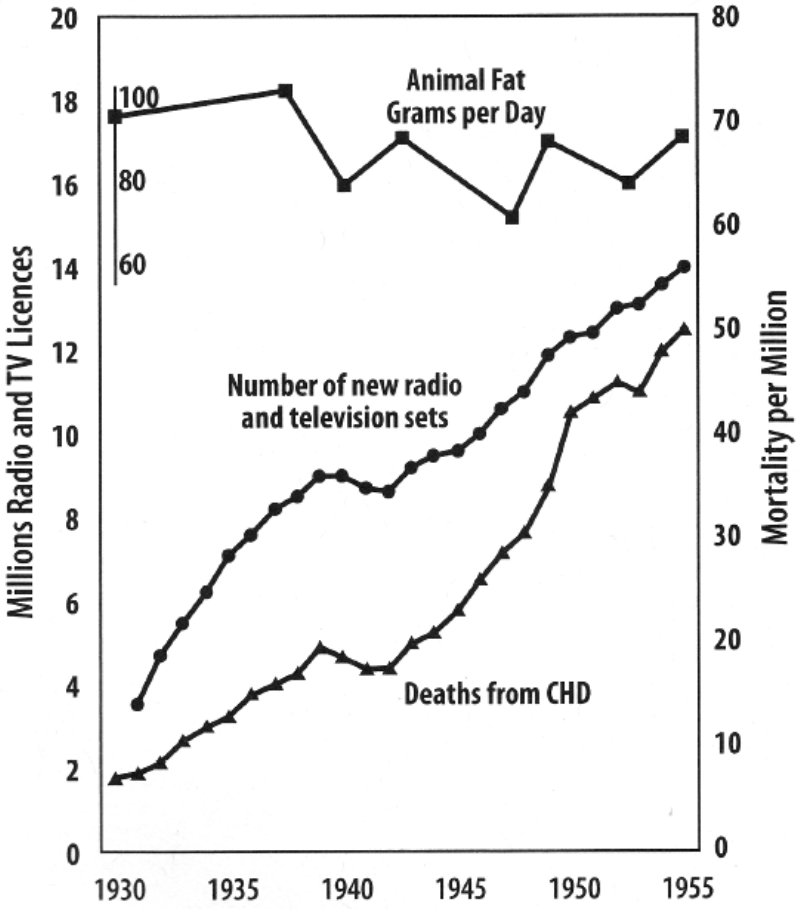


Figure 4B. Consumption of animal fat, number of new radio and television sets and number of deaths from coronary disease in England and Wales between 1910 and 1956. Redrawn from *The Cholesterol Myths* by Uffe Ravnskov.

John Yudkin's 1957 study shows the error of conflating correlation with causation. You can see from Figure 4B that owning a TV and radio had a much stronger association with Coronary Heart Disease (CHD) than any nutritional elements. 53

But no one would suggest that TV causes CHD, or that sacrificing our TVs will grant us a longer life. No one went on to investigate whether TVs produced heart-stopping emissions or blood-damaging toxins. No government health agency paid for people to throw out their TVs as a treatment for CHD. No one mistook association for causation.

Dr. Uffe Ravnskov made a graph (Figure 4C) showing that income tax rates correlate with CHD. According to his graph, if the tax rate dropped below 9.55 percent, the good citizens of Sweden would be free of the scourge of CHD.54

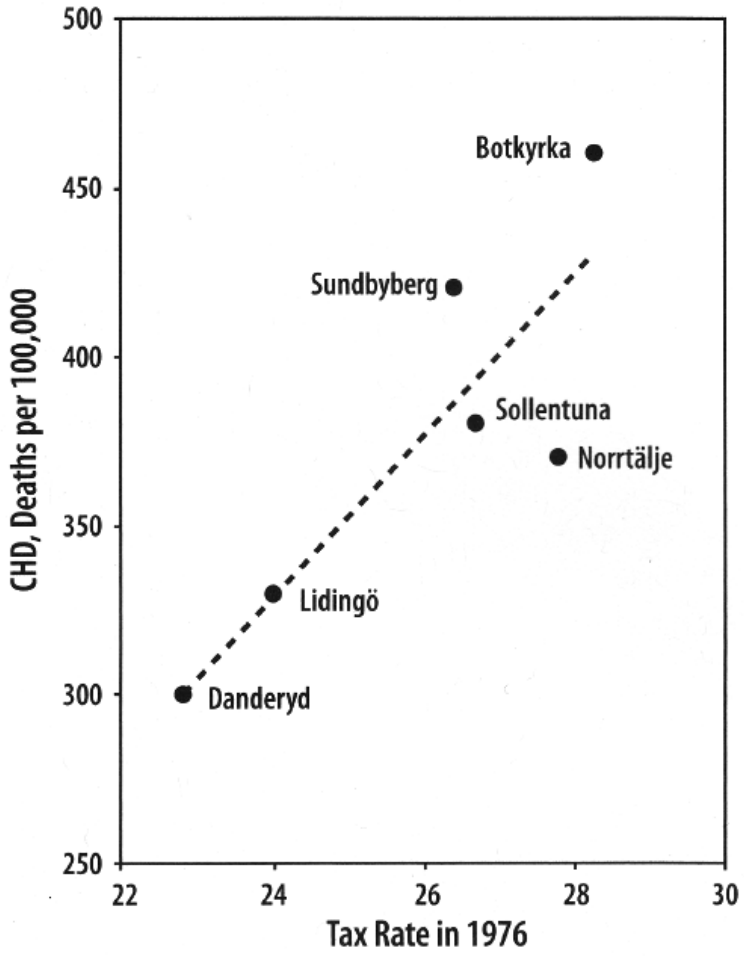


Figure 4C. Correlation between tax rate and coronary mortality in the municipal tax districts of the county of Stockholm, Sweden. According to this graph, if the tax rate drops to 9.55 percent, CHD will be conquered.
Redrawn from *The Cholesterol Myths* by Uffe Ravnskov.

These kinds of epidemiological studies make for snappy headlines. I see them all the time. There was one recently about body weight and sleep. Apparently researchers correlated subjects' weight and the amount they slept, and the relationship was an inverse proportion. The more you weigh, the less you sleep. Does that mean that if you sleep more you lose weight? To judge by the message boards, a fair number of people jumped right from correlation to causality, with no stop in between at rationality. Yes, it's one possible explanation for the correlation: less sleep somehow causes weight gain. So more sleep might help you lose weight. It could just as easily be the other way around: more body weight causes insomnia, and sleeping more will only help the insomnia. Or it could be a million other things.

My point here is to never, ever put your money-let alone your physical well-being-on an epidemiological study. And learn to distinguish between correlation and causality. Or, as one set of researchers put it, after their high-fat data refuted their low-fat hypothesis, "Observational studies on populations are only useful for formulating hypotheses and they cannot provide convincing evidence of causeand-effect relations."55

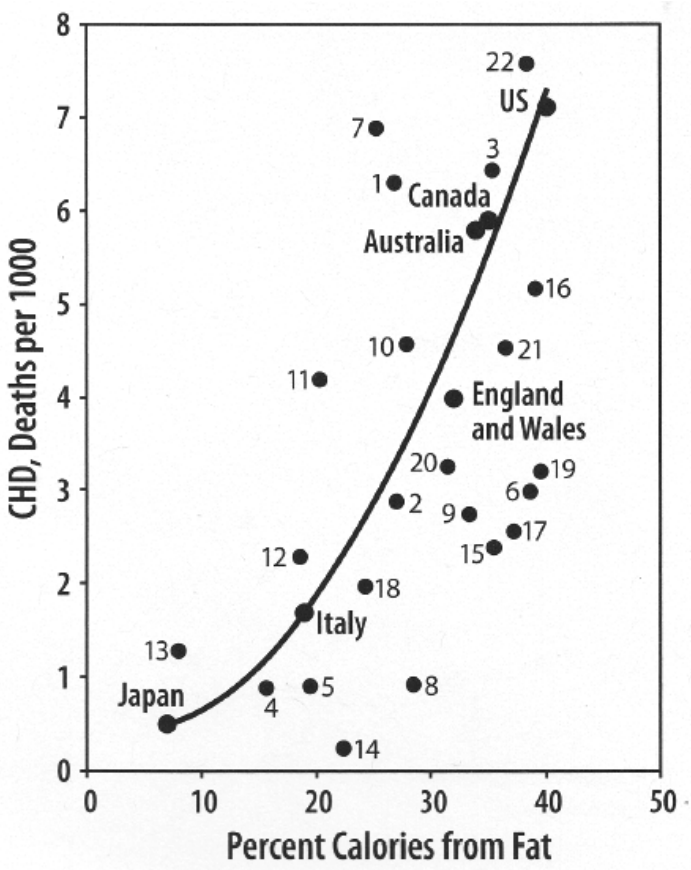


Figure 4D. Same as Figure 4A, but including all countries where data were available when Dr. Keys published his paper. 1. Australia; 2. Italy; 3. Canada; 4. Sri Lanka; 5. Chile; 6. Denmark; 7. Finland; 8. France; 9. West Germany; 10. Ireland; 11. Israel; 12. Italy; 13. Japan; 14. Mexico; 15. Holland; 16. New Zealand; 17. Norway; 18. Portugal; 19. Sweden; 20. Switzerland; 21. Great Britain; 22. USA. Redrawn from *The Cholesterol Myths* by Uffe Ravnskov.

Keys only used numbers that supported his point. He had nutritional data from twenty-two countries and he only used the ones that he liked. Figure 4D restores all the data he excluded. You can see how his hypothesis is utterly refuted by the data that he had-and willfully ignored.

Another researcher, Dr. George Mann, found that Keys had also removed those countries that correlated lack of exercise to CHD.56 Even on its own terms, Keys' study was a disaster until he tortured the data.

Dr. Malcolm Kendrick put together a similar chart (see Figure 4E) using updated data from the MONICA project of the WHO (World Health Organization). MONICA stands for "MONItor trends in CArdiovascular diseases." It was the single largest investigation into diet and CHD *ever,* including nutritional data from twenty-one countries and ten million people over ten years.

The results? Not even a correlation between cholesterol levels, fat intake, and cardiovascular mortality. Kendrick also notes that if Keys had chosen Germany, Switzerland, France, and Sweden instead of Greece, Former Yugoslavia, USA, and Japan, Keys would have "shown" the opposite correlation,

"Namely, the more saturated fat and cholesterol consumed the lower the risk of CHP." 57

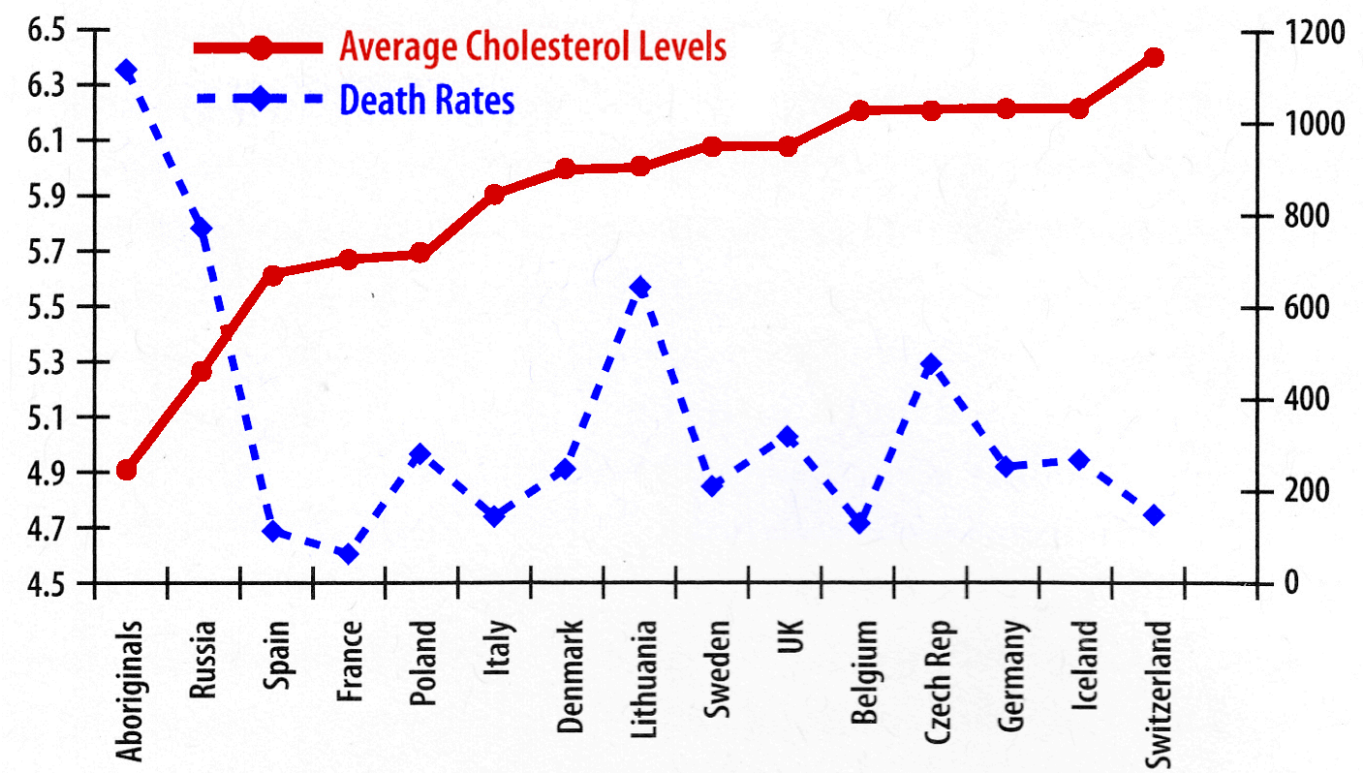


Figure 4E. Comparison between heart-disease rates in men aged 35-74 and average cholesterol levels in 15 populations. Redrawn from Dr. Malcolm Kendrick's *The Great Cholesterol Con*.

But the big kids over at the American Heart Association, the USDA, and Pfizer like their one-hooked villain. Though this information has been available for forty years, and numerous doctors and researchers have been decrying the Lipid Hypothesis as a fraud for as long, the orthodoxy still refers to "the Keys Equation" as "the most precise way to predict the effect of diet on the blood cholesterol levels of individuals and populations, and thus, their risk of coronary heart disease."58

Clearly it's up to us to figure out the truth about diet and health, fats and hearts, cause and effect.

CHD is responsible for vast amounts of death and disability in the US. I hope the evidence presented so far-especially the visual evidence-is compelling, hopefully compelling enough to be liberatory. Throw out that sickly canola margarine, that inedible skim milk, those endless fat-free soy extrusions whose only flavor is a rancid aftertaste you've vowed to ignore. Your body-your brain, your bones, and your heart-is hungry, and somewhere inside you, you know it's true. You have nothing to lose but your punishment.

If you want to dig deeper into the research, if you need more information to feel like you're on solid ground before an undertaking as serious as a dietary overhaul, I suggest the following guidelines.

1. Epidemiological studies are of limited use, since the endless number of variables they include can't be controlled.

2. If you do look at epidemiological studies, take care never ever to conflate correlation with causality.

3. Controlled studies are a better bet, but read them carefully. Do not ever believe the headline sound bite on Yahoo! News. And don't just trust the conclusions, but read the whole study. Data is often starved or force-fed to support the bias of the researcher. See for yourself whether every variable was the same *except for the one being tested.* And follow the money. Be ultra-wary of studies funded by drug manufacturers.

4. Never trust just one study, no matter how good it looks or how much you like its conclusions. Remember the basic principle of science: the results have to be reproducible to count.

5. Heed the words of Jessica Prentice, author of *Full Moon Feast,* who writes, "Although bookstores are full of advice on how to be healthy or thin, or both, and there is a constant stream of media telling us which foods are good for us and which bad, I have found very little of what I hear about food in contemporary America to be useful to me. The surfeit of information doesn't help me eat well-in fact, it confuses me and sets me back."59

I've been through that same confusion, which can feel as strong as terror, when something as basic as food, and as primary as identity, gives way, and the stable rules of good and bad, me and not me, collapse. You may feel driven to shore up those rules after viewing some of the graphs presented here. I have known that impulse, sometimes desperately, and it's a very human reaction. But to pursue the truth, we have to leave room for the possibility that we might be ignorant, or even wrong. We have to accept confusion, embrace the risk of not knowing. As a culture we've lost the moorings of traditional lifeways and their foods. Corporate America began taking over the food stream in the 1920s, and the process has been complete for over a generation. We have very little to go on, and the experts our culture offers in the place of wisdom have not proven trustworthy. If we acknowledge that this is difficult-that we are in for a bit of Mr. Toad's Wild Ride-it will go easier.

In order for the Lipid Hypothesis to become the Lipid Law, the following dots would have to be connected. Saturated fat would have to raise cholesterol levels, and cholesterol would have to cause CHD.

Saturated fat → raised cholesterol → CHD

There is a huge array of epidemiological studies that show *no correlation between saturated fat consumption, cholesterol levels, and heart disease.*

Let's look at some of those first, not because I think they're so great as a concept, but because proponents of the Lipid Hypothesis love epidemiological studies so much. First are all the paradoxes: the French Paradox, the Greek Paradox, the East African paradox, the Swiss Paradox, the Pacific Island Paradox. These countries have high levels of saturated fat consumption, but low levels of heart disease. France has one of the highest-the French consume four and a half times as much butter as US Americans, for instance-but the French have substantially lower CHD.60 The Masai of Kenya eat a diet almost entirely of meat, milk, and blood. On average, young Masai warriors ingest 300 grams of animal fat every day. Yet their cholesterol levels are some of the lowest found anywhere-averaging under

160-and heart disease is unknown. On autopsy, atheromas (bad arterial plaques) were absent. George Mann, the researcher who studied the Masai, was led by his findings to declare the Lipid Hypothesis "the public health diversion of this century ... the greatest scam in the history of medicine."61

A study of the Samburu tribe of Uganda yielded similar findings-neither heart disease nor elevated cholesterol levels, despite a daily diet of 400g of animal fat. They also had no rheumatoid arthritis, degenerative arthritis, or high blood pressure.[62]

Another African pastoralist culture is the Kalenjins of Kenya.

Raw and fermented dairy products form the bulk of their diet. Not only are they free of chronic and degenerative diseases, they are world-renowned runners. "Athletes from this one tribe of 3 million people have won 40 percent of all the highest international honors available in men's distance running," in track, cross-country, and road racing. 63 A Kalenjin has won the Boston Marathon four times since 1988. Ron Schmid calls this "an indication of profound natural forces at work."64

Another epidemiological study discovered the Pacific Island Paradox. Coconut is a staple food of the people of Pukapuka and Tokelau, and coconut oil is more highly saturated than animal fats. The two islands' inhabitants consumed 35% and 55%, respectively, of their calories in the form of saturated fat. Cardiovascular disease was absent, as were degenerative diseases in general.[65] To quote Dr. Malcolm Kendrick, "I would just ask, how many paradoxes do you need before the only paradox left is the diet-heart hypothesis itself?"66

The Japanese? They've increased their consumption both of total fat and animal fat over 250 percent since 1961-and they are now *the longest living people in the world.* Stroke was the number one cause of death in the 1960s, but both stroke incidence and mortality from strokes declined rapidly from 1960 to 1975. And was there a dietary change during this period? Yes. Consumption of both animal protein and fat *increased* significantly, as one would expect during a time of economic prosperity. Blood cholesterol also increased, while blood pressure and strokes went down. To get even more specific, Japanese researchers tracked 3,700 people from 1984 to 2001, and those who ate the most animal fat had a "sixty-two percent lower risk of ischemic stroke death."67

Want more? A survey of 40,000 Japanese subjects found that over a sixteen-year period, "those who ate the most eggs, dairy products, and fish had a twenty-eight percent lower risk of stroke than those who ate the least."68

Then there's India, where the incidence of CHD was examined in over a million men. The highest CHD rates were in Madras, which is in southern India. The lowest rates were in Punjab, which is in the north. Their dietary difference? In disease-prone Madras, fat consumption was lower and consisted of polyunsaturated vegetable oils. In healthier Punjab, milk products supplied the fat, with only 2 percent coming from polyunsaturates. The Punjab men with their protective saturated fats were "seven times less likely to die from heart disease than those in Madras," and their overall life span was eight years longer. This, despite the fact that they smoked more.[69]

And then there's China. There is a bizarre and entrenched myth among the health-conscious in the West that the Chinese don't have cardiovascular disease. The idea is that they eat a lot of rice and vegetables and very little protein or fat, are healthy, and thus are living proof of the vegetarian myth. Write the Drs. Eades:

However, the truth of the matter is that the Chinese do indeed have cardiovascular disease, and lots of it .... The rates of death from cardiovascular disease suffered by both rural and urban Chinese males is almost indistinguishable from the rate experienced by American males, while the rates of cardiovascular deaths for both rural and urban Chinese women is significantly higher than those suffered by American females .... The notion that the Chinese don't have disease of the heart and blood vessels is what we like to call a vampire myth-it simply refuses to die. The myth that low-fat, high-carbohydrate diets are healthy lives on and on.70

The difference between Chinese cardiovascular disease and cardiovascular disease in the US is simply the form it takes. In China, it's stroke; in the US, heart attack. For urban Chinese men, the rate of heart attack is about half that of US American men, but their rate of stroke is almost *six times higher*. For urban Chinese women, the heart attack rate is almost three-quarters the rate of US Americans and their stroke rate is about *five times higher*. 71

Had enough? Who knows what other factors are involved with countries on the other side of the world, you might be saying defensively. Fine, let's look at the United States.

The past fifteen years have seen a reduction in fat consumption of almost 25 percent,72 due to the relentless badgering of the medical establishment and the willingness of corporate food manufacturers to fabricate an endless array of faux-foods with their faux-fats: cheap polyunsaturated vegetable oils that have to be chemically altered to approach the mouthfeel that humans, craving our native saturated fats, will accept.

Twenty-five percent is a big reduction. Did you get healthier? Or did you notice that the incidence of diseases commonly blamed on animal products has gone from high to epidemic?

Type-2 diabetes has increased by a factor of more than *ten*. Heart disease deaths, after more than ten years of decline, took a turn for the worse in 1992 and have slowly been increasing since. An accurate measure of the increase in cardiovascular diseases can be seen in the rates of discharge from the hospital of patients with that diagnosis, which, according to the American Heart Association, have increased by 25 percent since 1976. The incidence of stroke is on the rise, and cancer continues its relentless and increasing toll with the very cancers most often blamed on fat consumption-cancer of the breast and prostate-leading the charge.73

Some of the experts have noticed, and even publicly admitted, that the dietary experiment inflicted on the US public has been an utter failure.l[4] William Willett of the Harvard School of Public Health has said, "Low-fat has been like a religion. But it was just a hypothesis to begin with."75 We've done what they told us-ate less fat, more carbohydrate-and have gotten sicker.

Or look at the famous Framingham Heart study. Started in 1948 to monitor the health of five thousand residents of a Boston suburb, it attempted to examine the Lipid Hypothesis by measuring serum cholesterol levels and CHD. It's well worth reading the whole study as an object lesson in denial. For example, *declining* cholesterol levels in people over 50 were associated with *increases* both in overall mortality and in death from CHD. "For every 1 *mgl* dl per year drop in cholesterol levels during the first 14 years of the Framingham study, there was a 14% increase in cardiovascular death and 11 % increase in overall mortality during the subsequent 18 years."76 Yet the study is claimed by proponents of the Lipid Hypothesis to prove the link between high cholesterol and CHD.

And the role of saturated fat in Framingham? Dr. William Castelli, the study's director, has written publicly that "In Framingham, Mass., the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower the person'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."77

Never mind the epidemiological studies. We don't like them anyway. What we really need is a rigorous, controlled study. Anthony Colpo describes what that perfect clinical trial would look like:

> "Such a trial would compare a group of subjects of similar sex, age and health status, who have been randomly assigned to eat diets that are identical in every respect, except that one contains a significant amount of saturated fat (the control group), while the other contains a greatly reduced amount (the treatment group). Ideally, this trial would be 'double-blind', meaning that both researchers and participants would be unaware of who is in the treatment group and who is in the control group, a safeguard that would help prevent researcher bias and the possibility of a placebo effect amongst the subjects."78

In fact, such studies have been done, and done relentlessly, trying to prove some link between saturated fat, cholesterol, and CHD. Some of them meet standards that are scientifically rigorous; others must be read with a cautious and educated eye. The very first was designed by Lester M. Morrison in 1946. It specifically sought to investigate the relationship between the reduction of fat consumption and cardiac deaths. One hundred heart attack survivors were divided into two groups. The intervention group was placed on a calorie-restricted, low-fat, high-protein diet supplemented with calcium, phosphorus, brewer's yeast and wheat germ. At year eight, twenty-two of the intervention group had died while thirty-eight of the control subjects died.

Hopefully you can start to see the problem with this study. This was a multifaceted intervention, and there is no way to know which of the variables was the one that did the cardiac trick. The higher protein? That's been linked to lower CHD. Some members of the intervention group lost weight and that alone can improve cardiovascular profiles. We know that B vitamins-present in both the brewer's yeast and the wheat germ-lower levels of homocysteine, which is an atherogenic agent. Selenium is an antioxidant that may have clinical benefit for CHD patients, and yeast is a good source. Any of these variables could be responsible, and there's no way of knowing which one until each element is controlled. So when advocates of the Lipid Hypothesis hold up this particular study as evidence-and some of them do-feel free to know better.

The first clinical study on the Lipid Hypothesis that was blinded, randomized, and controlled-the first one, in other words, worth mentioning-was done in London, England, in 1965. Researchers took eighty volunteers and substituted corn oil for the saturated fat in their food. Notice: the fat was the only thing that changed. And the results? The corn oil people saw an average drop in serum cholesterol of 23 *mgl* dl. They also died. There were more "CHD incidents, deaths and total deaths" in the intervention group than in the control group. Another group was put on olive oil with results almost as bad. In the doctor's words, "under the circumstances of this trial corn oil cannot be recommended in the treatment of ischaemic heart disease. It is most unlikely to be beneficial, and it is possibly harmful."79 Would that anyone had listened.

The first trial of the Lipid Hypothesis in the US was called the Anti-Coronary Club. Published in 1966, it compared eleven hundred men eating the "Prudent Diet" with a control group eating regularly. The Prudent Diet replaced saturated fat with polyunsaturated fat. The subjects' cholesterol levels dropped from an initial reading of 260 to an average of225. These are the details the summary crows about. You might think the study had a happy ending-unless you kept reading. Nine months later, a second article revealed that eight of the prudent subjects died from heart attacks, while *none* of the control group did. Further, the total deaths on the Prudent Diet numbered twenty-six; only six men in the control group died. The deaths are basically ignored in the authors' discussion.[80] Which proves something beyond the fallacy of the Lipid Hypothesis, something about the inhuman rationality of science and the egos involved therein that many of us would rather not know.

Maybe you don't need to read all the studies, or all the books that debunk them. Maybe knowing that there are cultures consuming 80 percent of their calories in the form of saturated fat with no CHD is enough. Maybe in the bottom of your mind is the place where love is food and food is love and you can still see the color of your grandmother's kitchen. You always knew she was right: butter was good, margarine a disgrace. You had real food once, made for you by a woman who knew what children needed because *her* mother knew, the generations a Russian nesting doll of nourishing wisdom. Somebody fed you once. Let yourself remember: it was good.

Or maybe it's not that easy, surrounded as we are by voices of authority bearing down on us with yet another reminder that our appetites are dangerous, our bodily hungers a war we need to fight. That war, of course, will be endless, the profits to the corporate food oligarchs immense. There will never be room in their annual reports for the local and sustainable, the truly nourishing, just as there is no room for the inconvenient dead in their scientific summaries.

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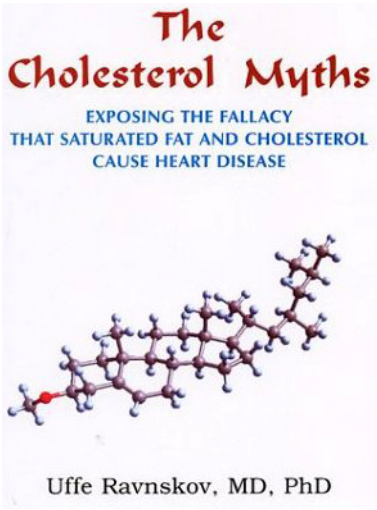
44 Colpo, p. 33.
45 Steiner and Kendall, p. 433.
46 Colpo, p. 33.
47 Ibid., p. 34.
48 Jacobs, p. 1046.
49 Colpo, p. 24.
50 Zuriek, p 137-143.
51 Horwich, p. 216.
52 Colpo, p. 54.
53 Yudkin, J., "Diet and coronary thrombosis."
54 Ravnskov, p. 25.
55 Ibid., xxiv.
56 Colpo, p. 38.
57 Kendrick, p.53. For quote, see Colpo, p. 42.
58 Ibid., p. 52.
59 Prentice, p. 6.
60 I realize that using the term "American" to describe people who live in the U.S.A. reflects the entitlement of dominance: Canadians and Mexicans also live in America, as do Nicaraguans and Brazilians. The problem is there's no parallel term-"United Statesian" doesn't exist. In previous chapters, I tried to use constructions like "citizens of the US" and "US American." In this chapter, it's

unwieldy to the point of incomprehensibility. I hope someone comes up with a better term soon. Until then, I need the reader to understand me, with apologies.

61 Mann, p. 1.
62 Fallon, *Nourishing Traditions,* p. 246.
63 Schmid, p. 121.
64 Ibid, p. 121.
65 Prior.
66 Kendrick, p .. 71.
67 Colpo, p. 50.
68 Ibid., p. 50.
69 Ibid., p. 49.
70 Eades and Eades, *Protein Power LifePlan,* p. xvi.
71 Ibid., p. xvi.
72 Ibid., p. xviii.
73 Ibid., p. xviii.
74 Ibid., p. xx. See for instance the *American Journal of Clinical Nutrition* March 1998 Supplement.
75 Ibid., p. xxi.
76 Colpo, p. 17.
77 Ibid., p. 51.
78. Ibid., p. 60.
79 Ibid, p. 65.
80 See Christakis et al., "Effect of the Anti-Coronary Club Program" and "The Anti-Coronary Club

BOOK: *THE CHOLESTEROL MYTHS: EXPOSING THE FALLACY THAT SATURATED FAT AND CHOLESTEROL CAUSE HEART DISEASE* BY DR. UFFE RAVNSKOV, MD, PHD

Source: Uffe Ravnskov, MD, PhD



Dr. Ravnskov takes aim at one of the biggest medical myths of our time--that saturated fat and cholesterol cause heart disease--and slays the Goliath with page after page of brilliant analysis. Anyone who has been told to go on a lowfat diet or take cholesterol-lowering drugs should read this book first. . . and then give it to his or her doctor.

Would you buy a book that was literally set on fire by its critics on a television show about it in Finland? I would and so should you. The long-awaited English version of debunker extroidinaire Dr. Uffe Ravnskov's notorious book is now available from New Trends Publishing.

Ravnskov, a medical doctor with a PhD in Chemistry, has had over 40 papers and letters published in peer-reviewed journals criticizing what Dr.

George Mann, formerly of Vanderbuilt University, once called "the greatest scam in the history of medicine": the Lipid Hypothesis of heart disease, the belief that dietary saturated fats and cholesterol clog arteries and cause atherosclerosis and heart disease.

If one thing comes through as you read the book, it is this: Ravnskov has done his homework. In painstaking detail, he critically analyzes and demolishes the nine main myths of the Lipid Hypothesis:

(1) High-fat foods cause heart disease,
(2) High cholesterol causes heart disease,
(3) High fat foods raise blood cholesterol,
(4) Cholesterol blocks arteries,
(5) Animal studies prove the diet-heart idea,
(6) Lowering your cholesterol will lengthen your life,
(7) Polyunsaturated oils are good for you,
(8) The cholesterol campaign is based on good science, and
(9) All scientists support the diet-heart idea.

Equipped with a razor-sharp mind, an impressive command of the literature, and a deadly, needling sarcasm, Ravnskov methodically slaughters the most famous Sacred Cow of modern medicine and the most profitable Cash Cow for assorted pharmaceutical companies. Sparing no one, Ravnskov again and again presents the tenets of the Lipid Hypothesis and the studies which supposedly prove them, and shows how the studies are flawed or based on manipulated statistics that actually prove nothing. Ravnskov then answers the objections or rationalizations offered by diet-heart supporters, desperate to explain away inconsistencies and contradictions in their own data.

For example, Ravnskov opens with an analysis of the study that kicked off the Lipid Hypothesis in the 1950s: Ancel Keys' Six Countries Study (and later, the more famous Seven Countries Study). As most health professionals know, Keys' study showed that countries with the highest animal fat intake have the highest rates of heart disease. Keys' conclusion was that there was a cause and effect relationship because the country with the lowest animal fat intake (at that time, Japan) had the lowest rates of heart disease. Sounds convincing, right? Not so, says Dr. Ravnskov. And in a few pages the reader is informed how Keys hand-picked the countries he included in his studies, namely, the ones that supported his hypothesis, and conveniently ignored all of the other countries that didn't.

And this is just the beginning!

Ravnskov approaches true brilliance in his review of the studies that supposedly showed benefit from the current wonder-drugs pushed by the pharmaceutical industry: the statins. Hailed as miracle substances that "significantly reduce cholesterol and incidence of heart attacks," Ravnskov shows that these substances are probable carcinogens (women on the drugs had a much higher incidence of breast cancer) and that the overall statistical reduction of heart disease in the drug trials is negligible. Nevertheless, despite the dismal results of the very first trial (the EXCEL Trial which Ravnskov soberingly describes to the reader), the industry and its well-funded doctors urge their use, even in people who do not have heart disease.

Ravnskov warns: "Because the latent period between exposure to carcinogen and the incidence of clinical cancer in humans may be 20 years or more, the absence of any controlled trials of this duration

means that we do not know whether statin treatment will lead to . . . cancer in coming decades. Thus, millions of people are being treated with medications the ultimate effects of which are not yet known."

If there is one weakness of the book, it is its lack of explanations of what DOES cause heart disease. Ravnskov comes close to fingering a few factors such as high stress, excessive polyunsaturated fat intake, trans-fatty acids, and smoking, but he never offers his own theory as to what causes the Western world's number one killer.

This is, however, a minor glitch. Ravnskov has done the world a major service in presenting his findings. All health professionals need to listen to this scholar and listen very carefully for the advice offered by the medical establishment for the last 50 years to beat heart disease has failed miserably. It is time to turn away from cholesterol-lowering drugs that have frightening side effects. It is time to turn away from tasteless low-fat diets that harm children and deprive people of fat-soluble vitamins. And it is time to turn away from the junk science that characterizes the Lipid Hypothesis and its supporters. It is time, instead, to listen to reason and view all of the evidence against a failed hypothesis and discover the true and varied risks and causes of heart disease. It is time to listen to Uffe Ravnskov....

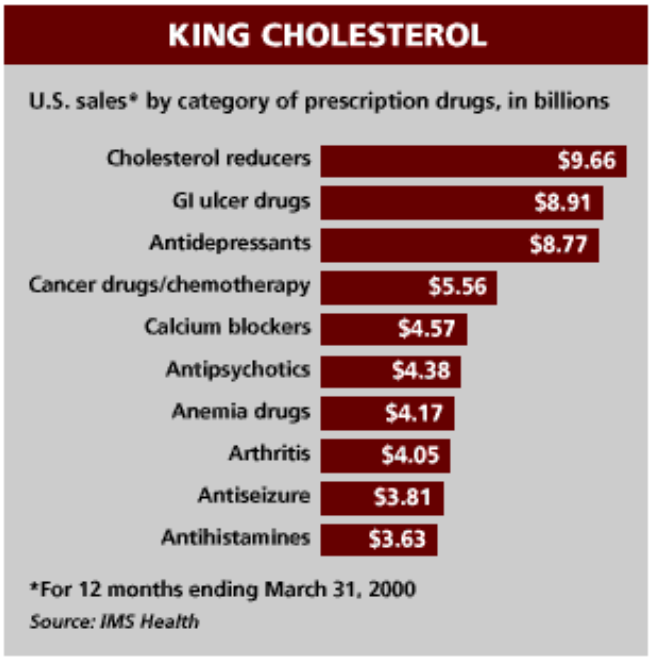
Biography

Uffe Ravnskov was born 1934 in Copenhagen, Denmark. He graduated in 1961 from the University of Copenhagen with an M.D, but has worked most of his time as a clinician and a researcher in Sweden, where he got his PhD from the University of Lund. He has published more than 100 papers and letters critical of the cholesterol campaign; most of them in major medical journals. Honoured by the Skrabanek Award 1999 given by Trinity College of Dublin, Ireland for original contributions in the field of medical skepticism, and by the 2007 Leo-Huss-Walin Prize for Independent Thinking in Natural Sciences And Medicine. He is a member of the editorial board of two medical journals and is the creator and spokesman of THINCS, The International Network of Cholesterol Skeptics (www.thincs.org), an organization that includes more than 100 researchers and other university graduates from all over the world. More details about Uffe Ravnskov are available on www.ravnskov.nu/uffe

IS MONEY INVOLVED?

Source: online research

How much are cholesterol drugs worth to pharmaceutical companies? Cholesterol lowering drugs are the single biggest market for pharmaceuticals!



Wells Fargo & Co Projects a decline in the sales of Cholesterol Drug Sales for Abbott Labs. . .
I wonder what the CEO of Abbott Laboratories will do when faced with shareholder's concerns about the loss of over a billion dollars in revenue.

The Pharmaceutical industry cant really plan and promote the sales of products like other industries can they?

Figure 1 Risk And Compliance Requirements That Pharma Faces Across The Business Cycle

	Product development and approval	Manufacturing and distribution	Marketing and promotion
Scope of compliance/ risk in each business area	Manage research and development to build and diversify drug portfolio. Regulations require firms to document the development process, clinical trials, and safety data.	Validate that processes, facilities, and controls to manufacture, package, and hold a drug meet safety, identity, quality, and purity standards in accordance with approved drug formulation, efficacy profile, and labeling regulation.	Record ongoing consumer safety and report on potential risks and adverse events. Manage brand reputation and avoid expensive litigation. Increase visibility into previous stages to minimize liability/ culpability that the firm may bear.
Pharma-specific compliance and risk examples	• R&D good clinical practices • New compound/product portfolio • Therapeutic/research portfolio • Toxicology • Clinical trials: Phase I-III • Regulatory affairs	• Labeling and annual reporting (SPL, PLR) • Inventory security vaulting • Manufacturing system validation (GMP) • Environmental health and safety • Process and analytical technology (PAT) • Audits/quality control • Sample accountability	• Chain of custody, pedigree, counterfeit/theft prevention • Brand/reputation management • Litigation, preservation holds • Medical affairs • AERS — adverse events safety warnings/reporting • Phase IV trials • Safety signal data mining • Corporate integrity, fraud, and abuse

38030 Source: Forrester Research, Inc.

Graphic source: Pharma Focus Asia Industry Reports
http://www.pharmafocusasia.com/knowledge_bank/industryreports/pharma_risk_managers.htm

VITAMIN D IS SYNTHESIZED FROM CHOLESTEROL AND FOUND IN CHOLESTEROL-RICH FOODS

Source: *http://www.cholesterol-and-health.com/Vitamin-D.html*

One of cholesterol's many functions in the body is to act as a precursor to vitamin D.

Vitamin D can also be obtained from foods. Interestingly, foods that provide this vitamin -- all of which are animal foods -- tend to be high in cholesterol.

Since cholesterol is a precursor to vitamin D, inhibiting the synthesis of cholesterol will also inhibit the synthesis of vitamin D. Since sunlight is required to turn cholesterol into vitamin D, avoiding the sun

will likewise undermine our ability to synthesize vitamin D. And since vitamin D-rich foods are also rich in cholesterol, low-cholesterol diets are inherently deficient in vitamin D.

Vitamin D is best known for its role in calcium metabolism and bone health, but new roles are continually being discovered for it, including roles in mental health, blood sugar regulation, the immune system, and cancer prevention. Yet standard modern advice -- take cholesterol-lowering drugs, avoid the sun, eat a low-cholesterol diet -- combined with a recommended daily intake of vitamin D that is only a tenth of what many researchers believe to be sufficient all seems to pave the way for widespread vitamin D deficiency.

Perhaps that's why, according to Dr. John Cannel, President of the Vitamin D Council, most whites and nearly all blacks in modern society are deficient in vitamin D.[1]

Sources of Vitamin D: Synthesis of Vitamin D in the Skin by Sunlight

When sunshine in the UV-B spectrum strikes the skin, it converts a substance in the skin called 7-dehydrocholesterol into vitamin D_3.[2]

7-dehydrocholesterol is a very close precursor to cholesterol. If you look at our flow chart showing the synthesis of cholesterol, you will see that it shows lanosterol being converted directly to cholesterol. This conversion is actually believed to take more than 18 different steps and hasn't been completely figured out, so it is usually simplified as one step.[3] 7-dehydrocholesterol occurs very close to the end of this conversion, so is often referred to as "cholesterol" or "a form of cholesterol."

Figure 1: The Chemical Structure of 7-Dehydrocholesterol

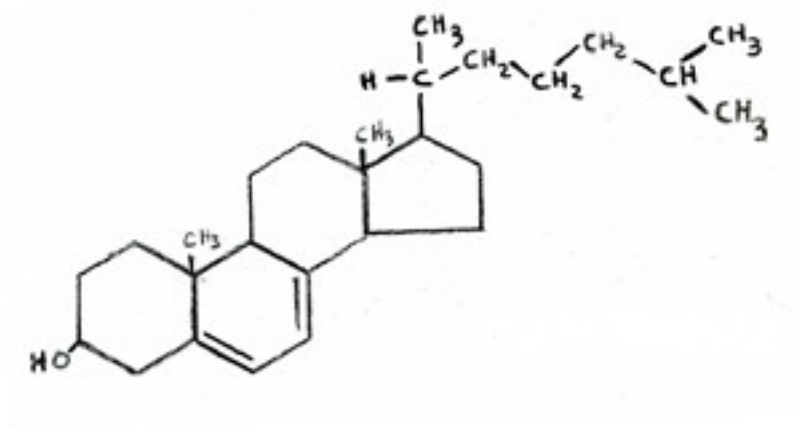
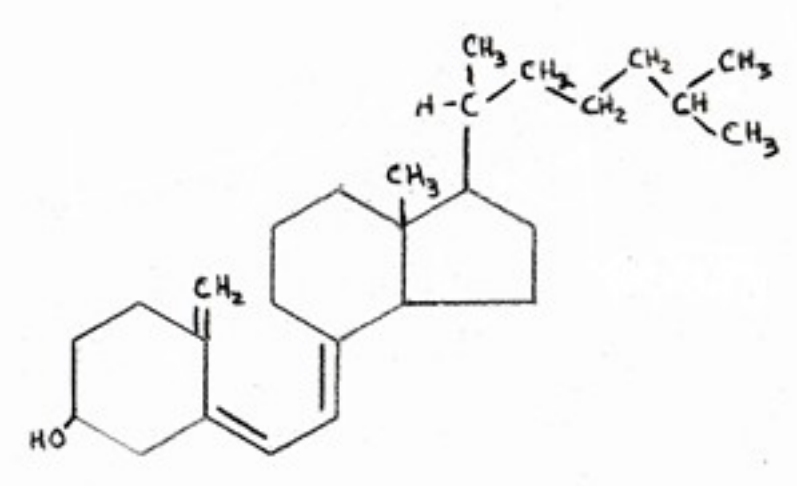


Figure 2: The Chemical Structure of Vitamin D



When atmospheric conditions are ideal and skies are clear, 30 minutes of whole-body exposure of pale skin to sunlight without clothing or sunscreen can result in the synthesis of between 10,000 and 20,000 IU of vitamin D. These quantities of vitamin D are large, and therefore capable of supplying the body's full needs.[2]

At the same time, the body has two mechanisms to prevent an excess of vitamin D from developing: first, further irradiation converts excess vitamin D in the skin to a variety of inactive metabolites; second, the pigment melanin begins to accumulate in skin tissues after the first exposure of the season, which decreases the production of vitamin D.[2]

The availability of UV-B rays, however, depends on the angle at which sunshine strikes the earth, making vitamin D synthesis impossible for most people at most latitudes during parts of the year called the "vitamin D winter."[4]

Outside the vitamin D winter, sufficient UV-B rays for full vitamin D synthesis do not suddenly become available: the window of time during each day in which vitamin D synthesis can occur gradually expands as the season progresses, as does the amount of UV-B radiation available within that window.[4]

Many different factors can make the availability of UV-B widely variable during any given time of the year. Clouds alone, for example, can eliminate up to 99 percent of UV-B radiation.[5]

Natural variations in the density of the ozone layer can cause the length of the vitamin D winter to increase or decrease by up to two months. Aerosols and buildings block UV-B radiation, while increased altitude or reflective surfaces such as snow increase exposure to UV-B radiation.[5]

In the past, researchers suggested that any place outside of 34 degrees latitude experiences some degree of vitamin D winter, that the vitamin D winter in Boston extended for four months from November through February, and that the vitamin D winter in Edmonton extended for six months from October through March.[5]

More recently, researchers found that so many factors influence the availability of UV-B light that vitamin D winters under some conditions in Boston and Edmonton could be much shorter, whereas under other conditions, vitamin D winters can even occur at the equator.[5]

Since most of us live at latitudes that are covered by a vitamin D winter for at least part of the year, and since most of us work indoors and wear clothing and sunblock when outdoors in the summer sun, it is necessary for most of us to consume vitamin D in food for at least part of the year, or to supplement with vitamin D.

In order to consume vitamin D as food, we must eat the cholesterol-rich animal foods we are so often told to avoid.

Sources of Vitamin D: Foods High in Vitamin D Are High in Cholesterol

By far the richest source of dietary vitamin D is cod liver oil -- a substance that takes the honor of being the food second richest in cholesterol. At 5.7 milligrams of cholesterol per gram of food, cod liver oil beats out its nearest competitor -- chicken liver -- by 0.09 mg/g, and is one third richer in cholesterol than the notorious egg. It is second only to the expensive delicacy of caviar, which comes in at 5.9 mg of cholesterol per gram.

The second richest source of vitamin D is lard. No, you didn't read that wrong -- lard. Lard ranks #18 on our list of the top 22 foods richest in cholesterol, and is over four times richer in vitamin D than its nearest competitor, herring. Granted, the pigs need to be exposed to sunlight to generate vitamin D.

Other sources of vitamin D include fatty fish, some shellfish, egg yolks, and butter -- foods selected almost entirely from the list of those richest in cholesterol.

The table below shows the overlap between foods' status as cholesterol-rich and vitamin D-rich.

Table: Dietary Sources of Vitamin D_3[6]

Food	Cholesterol per 100 g	Vitamin D per 100 g
Cod liver oil	570 mg*	10,000 IU (up to 25,555 IU)
Herring	12.9 mg	680 IU
Oysters	54 mg	642 IU
Catfish	81 mg	500 IU
Sardines	142 mg	480 IU
Mackerel	95 mg*	450 IU
Salmon	87 mg	320 IU
Caviar	588 mg*	232 IU
Shrimp	173 mg	172 IU
Butter	218 mg	56 IU
Whole Egg (contained in Yolk only)	424 mg	49 IU
All Plant Foods	0 mg	0 IU

The correlation between cholesterol content and vitamin D content certainly isn't perfect, but all of the foods that contain vitamin D contain substantial amounts of cholesterol, and most of the foods high in vitamin D are quite high in cholesterol.

Sources of Vitamin D:
Do Plant Foods and Mushrooms Really Contain Vitamin D?

Many "vitamin D" supplements contain vitamin D_2, which is obtained by subjecting ergosterol, a chemical found in plants, to radiation.

Recently, news reports declared that mushrooms subjected to ultraviolet radiation are "[zapped] into a giant serving" of vitamin D.[7] Like plants, any vitamin D contained in mushrooms is in the form of vitamin D_2.[8]

Should sources of vitamin D_2 really be considered sources of vitamin D?

Some researchers claim that vitamin D_2, also called "ergocalciferol" and called "viosterol" in the old days, and vitamin D_3, also called "cholecalciferol" are equally effective in humans because their ability to bind to the vitamin D receptors in our cells is equal.[2] But that's not the whole story.

Figure 3: The Chemical Structure of Vitamin D3 (Cholecalciferol)

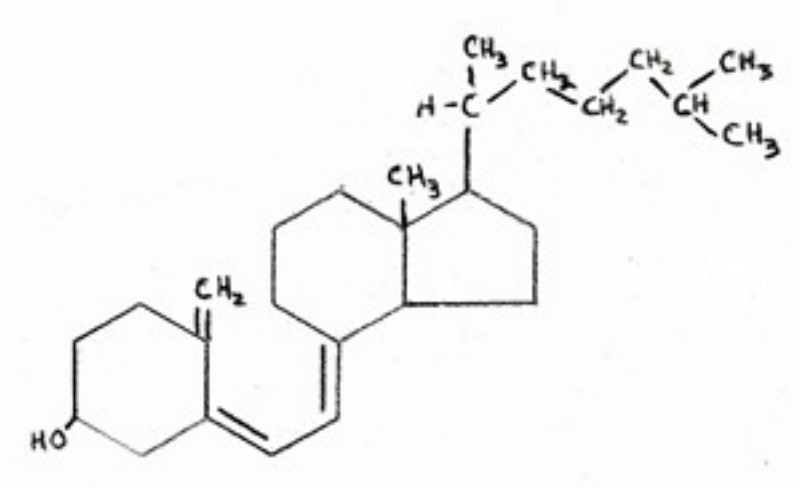
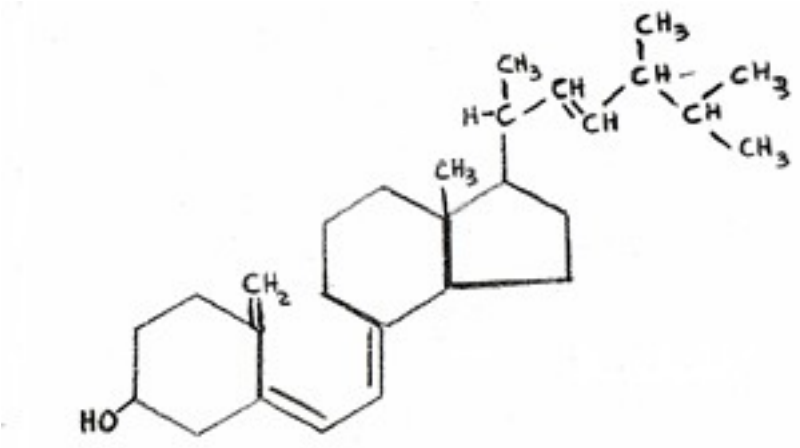


Figure 4: The Chemical Structure of Vitamin D2 (Ergosterol)



Vitamin D is carried in the blood by vitamin D-binding protein (DBP). DBP is kind of like a savings account for vitamin D. If you didn't have the DBP, you'd be forced to use all your vitamin D as soon as you absorb it, and excrete the rest. This would be a giant waste of vitamin D, because you can only use so much at a time. DBP thus helps to increase the effect of a given dose of vitamin D by holding on to what you don't need at any given moment for later use, and helps prevent toxicity by keeping the portion you don't need at any given moment from being delivered to your cells.[2]

Although vitamin D_2 binds well to the vitamin D receptor, it has very little affinity for vitamin D-binding protein. For this reason, it is well-known to be useless in chickens and other birds. When vitamin D was seen merely as a cure for rickets, vitamin D_2's ability to treat rickets in the small amounts needed led researchers to believe it equal in power to vitamin D_3 in humans. Now that researchers are uncovering the need for much higher levels of vitamin D to maintain optimal health, it is becoming clear that vitamin D_2 just doesn't fit the bill.

The researchers Laura Armas, Bruce Hollis, and Robert Heaney showed in 2004 that vitamin D_2's low affinity for the vitamin D-binding protein makes it nearly ten times less effective at raising long-term vitamin D levels.[9]

If vitamin D_2 has a lower affinity for the DBP, it follows that it is also much more likely to result in toxicity than is vitamin D_3. It is therefore unsurprising that, according to Dr. John Cannel, president of the Vitamin D Council, nearly *all* cases of toxicity from pharmacological doses of vitamin D resulted from the consumption of vitamin D_2.[10]

The vitamin D_2 synthesized from plant sterols should therefore not be considered true vitamin D for humans. Humans should obtain vitamin D from the sun and from the vitamin D-rich fatty animal foods that provide the form of vitamin D with which the sun provides us, and which we have consumed throughout our evolution.

The Many Functions of Vitamin D: More Than Just Bone Health and Calcium

Vitamin D is best known for its role in calcium metabolism, especially for its role in the treatment of rickets. Yet modern science is discovering that it has many more important roles, from mental health and immunity to blood sugar regulation and cancer prevention. In fact, there is some evidence that

the vitamin D found in those notoriously "artery-clogging" cholesterol-rich foods may help prevent heart disease.

Cod liver oil was first used as a therapeutic agent in the 1770's, and by the mid-1800's it was well-recognized as a cure for the childhood bone disease, rickets, which is marked by an expansion of the bone's metaphyseal plate and a buildup of unmineralized bone matrix, and its adult equivalent, osteomalacia. Upon the discovery of vitamin A in 1913 as a component of cod liver oil and butterfat, researchers assumed vitamin A to be responsible for cod liver oil's ability to cure rickets. In 1921, however, a team of researchers established that there was a separate component of cod liver oil responsible for its ability to cure rickets, which came to be known as vitamin D.[11]

In the early 1970's researchers discovered that vitamin D is actually activated into an endocrine hormone within the body, and over the course of the 1980s researchers discovered the vitamin D receptor, which is a nuclear receptor for vitamin D that enters the nucleus and alters the expression of genes once vitamin D binds to it.[12]

Since then, the scope of vitamin D research has broadened immensely. The Vitamin D Council, headed by Dr. John Jacob Cannel, MD, has compiled information on vitamin D's role in the following areas:

- Autoimmune illness
- Cancer
- Chronic pain
- Diabetes
- Heart disease
- Hyperparathyroidism
- Hypertension
- Mental illness
- Multiple sclerosis
- Muscle weakness and coordination
- Obesity
- Osteoarthritis

Dr. Cannel has proposed that a variety of the above conditions be used as indicators for "Vitamin D Deficiency Syndrome," and urges that physicians test the vitamin D levels of patients who exhibit those indicators and treat patients with deficient levels with sunlight or vitamin D supplementation.

Vitamin D Deficiency: Do Cholesterol-Lowering Statin Drugs Inhibit Vitamin D Synthesis?

Researchers know that vitamin D synthesis declines with age -- and so does the concentration of 7-dehydrocholesterol in the skin. Without 7-dehydrocholesterol in the skin, sunlight has nothing to turn into vitamin D. The researchers consider it likely, then, that the decreased synthesis of 7-dehydrocholesterol is responsible for the decreased synthesis of vitamin D that comes with age.[2]

It follows then, that the cholesterol-lowering drugs known as statins, or HMG CoA reductase-inhibitors, which inhibit the synthesis of 7-dehydrocholesterol, also inhibit the synthesis of vitamin D.

(View our flow chart of the cholesterol synthesis pathway here.)

As of May 25, 2006, there are no studies indexed for Medline that tested the effect of statins on vitamin D levels for longer than three months, and only one, single study out of three that tested the effect of statins on vitamin D levels for longer than one month -- conducted a whopping fifteen years ago. The small handful of short-term studies found no effect.[13]

By contrast, researchers who showed that statins induce dramatic deficiencies of coenzyme Q10 in humans first retested coenzyme Q10 levels after six months of administering the statin. They further found that coenzyme Q10 levels kept decreasing over time for over 18 months before settling.[14]

We would expect statins to take even longer to cause a drop in vitamin D levels, because, whereas coenzyme Q10 is measured directly in the blood, the 7-dehydrocholesterol takes time to migrate to the surface of the skin and accumulate there. So what is the effect of statins on vitamin D levels one year down the road? Two years? Five? Ten?

The truth is we have no idea, because no one has bothered to study it.

Vitamin D Requirements: How Much Do We Need?

Although the U.S. RDA for vitamin D is a mere 400 IU, modern research is showing that much higher levels are needed to maintain adequate vitamin D status for optimal health.

After vitamin D is obtained from the diet or absorbed from the skin, it is semi-activated to 25 (OH) D or 25-hydroxyvitamin D in the liver. This is the primary storage form of the vitamin that is carried by vitamin D-binding protein in the blood. As needed, 25 (OH) D in the blood is fully activated to 1, 25 $(OH)_2$ D or 1, 25-dihydroxyvitamin D -- called calcitriol -- in the kidney primarily in response to low calcium levels.

The skin also has the ability to produce small amounts of activated vitamin D, and most of the tissues in the body have the capacity to activate vitamin D when stimulated to do so by the immune system -- completely separate from the calcium-regulated system that governs vitamin D activation in the kidney.[2]

It is the 25 (OH) D form of vitamin D that is considered the valid test of one's nutritional vitamin D status. If you ask your doctor for a vitamin D test, make sure your doctor orders the 25 (OH) D test and not the 1, 25 $(OH)_2$ D test.

Figure 5: The Chemical Structure of Vitamin D

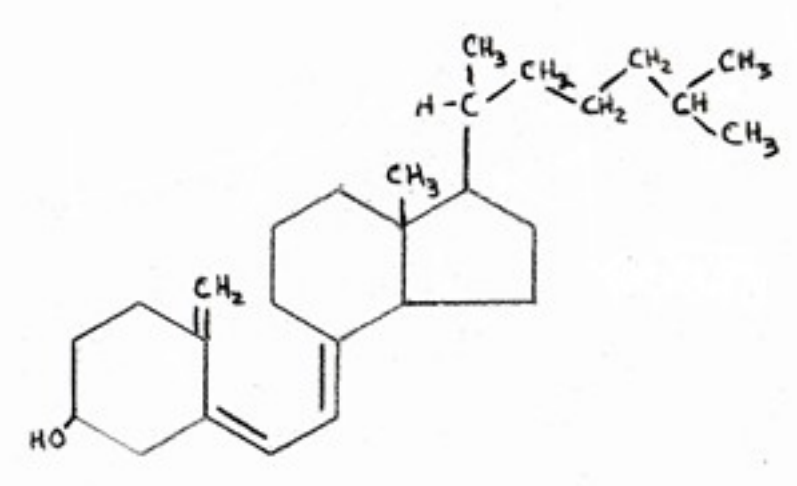


Figure 6: The Chemical Structure of 25 (OH) D

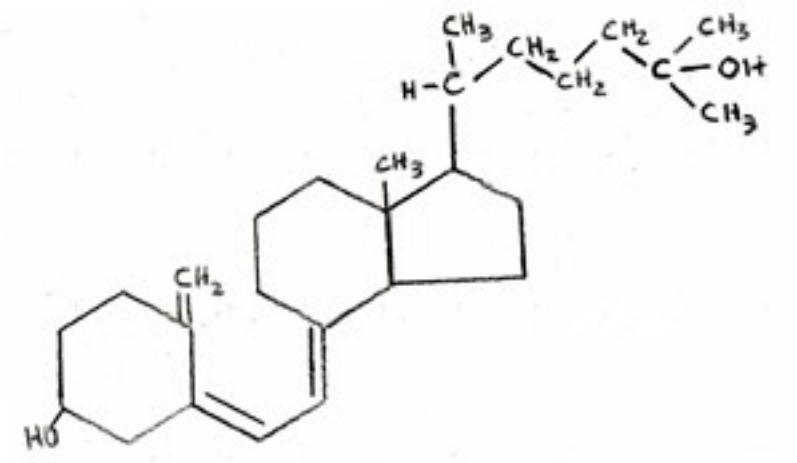
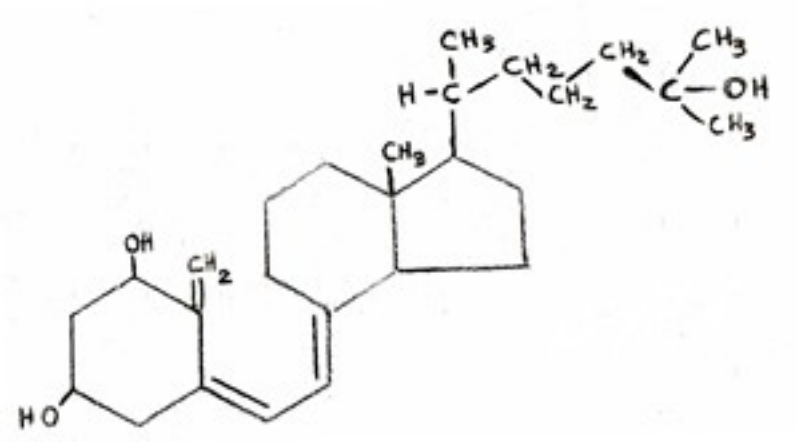


Figure 7: The Chemical Structure of 1, 25 $(OH)_2$ D



In order to maximize calcium absorption, blood levels of 25 (OH) D need to be maintained at 30 ng/mL, which, in the absence of UV-B light, would require roughly 2600 IU per day of vitamin D. The risk of fracture continues to decline at even higher 25 (OH) D levels because people with higher

vitamin D levels actually *fall less often,* suggesting a neuromuscular benefit to high levels of the vitamin. Even higher levels of 46 ng/mL are needed to maximize the body's ability to regulate blood sugar, and since dark-skinned agricultural workers in the tropics have 25 (OH) D levels of about 60 ng/mL, it's probable that this is closer to the ideal level.[15]

The researcher Robert Heaney showed that people use about 4,000 IU of vitamin D per day when they are running on storage deposits of the vitamin,[15] suggesting that this is the amount we should be consuming from food or supplements during a vitamin D winter.

Since many of us do not get full-body skin exposure even during the summer, and wear sunscreen when we do, it is probably ideal for most of us to consume some lesser amount of vitamin D even during the warmer months.

Dr. Cannel of the Vitamin D Council recommends testing your 25 (OH) D levels periodically if you consume more than 2000 IU of vitamin D just to be on the safe side. He says he personally takes 5,000 IU during the winter, 2,000 IU during the early spring and late fall, and none in the months in between.[16]

The Toxicity of Vitamin D

In 1997, the Food and Nutrition Board of the U.S. Institute of Medicine set the limit for the amount of vitamin D that was guaranteed safe to take at 2000 IU per day. According to the vitamin D researcher Robert Heaney, "the vitamin D content experts on the Upper Limits Panel objected to this 2000 IU/day figure on the grounds that extensive clinical experience had established the safety of substantially higher inputs," and several other investigators have since called for the Institute of Medicine to raise the upper limit.[15]

According to Heaney, extensive sun exposure produces vitamin D levels equivalent to taking 10,000 IU per day, from which no dangers have ever been observed.

Worse, the upper limit set by the Institute of Medicine is lower than the dose that many people need to take simply to gain sufficient levels of vitamin D. "It would be difficult," wrote Heaney, "to make a policy recommendation to provide the amounts needed for a major sector of the population in the face of [an upper limit] that remained as low as 2000 IU."[15]

The vitamin D researcher Reinhold Veith wrote a review in 1999 asserting the safety of 10,000 IU of vitamin D per day and challenging the medical community to produce any evidence for the widespread assumption that doses under 10,000 IU could be toxic.[10]

In 2001, Ian Monroe, the chair of the Institute of Medicine's committee on vitamin D toxicity wrote in praise of Veith's work and promised that it would be taken into account at a future meeting of the Institute, but this has yet to happen.

Dr. Cannel searched the literature for case reports of toxicity from pharmacological doses of vitamin D_3. Although he found cases of vitamin D_2 toxicity, he found only one, single case of vitamin D_3 toxicity: one man took vitamin D supplements for two years that were mislabeled, containing up to 430 times the amount of vitamin D as was listed on the label. He took between 156,000 IU and

2,604,000 IU per day for those two years, and "recovered uneventfully after the proper diagnosis, treatment with steroids and sunscreen."[10]

(You can read his report on vitamin D toxicity here.)

Vitamin D Safety — Make Sure You Get Your Vitamin A

Animal studies show that vitamins D and A each protect against toxic effects of the other -- suggesting that vitamin toxicity might be more a result of vitamin imbalance than vitamin excess.

In humans, supplementation with vitamin D appears from case reports to allow the average 75-kg human to take an *additional* 175,000 IU of vitamin A per day before vitamin A toxicity symptoms begin to develop.[17]

Vitamin D increases the need for vitamin A in chickens even in small amounts that are insufficient to guarantee freedom from rickets. One study showed that massive doses of vitamin A alone caused bone and growth problems in turkeys, while massive doses of vitamin D alone caused kidney problems -- yet when these doses were combined, the turkeys exhibited no signs of toxicity at all.[17]

Since most foods that are high in vitamin D are also high in vitamin A, it makes sense to either consume those foods that provide both, or, if one is to take high doses of vitamin D, to also supplement with vitamin A -- or to take a supplement that provides both, such as cod liver oil.

Likewise, if one is eating vitamin A-rich foods or taking vitamin A supplements, one should also make sure one is getting enough vitamin D.

Without Cholesterol, There Is No Vitamin D

Cholesterol's connection to vitamin D is intimate. It is the synthesis of cholesterol that ultimately provides for the synthesis of vitamin D, and it is cholesterol-rich foods that provide the dietary sources of vitamin D during times of the year when it is impossible for us to make our own.

Vitamin D is not *one* more reason to consider cholesterol good for the body. The days when vitamin D is seen as a unifunctional vitamin reponsible simply for calcium metabolism and bone health are quickly disappearing into the annals of history. Vitamin D is quickly being recognized as a hormone with myriad important roles in the body -- and thus credit for each of those benefits of vitamin D falls back on its close relative and precursor, cholesterol.

The lesson? Lots of sunshine and cholesterol-rich foods are good for you. If you skip the cholesterol and take vitamin D supplements, make sure they are vitamin D_3 and not vitamin D_2 -- and you can thank our favorite molecule that provided the precursor for the vitamin D in your supplement.

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CHOLESTEROL'S IMPORTANCE TO THE CELL MEMBRANE

Source: by Chris Masterjon http://www.cholesterol-and-health.com

Cholesterol is Abundant in Cell Membranes

Cholesterol is found in every cell of your body. It is especially abundant in the membranes of these cells, where it helps maintain the integrity of these membranes, and **plays a role in facilitating cell signaling-- meaning the ability of your cells to communicate with each other so you function as a human, rather than a pile of cells.**

Molecule for molecule, cholesterol can make up nearly half of the cell membrane.[1] Since it is smaller and weighs less than other molecules in the cell membrane, it makes up a lesser proportion of the cell membrane's mass, usually roughly 20 percent.[2]

Cholesterol is also present in membranes of organelles inside the cells, although it usually makes up a smaller proportion of the membrane. For example, the mitochondrion, the so-called "power-house" of the cell, contains only three percent cholesterol by mass, and the endoplasmic reticulum, which is involved in making and modifying proteins, is six percent cholesterol by mass. [3]

Cholesterol Maintains the Integrity of the Cell Membrane

Surrounding each of our cells is a membrane called the *plasma membrane*. The plasma membrane is a continuous double-layer of *phospholipids*, interweaved with cholesterol and proteins. *Phospholipids* are composed of two fatty acids attached to a phosphate compound as a head.

The phosphate head is water-soluble, also called "hydrophilic" (water-loving), and the fatty-acids are water-insoluble, or "hydrophobic" (water-fearing). Since outside the cell is a water-containing, or aqueous, environment, and inside the cell is also aqueous, the phosphate heads of the phospholipids face both the cell's inside and the environment outside the cell, while the fatty acids face the inside of the membrane.

The membrane is fluid, and the molecules are always moving. It has about the same consistency as olive oil.

Cholesterol is an *amphipathic molecule*, meaning, like phospholipids, it contains a hydrophilic *and* a hydrophobic portion. Cholesterol's hydroxyl (OH) group aligns with the phosphate heads of the phospholipids. The remaining portion of it tucks into the fatty acid portion of the membrane.

Because of the way cholesterol is shaped, part of the steroid ring (the four hydrocarbon rings in between the hydroxyl group and the hydrocarbon "tail") is closely attracted to part of the fatty acid chain on the nearest phospholipid. This helps slightly immobilize the outer surface of the membrane and make it less soluble to very small water-soluble molecules that could otherwise pass through more easily.[4]

Without cholesterol, cell membranes would be too fluid, not firm enough, and too permeable to some molecules. In other words, it keeps the membrane from turning to mush.

Cholesterol Helps Maintain the Fluidity of Cell Membranes

While cholesterol adds firmness and integrity to the plasma membrane and prevents it from becoming overly fluid, it also helps maintain its fluidity.

At the high concentrations it is found in our cell's plasma membranes (close to 50 percent, molecule for molecule) cholesterol helps separate the phospholipids so that the fatty acid chains can't come together and cyrstallize.[5]

Therefore, cholesterol helps prevent extremes-- whether too fluid, or too firm-- in the consistency of the cell membrane.

Cholesterol Helps Secure Important Proteins in the Membrane

The plasma membrane contains many proteins that perform important functions like channeling or pumping substances into and out of the cell, attaching to other cells, forming borders to keep other proteins in one specific part of the cell, communicating with nearby cells, or responding to endocrine hormones from far-away cells.

Because certain proteins' size or shape requires a thicker phospholipid bed to sit in, and because certain proteins need to stick together to function properly, the fluidity of the cell membrane, where the molecules are constantly moving randomly, could pose a problem.

Fortunately, the plasma membrane contains many *lipid rafts* where proteins are secured. A lipid raft contains high concentrations of cholesterol and *sphingolipids*-- a type of phospholipid-- containing longer and more saturated fatty acid tails.

Because the fatty acids are longer and more saturated (straighter), they aggregate more, which cholesterol also helps. That part of the membrane is also thicker, making it ideal for accommodating certain proteins.[6]

Since the fatty acids in lipid rafts are longer, the phospholipids also move in sync with the phospholipids on the other side of the membrane.

In the rest of the membrane, the phospholipids on one side of the membrane move independently of those on the other.[7]

By stabilizing certain proteins together in lipid rafts, cholesterol is important to helping these proteins maintain their function.

This could range from forming blood clots or thinning blood, to allowing sugar into your cells, to burning fat, to regulating calcium in your blood, and literally includes, in some way, most of the functions in your body, although which proteins exist in lipid rafts and which do not is still being researched.

It is the proteins, after all, by which cells communicate with one another. If cells didn't communicate with one another, you and I would be a large pile of unrelated cells rather than the individuals that we are.

CHOLESTEROL IS A PRECURSOR TO BILE ACIDS

Source: by, Chris Masterjohn ***http://www.cholesterol-and-health.com/Bile-Acids.html***

The human body uses cholesterol to synthesize bile acids, which are important for the digestion of fats. The primary bile acid, cholic acid, is very similar in structure to cholesterol. Cholic acid is missing the double bond in the second ring, has two more hydroxyl (OH) groups attached to the steroid ring structure, and has a shortened hydrocarbon tail, the ending of which has been converted to a **carboxyl (COOH) group**.

Bile Acids Are Emulsifying Agents

Bile acids are **amphipathic**. This means that they have both water-soluble and water-insoluble (or fat-soluble) parts. **Emulsifying agents** are amphipathic molecules that are able to mix fats with water. For example, eggs contain an amphipathic substance called **lecithin** that makes them useful as emulsifying agents in baking.

In order for the human digestive system to digest fats, they must be emulsified into the digestive juices, because the enzymes that break them down are water-soluble.

In bile acids, the hydroxyl (OH) groups are water-soluble, and the methyl (CH_3) groups are fat-soluble. The hydroxyl groups all face one direction — for example, toward you from the picture above — while the methyl groups face the opposite direction — for example, away from you from the picture above — making one side of the bile acid water-soluble and the other side fat-soluble.

This characteristic allows bile salts to break up large globs of fat, connecting to the fat on one side, and connecting to the water on the other, thus mixing the fats and water together.

Synthesis and Storage of Bile Acids

Bile acids are synthesized from cholesterol in the liver. First, hydroxyl (OH) groups are inserted at several points, shown in the above picture; second, the second ring of cholesterol loses its double-bond; finally, the hydrocarbon tail is shortened by three carbons, and a carboxyl group is added to the end.

The bile salt shown above is called cholic acid, which contains three hydroxyl groups. The other primary bile acid is called chenodeoxycholic acid, which contains only two hydroxyl groups. These are the "primary" bile acids, although there are other "secondary" bile acids synthesized from primary bile acids by intestinal bacteria as well.

Bile acid synthesis is up-regulated by cholesterol and down-regulated by cholic acid. This means that the higher the cholesterol to cholic acid ratio is, the faster bile acids will be produced. As bile acids are produced, and the concentration of cholesterol lowers and the concentration of cholic acid rises, bile acid synthesis slows down.

Bile Acids are Precursors to Bile Salts

Before bile acids leave the liver, they are converted to bile salts. This involves the replacement of the hydrogen on the end of the carboxyl group with either the amino acid glycine or the amino acid taurine. There are four primary bile salts formed from this reaction:

- Glycine + Cholic Acid --> Glycocholic Acid
- Glycine + Chenodeoxycholic Acid --> Glycochenodeoxycholic Acid
- Taurine + Cholic Acid --> Taurocholic Acid
- Taurine + Chenodeoxycholic Acid --> Taurochenodeoxycholic Acid

At the pH (a measure of acidity determined by the concentration of hydrogen ions in a solution) normally present in intestinal digestive juices, the glycine and taurine completely separate from the bile acids within the bile salts. On the other hand, bile acids alone, if not converted to bile salts, will contain a hydrogen ion that tends to stick to the carboxyl group.

Since the carboxyl group is more water-soluble when the hydrogen ion or amino acid is separated from it, bile salts, which have amino acids that completely separate from the carboxyl group, are more water-soluble than bile acids, which have a hydrogen ion that likes to stick to the carboxyl group.

This makes bile salts more effective than bile acids at mixing fats with water. Thus, bile salts are more effective at mixing fats with the water-soluble enzymes that digest them.

Glycine forms of bile salts outnumber taurine forms of bile salts by 3 to 1. After bile salts are produced in the liver, they either flow through the bile duct into the duodenum, which is the first of three sections of the small intestine, to be used immediately for digestion, or they are stored in the gall bladder, where they are saved for future digestive requirements.

Circulation of Bile Salts

Bile salts are produced in the liver, and secreted through the bile duct into the duodenum, the first section of the small intestine. 95% of bile salts are reabsorbed through the ileum, the third and final

part of the small intestine, where they travel through the blood, attached to a blood protein called "albumin," back to the liver. This circulation is called **enterohepatic circulation**.

About 15 to 30 grams of bile salts are circulated through this sequence each day, while about 0.5 grams are lost in the feces and about 0.5 grams are synthesized anew by the liver.

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HOW THE BODY USES CHOLESTEROL

Source: ***http://health.howstuffworks.com/diseases-conditions/cardiovascular/cholesterol/how-the-body-uses-cholesterol.htm***

As a vital part of the body's chemistry, cholesterol is used to produce the steroid hormones required for normal development and functioning. These include the sex hormones estrogen and progesterone in women and testosterone in men. These hormones trigger development of the physical traits characteristic of adult women and men; they also play a role in reproduction.

Other steroid hormones produced from cholesterol include cortisol, which is involved in regulating blood-sugar levels and defending the body against infection, and aldosterone, which is important for retaining salt and water in the body. The body can even use cholesterol to make a significant amount of vitamin D, the vitamin responsible for strong bones and teeth, when the skin is exposed to sunlight.

Cholesterol is also used to make bile, a greenish fluid that is produced by the liver and stored in the gallbladder. The body needs bile to digest foods that contain fat. Bile acts as an emulsifier -- it breaks down large globules of fat into smaller particles so they can mix better with the enzymes that digest fat.

Once the fat is digested, bile helps the body to absorb it. The presence of bile in the intestines is required before cholesterol can be absorbed from foods.

The body also needs bile in order to absorb vitamins A, D, E, and K, called fat-soluble vitamins, from food or supplements.

Your body has the ability to make all the cholesterol it needs for these various functions. A diet that contains animal products, however, also supplies cholesterol to the body. In an effort to balance these two sources of cholesterol, your body adjusts the amount it produces each day.

For example, if you eat many foods from animal sources, your body gets a substantial dose of cholesterol from the diet, called dietary cholesterol; your body then slows down its own production of cholesterol. On the other hand, when most of the foods you eat come from plant sources, your body manufactures more cholesterol in order to meet its needs.

Your body can also eliminate some excess cholesterol through bile. Whenever bile is released into the intestine, a portion of it is absorbed back into the body to be used again. The remaining bile is excreted in the feces. To help maintain the cholesterol balance, the body can dissolve excess cholesterol in the bile and can also convert more cholesterol into bile acids so that the cholesterol will be excreted with the feces.

TESTOSTERONE BUILDING FOR MEN: THE TIM FERRIS APPROACH

Source: *The Four Hour Body* by Tim Ferris

PROTOCOL #2: SHORT-TERM AND FUN "NITRO BOOST"

20–24 Hours Prior to Sex Eat at least 800 milligrams of cholesterol (example: four or more large whole eggs or egg yolks) within three hours of bedtime, the night before you want to have incredible sex.

The Wolverine intro to this chapter was partially thanks to two ¾-pound rib-eye steaks the night before, but it's easier to stomach hard-boiled eggs.

Why before bed? Testosterone is derived from cholesterol, which is primarily produced at night during sleep (between midnight and 4:00–6:00 A.M.).

Four Hours Prior to Sex
4 Brazil nuts
20 raw almonds
2 capsules of the above-mentioned fermented cod/butter combination

SHBG - The Party Spoiler

Sex-hormone binding globulin (SHBG) is the party spoiler.

SHBG binds to testosterone16 and renders it inert for our purposes, and "total testosterone" in blood tests can therefore be misleading. Some vegans have been shown to have higher testosterone levels than both meat-eaters and vegetarians, for example, but higher levels of SHBG cancel out this advantage. In other studies, consumption of cholesterol has been shown to be inversely correlated with SHBG. In other words, the more cholesterol you eat, the less SHBG you have.

From Carruthers's *Androgen Deficiency in the Adult Male: Causes, Diagnosis, and Treatment:*

Strict low cholesterol diets have been shown to lower total and free testosterone levels by 14%. Vegetarian diets, especially if low in protein, can increase SHBG, further reducing FT [free testosterone]. However, men put on a low-fat, high-fiber,

vegetarian diet have a 18% reduction in both total testosterone and FT, which is reversed when they go back on a normal diet…. *Conversely, high-protein, low-carbohydrate diets, such as the fashionable weight-reduction Atkins diet, may partly exert their slimming action by raising total testosterone and lowering SHBG.*

SHBG isn't evil, and we don't want to eliminate it, but a little less SHBG equals a little more free testosterone. This isn't evil either. It actually makes life much more interesting. This is the reason for our cholesterol-loading in Protocol 2.

Testosterone-Building Shake by Tim Ferris in *The Four Hour Body*

It appears that splitting up the 800 milligrams of cholesterol also works for the earlier "Nitro Boost" Protocol 2.

If you know your local sources and can avoid salmonella and raw milk issues**, I've found the following shake to produce incredible effects when mixed with a hand blender and consumed at both 4:00 P.M. and before bed.** It also helped me achieve the 100+-pound strength gains as detailed in "Effortless Superhuman":

12 oz whole raw milk
4 tbsp raw almond butter
2 raw egg yolks
3 tbsp chia seeds
1 tsp vanilla extract
½ tsp cinnamon

This is more appropriately called a "fat shake" instead of a protein shake, but I still dropped bodyfat while consuming it. How? The fat-loss was predicated on otherwise maintaining a slow-carb diet and taking the shakes only on workout days, no more than three times a week. If you've ever wondered what anabolics feel like, one week of these shakes will give you a good idea.

Here is the nutritional yield, with all percentages of USRDA (recommended daily allowance):

Total calories = 966
Calories from fat = 627
Fat grams = 73 g (113%)
Saturated fat grams = 15 g (76%)
Cholesterol = 456 g (152%)
Protein = 34 g (69%)
Carbohydrate grams = 55 g (18%)
Dietary fiber = 20 g (81%)
Sugars = 19 g
Calcium = 93%
Glycemic load = 15 (out of maximum 250)

Know your food sources, and the statistics on salmonella poisoning, etc., before consuming this. If raw

milk scares you, and I wouldn't blame you, use organic whole milk instead.

A Post-Vegan Experience and Perspective on Cholesterol and Animal Products

Source: Bert Grosman · Top Commenter · Journeyman at IATSE Local 200

Response to this comment (on this article):
http://www.naturalnews.com/036708_Alzheimers_type-3_diabetes_brain_disease.html

> *David Raymond*
> *Uhmm.. I have researched holistic/vegan diets for quite some time, and from my studies, our liver produces more than enough cholesterol in our bodies, we do not need to take in more of it.. But one thing I know for sure, a strict holistic/vegan diet has proven to reverse type-2 diabetes, lower blood sugar & lower cholesterol.. I suggest a holistic diet to cure almost all illnesses & diseases..*

David Raymond, **The problem is that your Liver has to work harder when it constantly has to make cholesterol. That is why your Liver recycles cholesterol instead of eliminating thru the kidneys.**

Plus, when the Liver is forced to make cholesterol due to lack of it in the diet, this cholesterol is made up from carbohydrates, PUFAS and or sugar and therefore an inferior type of cholesterol which is often oxidized. Saturated fats such as found in coconut oil and lard do not oxidize when heated whereas vegetable oils do. When these rancid oils are ingested they become free radicals in the body which further damages the endothelium which lines the arterial walls.

I was a vegan/vegetarian/macrobiotic and eventually pescatarian for 27 years. Followed the low-fat philosophy even after I started having medical issues, blindly following because I was essentially afraid to get off the "Band Wagon!"

Cut to the chase, I feel soooooooo much better since I changed my diet to include free range meat and raw dairy. I'll never go back !