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Wellness Institute

Cholesterol (Part 1)

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History

Cholesterol was discovered in 1775 as a major component of gallstones. The first animal research demonstrating damage to the blood vessels from consumption of cholesterol took place in 1912.

Studies of cholesterol and heart disease have yielded confusing and inconsistent results. One reason for this is that there is a rare, inherited disease called familial hypercholesterolemia. These people have considerably higher cholesterol than normal individuals, suffer with severe atherosclerosis and heart disease early in life.

The most comprehensive cholesterol study ever conducted was the Multiple Risk Factor Intervention Trial (MRFIT). When all was said and done according to Uffe Ravnskov, MD, PhD, this study of more than 300,000 individuals "demonstrated what we already knew--that patients with an inborn error of cholesterol metabolism have a greater risk of dying from heart disease."

There is surprisingly little evidence that cholesterol in and of itself is a contributor to heart disease unless one has the genetic marker which is associated with familial hypercholesterolemia. Quebec, which has one of the highest incidences in the world of familial hydpercholesterolemia, has 1 in 100,000 with the problem. Cholesterol levels of those with familial hypercholesterolemia can rise as high as 557-1532 mg/dl. The small number of people with a genetic tendency to have problems with cholesterol distorts statistical data in studies of cholesterol and has formed the basis for current recommendations to lower cholesterol.

The only form of cholesterol which is a major problem for the average individual is cholesterol which is highly oxidized. Any fat which is highly oxidized will contribute to health problems.

Robert S. Ford, head of Magnolia Laboratory figured it out and published his conclusions in 1969. He wrote, "By feeding experiments with animals and human beings consuming nearly a quarter million dollars in labor and materials over a period of seven years I finally determined that the true cause of arteriosclerosis is simple: **STALE FOOD**."

In 1956 a prominent organic chemist named Louis Fleser suggested that the cholesterol used in the feeding trials implicating that it was involved with heart disease was highly oxidized. In 1976 a group of researchers fed oxidized cholesterol to rabbits and demonstrated much higher damage to blood vessel walls after 24 hours than was evident in rabbits fed nonoxidized or purified cholesterol. After 7 weeks the rabbits fed oxidized cholesterol were demonstrating clear evidence of blood vessel damage remarkably similar to atherosclerosis in man, while no lesions were evident in the animals not given oxidized cholesterol.

In 1979 Taylor and his associates extended research on oxidized cholesterol and demonstrated that oxidized cholesterol is 500 times more damaging to the circulatory system than pure (unoxidized) cholesterol. Taylor concluded his review by saying, "It would appear that nearly all of the studies on the induction of atherosclerosis by feeding USP cholesterol stored in air at room temperature should be reevaluated. The cholesterol used in experimental diets, in the majority of instances, probably contained significant quantities of oxidized sterols that have a strikingly lethal effect on aortic smooth muscle cells."

Oxidized cholesterol initiates inflammatory and free radical dam-

age in the circulatory system leading to heart disease. Despite the fact that only oxidized cholesterol is a risk factor for heart disease for most people the focus of the medical profession has steadfastly and consistently set its sights on the objective of lowering cholesterol levels rather than reducing inflammation and oxidative damage to the fats in the diet and the fats in the human body.

A proper understanding of the mechanism by which cholesterol damages the circulatory system in experimental studies of animals alters one's perception of the risk of cholesterol and the appropriate measures which should be taken to reduce the risk of heart disease.

Prevention of damage from cholesterol ingestion should focus on avoiding highly oxidized cholesterol found in such foods as powdered eggs, aged cheeses and meats, and highly heated foods with large amounts of cholesterol. Less concern should be directed toward such foods as fresh eggs and meats which are unlikely to contain large quantities of oxidized cholesterol. Techniques of cooking and preservation of foods should also be directed toward reducing the chances that cholesterol in foods will become oxidized by use of high temperatures and/or long term storage of foods containing cholesterol—for example baked goods containing whole eggs and butter.

Increasing the intake of antioxidants becomes a significant nutritional strategy in the prevention of heart disease. We can be exposed to oxidized cholesterol in two ways. We can consume oxidized cholesterol in the diet, or the cholesterol can oxidize within the body. Deficiency of vitamin E and carotenoids increases the likelihood that cholesterol will auto-oxidize within the tissues including the circulatory system.

In the past medical recommendations have focused on replacing saturated fats with unsaturated fat. This has never proven out in scientific studies. Ingestion of excessive quantities of unsaturated fats increases oxidative activity in the tissues and almost invariably leads to increased risk of heart disease and cancer. Professor Melvin Werbach writes, "High blood levels of polyunsaturates, especially the omega-6 fatty acids, may increase the risk of atherosclerosis unless there is adequate antioxidant protection."

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Taylor, Ronald S., *Nutrition, Hypertension & Cardiovascular Disease*, Second Edition, Portland, Oregon: Lyncrean Press, 1989, 14-15.

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Trans Fats

Many early studies of saturated fat which demonstrated serious risks failed to distinguish between natural saturated fats and artificially saturated fats or what are called trans fats. These are unsaturated vegetable fats which have been hardened by saturating them with oxygen. Trans fats are now known to be a major contributor to heart disease. An article in the *New England Journal of Medicine* concluded that as much as a fifth of the cases of heart disease in the United States could be laid at the door of artificially saturated trans fats:

"On the basis of reported relations between trans fat intake and CHD events in prospective studies…10 to 19 percent of CHD events in the United States could be averted by reducing the intake of trans fat. Thus, given the 1.2 million annual myocardial infarctions and deaths from CHD in the United States, near-elimination of industrially produced trans fats might avert between 72,000 (6 percent) and 228,000 (19 percent) CHD events each year."

REFEREENCE:

Moxaffarian, Dariush, et al, Trans fatty acids and cardiovascular disease, *The New England Journal of Medicine*, Vol. 354: No 15, April 13,2006, 1601-1613.

Herbivores

Rabbits have often been used in studies of cholesterol for many years. Rabbits are herbivores which would normally consume a diet which contains no cholesterol. Natural herbivores genetically lack a liver/cholesterol feedback inhibition mechanism to control the amount of cholesterol added to the bloodstream.

Carnivores and omnivores do have a feedback mechanism to control the amount of cholesterol manufactured in the liver. Liver synthesis of cholesterol goes down when animal foods rich in cholesterol are consumed. Liver synthesis of cholesterol goes into high gear when omnivores or carnivores eat a plant based diet with little or no cholesterol.

This fundamental difference explains why the ability to induce atherosclerotic lesions in rabbits by cholesterol feeding is often incapable of being duplicated in other animal species which possess a liver/cholesterol feedback mechanism as man does.

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Cholesterol Synthesis

Cholesterol is synthesized from glucose in the human body. The slowest and most vulnerable point in the

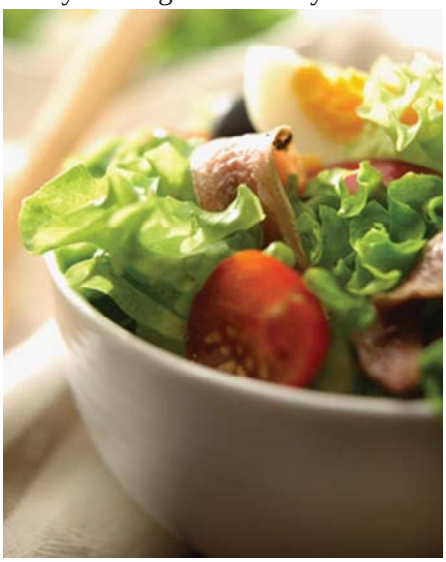
process of cholesterol synthesis is the enzyme HMG-CoA reductase. This enzyme leads to the production of mevalonate, a fatty acid derivative which is then converted into cholesterol.

HMG-CoA reductase is easily and conveniently inhibited by statin drugs. The result has been a bonanza for the pharmaceutical firms which sell a wide variety of these medications. In 2004 Lipitor (atorvastatin) and Zocor (simvastatin) were the two best selling pharmaceutical drugs and the statin class of drugs topped all other prescription drug sales for a whopping total of 15.5 billion in sales.

Our bodies are intricately designed to regulate cholesterol synthesis if intake of the appropriate nutrients is adequate. The key nutrient which regulates cholesterol synthesis is magnesium

Magnesium is crucial in the inhibition of the initial step in cholesterol synthesis. Cholesterol and ATP (the energy currency of the body) deactivate HMG-CoA reductase. Mildred Seelig, the world's leading expert on magnesium, writes, "As long as there is adequate magnesium, the inhibition of this enzyme can take place when necessary, stopping or decreasing the production of mevalonate. The end result is that cholesterol production is impeded and less cholesterol is formed."

Deficiency of magnesium severely deranges the body's cholesterol regulation mechanism. Seelig writes, "Conversely, if there is a deficiency of magnesium, the conversion of HMG-CoA to mevalonate is enhanced, thereby increasing cholesterol formation. This can give rise to the development of cholesterol-filled atheromas—fatty deposits in the arteries. This is a negative result of having too much cholesterol."



This discussion should make one point clear. The means by which cholesterol is elevated in laboratory animals is often quite different from the mechanism of cholesterol elevation in humans. Elevated cholesterol in humans is often associated with faulty energy production (lack of ATP) or deficiency of magnesium.

Magnesium is not an unabashed cholesterol lowering agent. It is a critical regulator of the cholesterol pathway. Cholesterol may be considered elevated by current medical thinking, but if the body is experiencing a deficiency of unoxidized cholesterol for important metabolic functions, magnesium will permit the synthesis of more cholesterol.

It should be pointed out here that excess sugar intake tends to rob the liver of ATP making sugar a culprit in loss of regulation of cholesterol synthesis. In one study the infusion of 50 grams of fructose lowered ATP levels in the liver by about half. Intake of sugar has a more dramatic effect on cholesterol regulation than the intake of cholesterol in foods.

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Johnson, Richard, *The Sugar Fix*, New York: Rodale, 2008, 90.

Magnesium Deficiency

The question arises, "Just how likely is a magnesium deficiency?" Assessment of magnesium deficiency is notoriously difficult. Ronald Smith writes, "Tissue concentrations of magnesium are ten times greater than serum concentrations and they are often not in equilibrium with each other. Infarcted, ischemic or damaged tissue normally releases magnesium into the serum, resulting in elevation of serum magnesium levels. We have the paradox, that just at the time when tissue concentrations are at their lowest, the serum concentrations are raised. This is one of the reasons the measurement of serum magnesium is not a reliable indicator of tissue status. The lack of reliability is a major reason magnesium is relatively neglected by many cardiologists."

Other tests for magnesium status are available, but are rarely performed and often have problems of their own. Among these are examination of the magnesium in red blood cells and a test which loads the body with a large amount of magnesium to see how much the body absorbs. The tendency to suffer muscle cramps or muscle twitches is often suggestive of magnesium deficiency.

Magnesium can be depleted by diets high in calcium and low in magnesium. (Many physicians supplement those with osteoporosis with calcium, but not magnesium.) Excessive intake of vitamin D can also moderately impair magnesium absorption.

Phosphates as found in sodas bind with magnesium and prevent it from being absorbed. I remember talking to a taxi driver one time who drank nothing but sodas. He wound up with an emergency visit to a hospital with severe magnesium depletion.

Phytates in grains can inhibit the absorption of a wide range of minerals including magnesium. Phytates are found primarily in the bran of wheat, rice, oats, and soybeans.

Recommended intakes of magnesium should probably be increased to 450-500 mg a day but most Americans

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do not even obtain the current recommended intake of 300-350 mg a day. A study of 50 colleges found an average intake of 250 mg of magnesium a day

DEFICIENCY

Cholesterol is one of the most important molecules found in the body and plays an important role in a wide variety of metabolic functions. Seelig writes, "...without adequate cholesterol, important substances cannot be formed. The steroid hormones, such as the sex hormones and the adrenal corticosteroids, derive from cholesterol in a number of enzyme dependent steps, as does vitamin D, in response to sunlight exposure.

Americans are so obsessed with the possibility of excessive cholesterol that they do not even consider the possibility that inhibition of cholesterol synthesis could have harmful repercussions. Uffe Ravnskov, M.D., Ph.D., a prominent cholesterol researcher in Europe writes, "As one scientific study after another has shown, people can gorge on animal fat for many years and still keep their blood cholesterol low. What we have also learned is that atherosclerosis and heart attacks may occur whether one's food is rich or lean and, most surprisingly, whether one's blood cholesterol is high or low. Given these facts, is there any reason to think that heart attacks can be prevented by reducing blood cholesterol with diet or drugs?"

Ravnskov summarizes his evaluation of the cholesterol data as follows: "…the chance of a healthy individual with high cholesterol of not dying from a heart attack in 5-6 years was 98.4 percent, a chance you could improve to 98.8 percent with statin treatment. In conclusion, the new guidelines may possibly prevent cardiovascular death in a small minority of patients with cardiovascular disease. But at the same time they may increase mortality from other diseases, transform healthy individuals into unhappy hypochondriacs obsessed with the chemical composition of their blood, reduce the income of ranchers and dairy farmers, undermine the art of cuisine, destroy the joy of eating, and divert health care money from the sick and the poor to the rich and healthy. The only winners are the drug companies and imitation food industry, and the researchers that they support."

Elevated cholesterol and heart disease result from serious nutritional problems, but excess intake of cholesterol itself is not part of the problem, unless that cholesterol is oxidized. The next newsletter will discuss potential problems associated with excessive lowering of cholesterol levels. It is not impossible to be cholesterol deficient.

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