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CHOLESTEROL, FATS, AND HEART ATTACKS – PART I

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At first, all cholesterol was bad; it clogged arteries and caused heart attacks. Then researchers sorted out "good" HDL-cholesterol from "bad" LDL-cholesterol; an unfavorable ratio increased heart attack risk. Later, particles of lipoprotein(a) and/or apolipoprotein-B were implicated followed by triglycerides. Americans were told to avoid dietary cholesterol. Then it was saturated fats. Finally, fats in general had to be reduced. What is the cholesterol-fat-heart attack connection?

CHOLESTEROL

"Cholesterol" is not a dirty word. Cholesterol is an alcohol (though it does not behave as one) and not really a fat. It is primarily produced by the liver (though all cells are able to produce it) and travels through the bloodstream to every cell, tissue, and organ. It is needed for fat metabolism, the development of cells, as an important constituent of cell walls, to maintain the strength of blood vessel walls, to synthesize bile components, in vitamin D production, for brain function, as a component of myelin sheath that protects nerves and nerve impulse propagation. It is essential for strength and resilience. It is used in seminal fluid and vaginal lubrication. It is the basic substance from which steroid hormones like DHEA, cortisol, estrogen, progesterone, and testosterone, are produced. It is required for normal development of embryos. Cholesterol is used to repair and protect tissues, and much more. The body goes to an awful lot of trouble to produce and balance cholesterol. It is essential for human life.

Most of the cholesterol the body needs – about 2000 milligrams (mg) per day – is synthesized by the liver. The average American ingests between 300 to 500 mg of cholesterol per day from animal foods such as meats, eggs, seafood, and dairy products. This means 80 to 85% of cholesterol is produced by the liver, and only 15 to 20% is obtained from dietary sources. Even without the intake of cholesterol-containing foods, the body sufficiently balances cholesterol. Consumption of cholesterol-containing foods does not in and of itself cause chronically high cholesterol levels in the blood. The amount of cholesterol produced by the liver is dependent on the total available cholesterol -- regardless of the source. A feedback system reduces its production if there is more than needed. Thus, dietary cholesterol may serve to reduce its synthesis in the body. Any excess cholesterol is simply excreted through the bile. So, if one eats too much cholesterol, the cells produce less. If one eats too little, the cells produce more. It is not easy to change one's cholesterol level by changing the diet. It can be done, but only by 5 to 10%. About 5% of the population has very high blood levels of cholesterol (350 and above) probably due to a genetic metabolic disorder that "is merely reflected in high cholesterol readings – like a fever indicates an infection and is not a disease itself." [i]

CHOLESTEROL NUMBERS

Blood cholesterol numbers currently used include: **Total cholesterol** – no risk: less than 150, low risk: 150 to 200, medium risk: 200 to 250, high risk: greater than 250. **Low density lipoprotein** (LDL) -- no risk: less than 100, low risk: 100 to 130, medium risk: 130 to 160, high risk: greater than 160. **High density lipoprotein** (HDL) – no risk: greater than 75, low risk: 60 to 75, medium risk: 40 to 60, high risk: less than 40. Ratio of **total cholesterol/HDL** – No risk: less than 3.5, low risk 3.5 to 4.5, medium risk: 4.5 to 5.5, high risk: greater than 5.5. Another marker appearing is **triglyceride/HDL** ratio – "should be" below 2.0.

Cholesterol researcher Uffe Ravnskov, M.D., Ph.D., challenges the limit of 200 arbitrarily placed on total cholesterol back in the 1980s. "This is a level invented without any evidence." What determines the blood cholesterol level is "difficult to tell because there are so many factors that influence" it from mental stress or anger to exercise, from weight gain or loss to tissue insults and injury. To set a limit of 200 and to proclaim that any number below that is healthy and any number above that is unhealthy is "pure speculation." It is based on the idea that cholesterol levels above 200 predict coronary heart disease. Some studies seem to show this, but others do not.

In his book, *The Cholesterol Myths*, Dr. Ravnskov provides a thorough account of numerous cholesterol studies. He reveals that some studies which seem to show a relationship between "high" cholesterol levels and coronary heart disease (CHD) include data that are inconsistent and highly questionable. In some cases, conclusions reached by researchers are completely contrary to what the data show. Research papers and

reviews by organizations like the National Heart, Lung & Blood Institute and American Heart Association "systematically ignore all contradictory evidence. They all cite the contradictory papers as if they were supportive." In some cases when researchers get results that are contrary to the cholesterol hypotheses, they write conclusions indicating that their findings support the idea. Most people (including doctors and other researchers) read only these conclusions as written in the summary. To find the contradictions, one would have to read the whole papers and meticulously study the graphs. Correlations between CHD and cholesterol levels are often weak. False correlations are not unusual. For example, a false correlation between atherosclerosis and blood cholesterol may be made when the actual relationship is between atherosclerosis and age or between cholesterol and age. Or a correlation between cholesterol and the degree of atherosclerosis may appear in studies that include people with familial hypercholesterolemia (genetically-induced high cholesterol); if these participants are excluded from the statistics, the correlation disappears. Many studies have found no association between cholesterol levels and heart mortality. Some studies indicate **low** cholesterol predicts CHD.

From the rhetoric, it is assumed that people who have heart attacks almost always have large amounts of cholesterol in their blood. Yet data demonstrate that the difference between those who have CHD and those who do not is marginal. For example, a graph from the famous Framingham heart study shows that almost half of those who had a heart attack had low cholesterol. As time passed in this 30-year study, a "few" more people with high cholesterol levels died – on average one percent of all men with high cholesterol died each year. Only half as many died among those with the lowest cholesterol values. Women with low cholesterol died just as often as women with high cholesterol. It appeared that high cholesterol was more dangerous, but the figures released included death from ALL causes, not just heart mortality. And, cholesterol levels made no difference for men over the age of 47 – those who had low cholesterol at age 48 and older died just as often as those with high cholesterol. Evidently, if you reach age 47, it does not matter whether your cholesterol is high or low! More than 95% of all heart attacks occur in people older than 48. If cholesterol levels are important for the few who have heart attacks before age 48, why should everyone else worry about blood cholesterol levels? Actually, during the 30-year Framingham study, those whose cholesterol had **decreased** "by itself" actually ran a greater risk of dying than those whose cholesterol had **increased**. The report stated: "For each 1 mg/dl drop of cholesterol there was an 11 percent increase in coronary and total mortality."

For many years the public has been told how important it is to lower their cholesterol levels to prevent coronary heart disease. Yet the large Framingham study demonstrated that if blood cholesterol decreases by itself, the risk of dying increases. The report clearly shows that mortality **increased**, yet the written review stated that mortality **decreased**. This was only one of many "mistakes" presented to the public.

High cholesterol in women is not a risk factor. Studies show that it is more dangerous for women to have low cholesterol than high. "Excess dietary cholesterol" does not increase the risk of developing CHD in women. Elderly women with very high cholesterol live the longest. Although high cholesterol levels appear to have a "slight association" with increased risk for men in the US, it has no such association for men in Canada. Neither is blood cholesterol important for those who have already had a heart attack. In Russia, **low** cholesterol is associated with increased risk of CHD. In Stockholm, men with low cholesterol died from heart disease just as often as those with high cholesterol. The people of the Maasai tribe in Kenya eat a diet of milk and meat with twice the fat and cholesterol content of most Western diets, and yet they are basically free of heart disease. The Maori, Polynesians who migrated to New Zealand hundreds of years ago, may die from heart attacks, but do so whether their cholesterol is low or high. The Batemi people of Tanzania average up to 2,000 milligrams of cholesterol a day in their diets, well over the "maximum recommended daily intake of 300 milligrams." Yet their blood cholesterol levels are low (about one-third the levels of the average American) and they do not suffer with CHD. The Mennonites, an agrarian community similar to the Amish, follow a traditional diet high in cholesterol and saturated fat – with abundant dairy products, eggs, and red meat -- but have serum cholesterol and blood pressure levels lower than other Americans.

Research shows that "there is little relationship between serum cholesterol values and coronary heart disease in those over 70." **Low** blood cholesterol levels are associated with malnutrition, disease, and death, especially among the elderly. Cholesterol levels higher than "normal" are associated with increased longevity in people over age 85. With increasing age, persistence of **low** cholesterol levels increases risk of death.

Thus, high cholesterol is said to be dangerous, but not for Canadians, Russians, Stockholmers, Maasais, Maoris, Batemi, or Mennonites. High cholesterol is said to be dangerous to men, but

not to women; dangerous for healthy men, but not for those who had heart attacks; dangerous for men under age 47 but not those 48 and older. High cholesterol may even be beneficial for older people. Obviously, any association between high cholesterol and CHD is not one of simple cause and effect. "The most likely interpretation is that high cholesterol is not dangerous in itself but a marker for something else." High or low cholesterol concentrations are "not pathogenic by themselves but are secondary to other, more important factors." It follows that lowering cholesterol levels by diet does not lower the risk of heart mortality. One indication that high cholesterol is not pathogenic by itself is the percentage of people who have familial high cholesterol levels and reach a normal life span with a lower risk of CHD than the general population. There are "environmental factors of much greater importance than the cholesterol concentration."

Smoking; overweight; high blood pressure; stress; altered or damaged dietary fats; refined sugars; nutritional deficiencies; fragility, loss of elasticity, and lesions of the coronary arteries – all have a much stronger association with CHD than cholesterol levels. Serum total cholesterol levels are elevated in liver imbalances, disease, or toxic overload; hypothyroidism; diabetes; kidney disease; and other chronic problems or stresses. "Cholesterol is nature's healing substance. Without it wounds would not heal and our cells could not maintain their integrity." Just as plant cells depend on their structure and firmness from cellulose, human cells – including cells forming blood vessels -- obtain their shape and strength from cholesterol. Thus elevated cholesterol levels indicate an increased need to support, protect, or replenish when there has been insult, injury, or depletion. When lifestyle improvements including a healthier diet result in a lowering of serum cholesterol, it means that the body no longer requires the extra circulating cholesterol. The repair or protection has been completed, or the excessive stress has been reduced. For example, physical or emotional distress may induce the adrenal glands to produce larger than usual amounts of steroid hormones to cope. Increased amounts of cholesterol are needed as raw material for the hormone production. Once the distress is reduced and the adrenal glands recuperate, the need for extra cholesterol diminishes.

Back in 1990, a study from Georgetown University showed that total cholesterol levels varied by more than 20% in 75% in participants. Similarly, LDL and HDL cholesterol fluctuations of the same magnitude were found in 65 to 95% of the subjects. With retesting, 40% of the participants moved from one risk category to another and 10% moved from the lowest risk category to the highest risk category or vise versa. "Fluctuations occurred randomly from week to week, and were unrelated to age, sex, or the serum levels of lipoproteins." Cholesterol numbers vary among individuals (biochemical individuality) and will change when the need for cholesterol changes. Results of a cholesterol blood test can be influenced by changes in diet, fluctuations in weight, amount of alcohol intake, injury, surgery, infection, physical strain, most any stress, and numerous other circumstances. Not only an individual's past readings must be considered to determine what is basically "normal" for him or her, but also present and varying circumstances must be taken into account. From 50% to 60% of all heart attacks occur in people with "acceptable" or "desirable" cholesterol levels.

Scientists admit that "not all epidemiological studies show a correlation between dietary cholesterol alone and either serum cholesterol or coronary heart disease." The "large difference in absolute CHD mortality rates at a given cholesterol level...indicates that other factors, such as diet, that are typical for cultures with a low CHD risk are also important with respect to primary prevention." In other words, serum cholesterol levels are generally meaningless when it comes to heart disease. And a healthful diet, whether or not it contains cholesterol-rich foods, is essential to prevent CHD. People with CHD and "normal" levels of cholesterol who are given cholesterol-lowering drugs respond with significantly lower serum cholesterol levels but **no** "**measurable benefit on the coronary arteries**." If there is no benefit to the blood vessels that are supposedly "clogged" with cholesterol, than something other than cholesterol must be causing the problem!

Indeed, the "consistency of the clinical and the epidemiological data demonstrating that dietary cholesterol has little effect on plasma cholesterol in most individuals raises a number of questions regarding the justification of population-wide restrictions on dietary cholesterol intake." Scientists, despite a vast array of cholesterol studies, have been unable "to detect associations between diet and serum cholesterol." Every cell in every animal, including humans, contains cholesterol. Animal fat, composed of animal cells, contains cholesterol. Lean meat, also composed of animal cells, contains cholesterol. Some animal foods contain more cholesterol than others. Fat does not determine the cholesterol content. For example, butter and lard are high in fat but low in cholesterol. Eggs and liver are low in fat but high in cholesterol. Eggs are now approved by heart associations and dieticians as

part of a healthy, CHD-preventive diet. Natural unaltered foods, even those high in cholesterol, do not increase risk for CHD. A low-cholesterol diet will not prevent heart attacks. The blood level of cholesterol will not determine if an individual is more likely to have a heart attack or not. [ii]

LIPOPROTEINS

What about so-called "good" high-density lipoprotein (HDL) cholesterol and so-called "bad" low density lipoprotein (LDL) cholesterol?

The cholesterol molecule is arranged in an intricate network that is impossible to dissolve in water. Because it is insoluble, it circulates in the blood inside round particles made of fats (lipids) and proteins – lipoproteins. The outside of lipoproteins is composed mostly of water-soluble proteins. The inside is composed of lipids with room to carry water-insoluble molecules like cholesterol. Lipoproteins serve as 'vehicles' to transport cholesterol through the blood and are categorized by their protein density (high density, low density, etc.).

The primary job of HDL is to pick up used or unneeded cholesterol molecules and cholesterol esters **from** all peripheral tissues, including artery walls, and transport or return them **to** the liver as part of a recycling process. In the liver, cholesterol is excreted with the bile or used for other purposes. When cholesterol needs to be removed from cells, it is HDL that usually does the job. Some cholesterol is always sloughing off cellular membranes into the plasma. HDL is believed to be protective, preventing a buildup of cholesterol and lowering risk of CHD by removing unused cholesterol from the blood. It is also thought that HDL may be able to collect cholesterol from artery plaque, reversing the atherosclerotic process leading to heart attacks.

The LDL particles primarily carry cholesterol **from** the liver (where most of the body's cholesterol is synthesized) **to** the peripheral tissues, including blood vessel walls. When cells need extra cholesterol, it is the LDL vehicles that deliver cholesterol into the cellular interiors. In most (not all) people, LDL contains a higher percentage of cholesterol, so has been dubbed "bad." When a cell needs more cholesterol, it produces more LDL receptors on its plasma membrane. This allows the cell to bind more LDL, ingest it, and obtain its cholesterol -- a process the cell prefers over making cholesterol itself. Generally, when cells need cholesterol, LDLs come to the rescue.

Between 60 and 80% of cholesterol in the blood is transported by LDL. About 15 to 20% is carried by HDL. Smaller amounts of cholesterol are carried in circulation by other types of lipoproteins such as VLDL (very low density lipoprotein). The liver is "the center of the cholesterol universe." It synthesizes new cholesterol, recycles used cholesterol, and secretes old cholesterol into bile, transforming it into bile acids. The production of cholesterol by the liver is inhibited whenever dietary cholesterol is increased, and stimulated when dietary cholesterol is reduced. This homeostatic control is the primary reason why it is actually difficult to alter plasma cholesterol very much in either direction by altering the diet. So attention was focused on the "messengers" HDL and LDL regarding CHD risk. Some health professionals point to LDL levels that are too high and HDL levels that are too low. Others consider ratios as most telling – ratios between HDL and LDL or between HDL and total cholesterol. The average US ratio of total cholesterol to HDL is 5 to 1 (HDL representing one-fifth or 20% of total cholesterol) and is not considered healthy. A better ratio is thought to be 3 to 1 – 33% of total cholesterol. Still, many experts feel that individual levels of total cholesterol, HDL, and LDL are more important than ratios. What does it all mean?

Excess weight gain results in an increased risk of CHD as well as higher LDL and lower HDL. Lack of physical exercise, hypertension, and smoking do the same. The question is: do these or other known CHD risk factors bring about heart attacks BECAUSE of the increased LDL and decreased HDL? Or are the changes in LDL and HDL a RESULT of insult, injury, or imbalance that triggers an increased need for cholesterol in the cells involved? When a person becomes overweight, for example, there is more stress on the heart, blood vessels, liver, and more. Cells become less sensitive to insulin, predisposing to diabetes. Atherosclerosis and other vascular damage commonly occur in early diabetics, even those with normal cholesterol levels. Inactivity increases CHD risk by mechanisms other than an abnormal HDL/LDL ratio, including constriction of blood vessels. The vascular channels in a well-trained or fit cardiovascular system are broader. Smoking damages blood vessel walls, including coronary arteries. Hypertension puts increased stress on specific areas in blood vessel walls; it is often a result of excessive stimulation by the sympathetic nervous system. The underlying CAUSES of these problems are not disrupted HDL/LDL numbers. But the body's innate methods of dealing with them may RESULT in higher LDL and lower HDL.

Studies indicate that people who had suffered heart attacks had a lower HDL-cholesterol primarily because they were older, fatter, had higher blood pressure, and smoked more than those who had not had a heart attack. Some

studies did not find that HDL cholesterol was a major predictor or risk factor for CHD or, at best, was of marginal value for risk prediction. The researchers often admitted that the difference could as well have been due to other risk factors like stress or lack of exercise.

Theoretically, LDL cholesterol should have the strongest relationship to risk of CHD and should be a better predictor than total or HDL cholesterol. But it is not. Some studies found that total cholesterol, not LDL-cholesterol, had a stronger relationship to risk of CHD. Others indicated there was a greater risk of heart attack if LDL cholesterol was low than if it was high. One report showed the predictive power of LDL cholesterol was statistically insignificant. An interesting study indicated that LDL was predictive for CHD only for men between ages 35 and 49 and only for women between ages 40 and 44. A review of the studies leads to the conclusion that LDL-cholesterol is not centrally or causally important; it does not have the strongest or most consistent relationship to risk for CHD. In fact, the endorsement of LDL-cholesterol as a risk factor by the National Cholesterol Education Program is "loaded with misquotations and even false statements."

Many people with low HDL ("good") cholesterol have no CHD. Data eventually appeared that indicated a low HDL cholesterol level may not be so bad as long as enough of it is wrapped in a protein called apolipoprotein A-1 (apo A-1). Apolipoprotein B (apo B) was dubbed "bad" and thought to be a better predictor of cardiovascular risk than LDL. Yet, screening people for CHD by measuring apo B alone or with apo A1 is "too poor to discriminate between recommending drug therapy or lifestyle change for some and not others." Not helpful.

Then some people were found to have small LDL cholesterol particles and others to have large LDLs. The smaller the LDLs, the greater the risk of CHD. Low-fat diets reduce LDL more in people with small particles than those with bigger particles. The smaller LDL particles are more easily oxidized (made rancid and toxic). However, people with larger LDL molecules tend to have abnormal levels of other blood fats. So, it is difficult if not impossible to determine if particle size is an independent risk factor.

A high level of lipoprotein(a) [Lp(a)], a group of varied particles in the blood closely related to LDL, was thought to increase risk of CHD and stroke. But not all studies found this, and it is not known if lowering high Lp(a) levels will prevent CHD. Diet does not seem to affect Lp(a) levels. However, a high Lp(a) – above 30 milligrams – may not be harmful if the LDL level is normal. In others words, it is not an independent risk factor. Some studies found no evidence of an association between Lp(a) and risk of future CHD. A study of people over 100 years of age found that 25% of this group had high Lp(a) serum levels even though they never had atherosclerosis-related diseases. Most of these folks also had low HDL levels and relatively high triglyceride levels, which together are considered to be strong risk factors for CHD. *Trans* fatty acids (altered, detrimental fats found in margarines, shortenings, partially hydrogenated vegetable oils, fried foods) may raise Lp(a) levels. Saturated fats from whole natural foods lower Lp(a). A large study suggested that elevated Lp(a) may be the result of coronary artery damage rather than a cause.

Since data show that people who live long may have "risk" factors for CHD yet manage to live that long without atherosclerotic problems like heart attacks, the conclusion must be that the numbers (HDL, LDL, Lp(a), etc.) change according to the individual's needs as he/she ages, the degree of toxic offence, and need for repair, and do not indicate risk for heart attacks.

Oxidation of low density lipoprotein is thought to be involved in CHD somehow. This means the problem with LDL-cholesterol is not so much its presence or its quantity in the blood, but that it is easily oxidized or made rancid. This rancidity may contribute to tissue insult or injury. What causes LDL to become oxidized? For one thing, consumption of damaged or altered fats in the diet introduces unstable, rancid, unnatural fats to the tissues that can be poisonous and harmful. Most commercial vegetable oils fit this category. So do partially hydrogenated fats – containing trans fatty acids (found in most processed foods) – which have been shown to significantly increase the risk of CHD. Foods that are stored too long, stale, degraded, denatured, deteriorating, chopped, ground, mixed, or prepared also contain increased amounts of oxidized cholesterol molecules and fats as well as decreased amounts of antioxidants and other nutrients that prevent excessive or premature oxidation. Refined sugars increase oxidation damage, cross-link proteins, inhibit immune functions, and interfere with the transport of vitamin C complex (essential to the integrity of blood vessel walls). Deficiencies of nutrients that protect LDL – including the antioxidant portions of nutrient complexes as well as their more functional parts – contribute to the problem. Deficits of the vitamins A, B, C and E complexes have been implicated as well as minerals like calcium, magnesium, potassium, selenium, etc., many phytochemicals, and fatty acids.

In multiple studies, "dietary cholesterol was not a predictor of plasma total or LDL cholesterol

levels." But a high intake of calories, primarily from nonfoods containing little or no nutritional value, has been associated with elevated cholesterol levels. A high intake of refined carbohydrates (refined sugars and flours, etc.), for example, can result in elevated insulin levels which may increase cholesterol levels. A diet low in refined carbohydrates lowers elevated total and LDL cholesterol. [iii]

LOW CHOLESTEROL LEVELS

Numerous reports and studies indicate that **low** levels of blood cholesterol are associated with increased rates of depression, mood disorders, aggressive or disorganized behavior, violence, stroke, and suicide. These are just the adverse effects known at this time. Adequate serum cholesterol is needed for the proper function of the brain including its serotonin receptors. Serotonin is called one of the "feel good" biochemicals. People with chronically low cholesterol levels often show reduced serotonin levels. Cholesterol serves as precursor for most all steroid hormones such as pregnenolone, estrogen, progesterone, testosterone, DHEA, and cortisol, – all of which can affect mood and behavior.

Cholesterol levels below 150 are potentially harmful. A good portion of the population already takes drugs to force their cholesterol levels lower and half the population is targeted for administration of statin drugs in the future. Such a widespread practice of forcing cholesterol levels lower through severe diets or toxic drugs for supposed reductions in heart disease risk raises some serious questions. In fact, a study spanning about 20 years showed that long-term low cholesterol increases the risk of death in the elderly. The earlier people experience lowered cholesterol concentrations, the greater the risk of death. Researchers are now beginning to question whether there is "scientific justification for attempts to lower cholesterol" to concentrations below 180 mg/dl in elderly people. Elderly people are at higher risk for heart attacks, but lowering their cholesterol levels – especially too low – actually increases their risk for death.

Removing as many dietary sources of fat and cholesterol as is possible may, for a time, cause the body to mobilize, reabsorb and digest stored excess fats. But after six to 12 weeks or so, the excess fats will be gone and problems can begin to develop. Lowered sexual activity, impotence, dry skin, fatigue, loss of energy and motivation, premature aging and wrinkling, nervousness, irritability, depression, and other consequences are not unusual. It is also not unusual for blood concentrations of cholesterol to stay where they were or to go even higher. During the last two decades, the death rate from heart disease has dropped. During the same period, the nation's fat intake dropped a mere 6%. Perhaps emergency medical treatment is one reason the death rate has dropped, though CHD is still the number one killer. Whatever the reason(s) for the lowered death rate, "it isn't the low-fat diet, and it isn't reduced intake of dietary cholesterol." [iv]

To be continued in Part II.

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