The Weston A. Price Foundation

What Causes Heart Disease?

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For almost forty years, the lipid hypothesis or diet-heart idea has dominated medical thinking about heart disease. In broad outlines, this theory proposes that when we eat foods rich in saturated fat and cholesterol, cholesterol is then deposited in our arteries in the form of plaque or atheromas that cause blockages. If the blockages become severe, or if a clot forms that cannot get past the plaque, the heart is starved of blood and a heart attack occurs.

Many distinguished scientists have pointed to serious flaws in this theory, beginning with the fact that heart disease in America has increased during the period when consumption of saturated fat has decreased. “The diet-heart idea,” said the distinguished George Mann, “is the greatest scam in the history of medicine.”And the chorus of dissidents continues to grow, even as this increasingly untenable theory has been applied to the whole population, starting with lowfat diets for growing children and mass medication with cholesterol-lowering drugs for adults.

But if it ain’t cholesterol, what causes heart disease? We don’t know enough to say for sure but we do have many clues; and although these clues present a complicated picture, it is not beyond the abilities of dedicated scientists to unravel them. Nor is the picture so complex that the consumer cannot make reasonable life-style adjustments to improve his chances.

What Is Heart Disease?

Coronary Heart Disease (CHD) is not a single disease, but a complex of diseases of varied etiology. Some of the recognized causes of heart disease include damage to the heart muscle

or valves due to a congenital defect; or to inflammation and damage associated with various viral, bacterial, fungal, rickettsial or parasitic diseases. Rheumatic fever or syphilis can lead to heart disease, as can genetic or autoimmune disorders in which cellular proteins in the heart muscle are deranged or which disrupt enzymes affecting cardiac function.

These factors probably contributed to most cases of heart disease recorded in the early part of the century, when rates of infectious diseases were much higher and antibiotics were not in use. Nevertheless, according to CDC statistics, heart disease was relatively rare in 1900, accounting for approximately 9 percent of all deaths in the US (http://www.cdc.gov/nchs/data/dvs/lead1900_98.pdf (http://www.cdc.gov/nchs/data/dvs/lead1900_98.pdf)).

But by 1950, CHD (including stroke) was the leading cause of mortality in the US, accounting for 48 percent of all deaths. Since that time, mortality rates from CHD have declined somewhat. In 1998, CHD (including stroke) accounted for about 38 percent of all deaths. One reason for the decline is the fact that victims of heart disease are living longer, due most likely to improved surgical techniques, the advent of angioplasty and the use of anti-clotting drugs given to heart attack victims. But the morbidity rates—the incidence of heart disease—remains high. Of greatest concern is the high rate of heart disease in American men between the ages of 45 to 65—during the period of greatest family and career responsibilities.

The interesting thing is that most cases of heart disease in the twentieth century are of a form that is new, namely heart attack or myocardial infarction—a massive blood clot leading to obstruction of a coronary artery and consequent death to the heart muscle. Myocardial infarction (MI) was almost nonexistent in 1910 and caused no more than 3,000 deaths per year in 1930. Dr. Paul Dudley White, who introduced the electrocardiograph machine to America, stated the following during a 1956 American Heart Association televised fund-raiser: “I began my practice as a cardiologist in 1921 and I never saw an MI patient until 1928.” By

1960, there were at least 500,000 MI deaths per year in the US. Rates of stroke have also increased and the cause is similar—blockage in the large arteries supplying the brain with blood.

The factors that initiate a heart attack (or a stroke) are twofold. One is the pathological buildup of abnormal plaque, or atheromas, in the arteries, plaque that gradually hardens through calcification. Blockage most often occurs in the large arteries feeding the heart or the brain. This abnormal plaque or atherosclerosis should not be confused with the fatty streaks and thickening that is found in the arteries of both primitive and industrialized peoples throughout the world. This thickening is a protective mechanism that occurs in areas where the arteries branch or make a turn and therefore incur the greatest levels of pressure from the blood. Without this natural thickening, our arteries would weaken in these areas as we age, leading to aneurysms and ruptures. With normal thickening, the blood vessel usually widens to accommodate the change. But with atherosclerosis the vessel ultimately becomes more narrow so that even small blood clots may cause an obstruction.

The other half of the MI equation is the blood clot or thrombus that blocks blood flow to the heart or brain. Thus, any search for the causes of heart disease must consider complex factors in the blood that promote clotting at inappropriate times, that is, other than in response to bleeding from a rupture or wound. In fact, while a great deal of attention has been focused on the cause and solution to atherosclerosis, the role played by clotting factors in the blood has been relatively neglected. Yet a heart attack due to a clot can occur even in the absence of arterial blockages.

Inflammation may also cause blockages. In fact, a new view of coronary artery disease is that it is an inflammatory process, characterized by cycles of irritation, injury, healing and reinjury inside the blood vessels.[1] The inflammatory response is actually a defense mechanism that helps the body heal but when the inflammatory process goes awry, plaques may rupture, provoking clots that lead to heart attacks.

The health and integrity of the blood vessel walls is another factor that must be considered. Aneurysms, the dilation and rupture of blood vessels due to weakness in the vessel walls, will naturally provoke a clotting response, not to mention the more immediate danger of rapid

blood loss. In addition, biochemical imbalances in the smooth muscle cells may result in spasms that can be just as effective as a blood clot in cutting off blood flow to the heart.

Finally, arrythmias—abnormalities in the rhythm of the heart's pumping mechanism—can

lead to interrupted blood flow, oxygen starvation of the heart muscle or complete shut down of the heart—the so-called cardiac arrest. Regulation of the nervous impulses that govern the heart depends on a large number of factors—from mineral status to the integrity of the myelin sheath.

Known Risk Factors

There are dozens of risk factors for heart disease. Those cited most often by medical orthodoxy include high blood cholesterol, smoking, lack of exercise, stress and overweight. A high level of cholesterol in the blood is a mild risk factor for individuals with familial hypercholesterolemia (cholesterol levels chronically above 350 mg/dl) but for most of us, there is no greater risk of heart disease between cholesterol levels that are “high” (over 300 mg/dl) and those that are “low” (under 200 mg/dl).

One factor of apparent importance is smoking, which has been associated in many studies with an increased risk of coronary mortality, even after correction for other risk factors. It is easy to speculate on the mechanism by which smoking causes heart disease. Exposure to fumes containing free radicals may promote the growth of atherosclerotic plaques. Perhaps chronic carbon monoxide intoxication limits the heart’s utilization of oxygen.

But the picture is more complex than simple cause and effect. In a multi-year British study involving several thousand men, half were asked to reduce saturated fat and cholesterol in their diets, to stop smoking and to increase the amounts of unsaturated oils such as margarine and vegetable oils. After one year, those on the “good” diet had 100 percent more deaths than those on the “bad” diet, in spite of the fact that those men on the “bad” diet continued to smoke.[2] In a study of Indians from Bombay and Punjab, researchers found that those from Punjab had one-fifth the number of heart attacks even though they smoked eight times more cigarettes.[3] And while smoking was widespread at the turn of the century, myocardial infarction was not. This suggests that there may be factors in traditional diets that

protect against the negative effects of smoking. It also raises the question of whether additives now used in cigarette paper and filters and changes in the curing process itself have exacerbated the harmful effects of cigarette use.

Perhaps the association between smoking and heart disease is really an association with some other factor—stress, biochemical imbalances, nutrient deficiencies—that creates the desire or the need to smoke. Often when people quit smoking they become nervous and overweight, which may seem a bad bargain of one risk factor in exchange for two more.

Regular physical activity is one of the few risk factors that has proved consistent. In all studies, regular physical activity is inversely associated with mortality from CHD, and physical activity is the only factor that has shown dose-response in the trials. Common sense tells us why exercise may be beneficial. When we exercise, our heart beats more rapidly, the arteries widen to provide more oxygen and arterial blood flow improves.

Lack of exercise may also be a risk factor because it is a marker for something else that is the true cause. People who are overweight, for example, are less inclined to exercise. Prosperous people who have leisure time are more likely to exercise than those who must work long hours to make ends meet—and we know that heart disease in westernized nations is more prevalent among the poor.[4] Dietary factors may make people less inclined to exercise. An interesting finding in the Framingham study was that those who ate the most saturated fat, the most calories and the most cholesterol were the most physically active.[5] They also weighed the least and had the lowest levels of serum cholesterol!

Common sense also tells us why overweight may be a risk factor. People who are overweight are less inclined to exercise. They probably eat large quantities of refined foods that provide lots of calories but little nourishment. They may have biochemical imbalances that contribute not only to overweight but also to some of the many aspects of heart disease, such as the tendency to form blood clots.

Many doctors have noticed that heart attack strikes in the months just after severe emotional trauma—loss of a spouse or close friend, bankruptcy, layoff or disappointment. We know that grief changes many aspects of the body chemistry, making us more vulnerable to all sorts of

diseases—not just heart disease but also cancer, allergies, tuberculosis and depression. But mankind has always suffered loss and grief. The question is why these traumas cause heart attacks today but did not in 1900.

Although the known risk factors may not be the underlying causes, it makes sense to exercise regularly, to avoid smoking, to maintain an appropriate body weight and to minimize stress. Unfortunately, avoidance of these risk factors is no guarantee. We all know of slim, nonsmoking, active, successful individuals who have developed heart disease—including athletes who have keeled over while jogging. And stress cannot always be avoided. All of us face loss and challenge. The question is, how do we fortify the body to deal with stress in a way that minimizes its impact on the physical body?

The ABCs of Nutrient Deficiencies

In 1930, Dr. Weston Price published an interesting paper in the Journal of the American Dental Society.[6] For years, Dr. Price had been analyzing the amount of vitamin A and vitamin D in butterfat. He noted that these nutrients were most plentiful in the spring and fall, when cows had access to rapidly growing green grass. During the winter and the dry summer months, levels of these vitamins in butterfat declined or disappeared completely.

Dr. Price also tabulated the number of deaths from heart attacks in local hospitals. When he plotted these two variables against time on the same graph he found that deaths from heart disease were inversely proportional to the vitamin content in the butter. In other words, when nutrient levels were high, deaths from heart disease were low; and when nutrient levels were low in the winter and summer, deaths from heart disease were high. He found this pattern in many different localities, even in areas in the far north where there was only one vitamin peak, in midsummer, due to the short growing season.

Heart disease researchers have largely ignored the possible role of vitamin A and D in protecting the heart, probably because these fat-soluble vitamins are found only in the foods they have demonized—animal fats. Yet both nutrients play numerous important roles in the body chemistry, principally as catalysts for protein and mineral assimilation.[7] Both nutrients support endocrine function and protect against inflammation. Vitamin A is needed for the conversion of cholesterol into steroid hormones and, in fact, is rapidly depleted by stress. Cholesterol-lowering drugs increase the body's need for vitamin A.

Vitamin D helps prevent high blood pressure and protects against spasms. As vitamin D is needed for calcium absorption, it contributes to a healthy nervous system and helps prevent arrythmias.

In the 1960s, a pair of Canadian doctors named Wilfred and Evan Shute claimed to prevent recurrence of problems in CHD patients with the administration of vitamin E.[8] They pointed out that lack of vitamin E in the American diet is partially due to the milling process which eliminates the highly perishable wheat germ, a significant source of vitamin E. High levels of omega-6 fatty acids from commercial vegetable oils can actually raise the body's requirements for vitamin E. Vitamin E is an antioxidant that can prevent free radicals from causing damage at the cellular level and it plays an essential role in cellular respiration, particularly in the cardiac muscles. Vitamin E makes it possible for these muscles and their nerves to function with less oxygen. It promotes dilation of the blood vessels and inhibits coagulation of the blood by preventing clots from forming.

Dr. Linus Pauling, famous for his work on vitamin C, proposed vitamin C deficiency as a possible cause of CHD.[9] A six-year Finnish study linked low blood levels of vitamin C to increased risk of heart attack during subsequent years.[10] As an antioxidant, vitamin C protects against free radical damage. It has the effect of making oxygen metabolism more effective and may also help prevent clot formation. Vitamin C is essential for the production of collagen and therefore protects the integrity of the artery walls. Vitamin C is used up very quickly during periods of stress.

Researcher Kilmer McCully has found a positive relationship between deficiencies in folic acid, B6 and B12 and severity of hardening or stiffness of the arteries, as well as the buildup of pathogenic plaque.[11] Vitamin B6 and vitamin B12 are found almost exclusively in animal products—the foods that proponents of the lipid hypothesis advise us to avoid.

Another nutrient found exclusively in animal products, particularly in red meat and organ meats, is coenzyme Q10, which serves as an antioxidant and as fuel for the mitochondria in the cells. In the body, coenzyme Q10 is most concentrated in the heart muscle cells. It seems to be helpful in reducing inflammation and has been used successfully in the treatment of

heart disease.[12] Cholesterol-lowering drugs greatly increase the body's need for coenzyme Q10.

Deficiencies of certain minerals have also been proposed as possible causes of heart disease. According to Dr. Roger Williams, an inadequate supply of magnesium may result in the formation of clots and contribute to calcium deposits in the blood vessels.[13] Heart attack patients improve their survival chances from 50 to 82 percent when given intravenous magnesium in the first 24 hours following myocardial infarction.[14]

Many other minerals play a role in cardiovascular health. Copper and zinc, for example, are contained in enzymes that the body uses to defuse free radicals and that help create healthy collagen. These minerals are most easily assimilated from animal foods.

Deficiency of selenium has been linked to CHD[15] and is associated with Keshan disease, characterized by fibrotic lesions in the heart.[16] In conjunction with vitamin E, selenium has been used successfully to reduce or eliminate angina attacks. Soils in most of Finland are deficient in selenium, which may account in part for the fact that heart disease in that country is high. A national program to add selenium to the soil, initiated in 1985, may offer partial explanation for the decline in heart disease in Finland (although the decline began before the selenium enrichment program was instituted).

It is easy to make the case that, in spite of our prosperity, the actual nutrient content of our foods has declined during the last 70 years. A number of researchers have cataloged the decline of minerals in our soils, due to intensive farming practices.[17] Most milk in the US today comes from cows housed in confinement dairies. They are fed dry feed and never see the green grass their bodies need to make large quantities of vitamin A and vitamin D. Isolated isomers of vitamin D are added to milk in an attempt to rectify this situation. Processed food, usually based on sugar, white flour and vegetable oils, has replaced many nutrient-dense foods that were eaten routinely in the past. Few Americans eat liver on a weekly basis or take cod liver oil as our ancestors did.

Nor do they use lard, which is another rich source of vitamin D. Like humans, pigs can get sunburned and, like humans, they make vitamin D through the action of sunlight on their skin and store the nutrient in their fat. Pigs raised in confinement will die if not exposed to UV-B light, the wave length needed for vitamin-D production. Fifty years ago, lard contributed

important nutrients to the American diet but few people use it today.

Elusive Answers

The problem is that it is difficult to turn these clues and theories into solid scientific research. As vitamins and minerals work in synergy, it is impossible to accurately assess their effects as separate entities. For example, vitamin A and vitamin D are needed for magnesium and calcium absorption; vitamin C works with vitamin E and vitamin E works with selenium.

And whether nutrients are absorbed is also dependent on many factors. Phytic acid and oxalic acid in plant foods like soy and certain raw vegetables, for example, can block absorption of many minerals. Endocrine insufficiencies and lack of beneficial intestinal flora may inhibit nutrient absorption, even though the nutrients are plentiful in the food consumed.

Added to this is the fact that vitamin and mineral content of our foods varies enormously. Researchers cannot rely on nutrient tables to determine the quantities of vitamins and minerals their patients are consuming. They must analyze all the foods eaten to get accurate numbers—an expensive undertaking.

Scientists may attempt to get around this problem by giving synthetic vitamins in pill form, but this practice presents problems as well. Synthetic vitamin D2 added to milk actually has the opposite effect of naturally occurring vitamin D complex, causing decalcification of the hard tissues and calcification of the soft tissues, including the soft tissues of the arteries.[18] For this reason, D2 has been quietly dropped as an additive and replaced by D3, but there is evidence that synthetic D3 is poorly absorbed.[19] Synthetic vitamin E has had disappointing results in trials[20]—the Shute brothers actually used wheat germ oil, a source of natural vitamin E complex. Synthetic vitamins B1 and B2 can cause imbalances affecting the

utilization of B6. In general, vitamins from food work more efficiently and are needed in smaller quantities than synthetic vitamins. Animal studies indicate that minerals taken in as a part of whole foods have more beneficial effects than those given as supplements.

Vitamins and minerals can be ineffective or even toxic in large amounts. Individuals with high levels of serum vitamin C had no better long term survival rates that those with levels that were in the normal range.[21] The single negative study showing that magnesium had a worsening effect on CHD survival employed a far higher dose of magnesium than in the other studies.[22]

These complications do not mean that the effects of vitamins and minerals on cardiovascular health cannot be studied. It does mean that these studies must be performed with great care. Experts in the biochemistry of human nutrition should be involved in the design of such studies—something that rarely occurs. Study design must also include built-in protection against bias—from both those who are antagonistic to the view that nutrition plays a role in heart disease and those who may be too eager to embrace a strategy that relies on supplements.

Many opportunities to find dietary causes of heart disease have been squandered. Dr. Price's research on butter and heart disease, for example, could not be repeated today, partly because Americans no longer consume foods grown locally and partly because most have given up eating any butter at all. Data from the 1960s cited by Ancel Keys in his Seven Countries Study found a fivefold difference in rates of heart disease between Crete and Corfu.[23] Keys and his colleagues had a unique opportunity to look at subtle dietary differences, including differences in soil composition, water content and cooking methods, because both populations consumed mostly locally grown food at the time but probably no longer do. Unfortunately, no one pursued this line of research.

Adventures in Macronutrient Land

Macronutrients are the larger components of our food—protein, carbohydrates and fats. Proponents of the lipid hypothesis have zeroed in on the fat component of our diet, blaming either all fats or just saturated fats for the CHD epidemic. The "prudent" diet calls for

reduction of fat consumption to 30 percent of caloric intake and of saturated fat consumption to just 10 percent of caloric intake, or less than two tablespoons of saturated fatty acids in a diet of 2400 calories.

What clues can we derive from a study of lipid consumption patterns? One is that the actual amount of fat in the diet probably does not matter (except when it is so low as to result in deficiencies). The amount of fat in the American diet has held fairly steady at 35-40 percent of calories for the last 90 years, during the period when rates of heart disease were rising. The Masai, with 60 percent of their calories from fat, are free of heart disease. The traditional diet of the Eskimo and the North American Indians contained as much as 80 percent of calories as fat and there is no indication that they suffered from heart disease.

What consumption patterns do indicate, however, is that it is the type or quality of fat that matters. Ninety years ago, Americans consumed mostly animal fats—lard, butter and tallow from pasture-fed animals. These fats were stable and provided many important nutrients. Today most of the fats in the American diet are derived from plants—as liquid vegetable oils or oils that have been hardened through the process of hydrogenation. Large amounts of calories from polyunsaturated vegetable oils are new to the human diet and should certainly be explored more fully as a contributing factor.

There are several ways in which modern vegetable oils may have an adverse effect on CHD. First, because of modern processing methods, they tend to be rancid. Rancid fats contain large numbers of free radicals, molecules with unpaired electrons that are highly reactive. Free radical damage in the arteries is thought to be an important factor in the initiation of plaque. Secondly, these oils lack vitamins A and D found in animal fats and through processing are likely to be shorn of naturally occurring vitamin E and other antioxidants.

Another problem is that when polyunsaturated oils are consumed in large amounts, imbalances can occur that may predispose to heart disease. Research suggests that traditional diets contained from four to 10 percent of calories as polyunsaturated fatty acids with a ratio of about twice as many omega-6 fatty acids (mostly lin-oleic acid) as omega-3 fatty acids (mostly a-linolenic acid).[24]

Individuals who are trying to avoid saturated fats often end up with over 20 percent of calories as polyunsaturated fatty acids. The situation is further complicated by the fact that commercial vegetable oils contain mostly omega-6 fatty acids. The body uses these types of fatty acids to make localized hormones, called prostaglandins, that initiate the process of

blood clotting and of inflammation. This is an important mechanism. Without it, we would bleed to death when we cut ourselves and wounds would not heal. The problem occurs when these clot- and inflammation-promoting prostaglandins are not balanced by prostaglandins that inhibit clotting.

Many of the anti-inflammatory and clot-inhibiting prostaglandins are made from omega-3 fatty acids, of which there are very few in commercial vegetable oils, or indeed in fruits, vegetables, fish and eggs raised by modern farming methods. Thus, when the diet contains too much of omega-6 fatty acids and not enough of omega-3 fatty acids, there may be a tendency to form blood clots leading to heart attacks.[25]

The research on omega-3 fatty acids is not conclusive. While some studies indicate that omega-3 fatty acids may be helpful, others showed no effect. One explanation for this may be found in the fact that saturated fats help the body store and use omega-3 fatty acids more effectively.[26] Therefore, we would expect to find a correlation with consumption of omega-3 fatty acids and low rates of heart attacks in populations that use traditional diets containing saturated animal fats. But when omega-3 fatty acids are given to individuals who are avoiding saturated fats, the outcome may not be positive. In fact, there is evidence that overconsumption of omega-3 fatty acids in a diet lacking in saturated fats may actually be bad for the heart. In test animals, diets high in canola oil, which is relatively high in omega-3 fatty acids but low in saturated fats, caused fibrotic heart lesions, vitamin E deficiencies and abnormal changes to the blood platelets.[27] When the diets contained higher levels of saturated fats, these problems did not occur.

Although research on trans fatty acids found in hydrogenated fats has not received much publicity, it adds up to a strong case for the theory that these manufactured fats contribute to heart disease.[28] The tragedy is that those who are trying to avoid saturated fats and cholesterol will probably eat more trans fatty acids, because these are used in foods promoted as low in saturated fat and cholesterol.

Those who are trying to avoid eating lots of fat often replace fat calories with carbohydrate calories, usually calories in the form of refined flour and sugar. Yet several researchers have published studies linking consumption of refined carbohydrates, particularly sugar, with increased heart disease, including Yudkin in the 1950s and Lopez in the 1960s. Yudkin found

that use of sugar was associated with increased adhesiveness of the blood platelets, increased blood insulin levels and increased blood corticosteroid levels.[29]

In addition, sugar consumption is associated with increased incidence of diabetes, and diabetics are said to be prone to heart disease. One researcher has proposed that a diet high in any type of carbohydrate, including carbohydrates from whole cereal grains, is associated with CHD.[30] Of course, many products containing white flour and sugar also contain high levels of trans fatty acids and improperly prepared whole grains contain phytic acid that can block the uptake of important minerals including magnesium, zinc and copper.

Protein, the third macronutrient, also plays a role in heart health. When protein intake is inadequate, the heart muscle shrinks and cannot perform effectively.[31] Soy-based liquid protein drinks and other foods predispose to arrhythmias.[32] High protein diets that do not contain fats, particularly animal fats, can deplete stores of vitamin A and D and consequently interfere with mineral assimilation.[33]

Cholesterol Again

By now you know that the cholesterol that our bodies make, and that we get from traditional foods, does not cause heart disease. But cholesterol, like polyunsaturated fatty acids, may become oxidized or rancid when it is processed at high temperatures. In early experiments with vegetarian rabbits, purified solutions of processed cholesterol were used, cholesterol that was rancid or oxidized. Oxidized cholesterol accumulates in the foam cells that are involved in the buildup of pathogenic plaque.[34]

Rancid or oxidized cholesterol occurs in powdered eggs and milk, both used in many processed foods. Powdered milk is added to lowfat milks to give them body.

Another type of cholesterol is Lp(a) which occurs in humans, other primates and guinea pigs, organisms that do not manufacture vitamin C. Nobel laureate Linus Pauling and his colleague Mathias Rath proposed that our bodies produce Lp(a) to compensate for low levels of vitamin C.[35] They caused atherosclerosis in guinea pigs by depleting their bodies of vitamin C. Vitamin

C depletion caused Lp(a) to appear in the plaque. A high level of Lp(a) is a risk factor for heart disease.[36] That does not mean the Lp(a) is the cause. The cause may be vitamin C deficiency in association with other factors, such as low levels of vitamin B3 (niacin), which also lowers Lp(a). Consumption of trans fatty acids causes levels of Lp(a) to rise while consumption of saturated fats lowers blood levels of Lp(a).[37]

Infection and Heart Disease

A number of pathogens have been associated with the development of CHD or have been found in the atherosclerotic lesions at autopsy, including both viruses and bacteria.[38] These pathogens have been around as long as man has lived on the earth. The culprit, therefore, is not the microbes but a compromised immune system which can no longer deal with them appropriately. A healthy immune system depends on an array of nutrients, including vitamin A, vitamin C and various minerals that play an antioxidant role.

One of the most tragic aspects of the cholesterol campaign is that it has caused Americans and Europeans to abandon fats that provide protection against infection. Not only do animal fats carry vitamin A, they also contain palmitoleic acid, a 16-carbon monounsaturated fatty acid that has strong antimicrobial properties. Butterfat and coconut oil contain fatty acids that have similar properties. They protect against viruses and pathogenic bacteria and enhance the immune system. Areas of the world where coconut is consumed have low levels of heart disease.

Thyroid

Thyroid insufficiency has been identified as a risk factor for heart disease, but treatment with thyroid hormone replacement does not necessarily improve the outcome.[39] Hormones taken orally may have unexpected effects compared to those produced by the body, effects that may increase the risk of heart disease, such as the provocation of arrythmias. Thyroid health depends on iodine status, but other factors are involved. Vitamin A, for example, plays a key

role in thyroid health.[40] As individuals with poor thyroid function have difficulty converting carotenes in plant foods into true vitamin A, they must obtain adequate vitamin A from animal foods. Unfortunately, patients with thyroid problems are often advised to follow a lowfat diet because they are prone to heart disease.

Other Theories

Many other theories have been proposed to account for the current epidemic in CHD: Chlorine and fluoride added to water; pesticides that mimic human estrogens or that provoke free radical reactions; carbon monoxide fumes; industrial chemicals; artificial lighting; synthetic vitamins; minerals that are toxic or that are consumed in toxic amounts; pasteurization and homogenization of milk; legal and illegal drugs; consumption of coffee and other stimulants; and additives in processed foods. Most are factors unique to the twentieth century and all need further study.

But who will do this work? Even today, all but a small fraction of the research dollar still goes to further study of the lipid hypothesis, and vested interests have the power to prevent funding for studies that may prove embarrassing.

Solutions

How can we protect ourselves against heart disease? Based on what we have learned from the scientific studies, it is possible to formulate a set of guidelines for heart disease prevention, guidelines that include both avoidance of external stresses and common sense dietary advice. Not all external stresses can be avoided, not in today's fast-paced industrial age, but a good diet can provide many factors that help the body deal with environmental toxins and high levels of stress.

There are many points contained in the following guidelines that can be debated but one thing is certain: If you are still afraid of saturated fats and cholesterol, you will find yourself on the wrong dietary path. If you are avoiding foods containing saturated fat and cholesterol, you will not only deprive your body of vital nutrients, but the foods that you consume as substitutes will contain many components—polyunsaturated oils, trans fatty acids, refined sugar—that have been associated with increased rates of heart disease.

Ten Commandments for Avoiding CHD

1. Don't smoke. If you find it impossible to quit, at least try to cut back and smoke only additive-free cigarettes. Smokers should avoid polyunsaturated oils at all costs.

Saturated fats and vitamins A and D are particularly protective of the lungs.
2. Exercise regularly but you needn't overdo. A brisk daily walk, 10 minutes on the trampoline, swimming, and sports are all appropriate.
3. Avoid overweight by eating nutrient-dense foods and keeping sweets to a minimum, but avoid crash dieting.
4. Don't work too hard. Counteract stress by doing something that you love to do everyday. During periods of unavoidable hardship or loss, increase consumption of foods rich in protective nutrients.
5. As much as possible, avoid exposure to fumes, chemicals, pollutants and pesticides.
6. Avoid all processed foods labeled "lowfat" or that contain polyunsaturated vegetable oils, hydrogenated fats, white flour, refined sugar and additives.
7. Consume high-quality animal products including a variety of seafood and milk, butter, cheese, eggs, meat, fats and organ meats from animals raised on green pasture.
8. Consume a variety of fresh vegetables and fruits, preferably organically grown.
9. Ensure sufficient mineral intake by using whole dairy products; bone broths; and whole grains, legumes and nuts that have been properly prepared to reduce phytic acid and other factors that block mineral absorption.[41]
10. Supplement the diet with foods rich in protective factors including small amounts of cod liver oil (vitamins A and D); wheat germ oil (vitamin E); flax oil (omega-3 fatty acids); kelp (iodine); brewers yeast (B vitamins); desiccated liver (vitamin B12); rose hip or acerola powder (vitamin C); and coconut oil (antimicrobial fatty acids).

Sidebars

Cholesterol – Your Body's Best Friend

Cholesterol is the body's repair substance. Scar tissue contains high levels of cholesterol. When your arteries develop irritations or tears, cholesterol is there to do its job of patching up the damage.

Along with saturated fats, cholesterol in the cell membrane gives our cells necessary stiffness and stability. When the diet contains an excess of polyunsaturated fatty acids, these replace saturated fatty acids in the cell membrane so that the cell walls actually become flabby. When this happens, cholesterol from the blood is "driven" into the tissues to give them structural

integrity. This is why serum cholesterol levels may go down temporarily when we replace saturated fats with polyunsaturated oils in the diet, even though the body's overall cholesterol levels actually go up.

Cholesterol acts as a precursor to vital corticosteroids, hormones that help us deal with stress and protect the body against heart disease and cancer; and to the sex hormones like androgen, testosterone, estrogen and progesterone. Cholesterol is also a precursor to vitamin D and to the bile salts. Bile is vital for digestion and assimilation of fats in the diet.

Recent research shows that cholesterol acts as an antioxidant. This is the likely explanation for the fact that cholesterol levels go up with age. As an antioxidant, cholesterol protects us against free radical damage that leads to heart disease and cancer.

Cholesterol is needed for proper function of serotonin receptors in the brain. Serotonin is the body's natural "feel-good" chemical. Low cholesterol levels have been linked to aggressive and violent behavior, depression and suicide.

Mother's milk is especially rich in cholesterol and contains a special enzyme that helps the baby utilize this nutrient. Babies and children need cholesterol-rich foods throughout their growing years to ensure proper development of the brain and nervous system.

Dietary cholesterol plays an important role in maintaining the health of the intestinal wall. This is why low-cholesterol vegetarian diets can lead to leaky gut syndrome and other intestinal disorders.

Men who have cholesterol levels over 350 mg/dl are at slightly greater risk for heart disease. For women, there is no greater risk for heart disease, even at levels as high as 1000 mg/dl. In fact, mortality is higher for women with low cholesterol than for women with high cholesterol.

Cholesterol readings are highly inaccurate. They vary with the time of day, time of the patient's last meal, levels of stress and the type of test used. Tests for HDL and LDL are especially subject to inaccuracies.

All About Angiopathy

One method doctors use to determine the effectiveness of various drug and dietary treatments for heart disease is coronary angiography. It is performed by injecting iodine atoms into the blood vessel and taking an X-ray. A narrow and flexible plastic tube is inserted into the femoral artery in the groin and pushed gently upwards through the aorta, the chief artery of the human body, until it reaches the vessel to be investigated, such as the coronary vessels, those that provide the heart muscle with blood. When the tip of the catheter reaches the entrance of one of the coronary vessels, the iodine solution is slowly injected.

Let us keep in mind that a change in diameter of the coronary artery is nothing but a surrogate outcome. It is assumed that a widening of a coronary vessel on an X-ray means less atherosclerosis and thus a better chance to avoid a heart attack, but this is only an assumption. It is also important to realize that the differences observed in vessel diameter involve only very small changes, changes measured in hundreths of a millimeter.

Artery walls are surrounded by smooth muscle cells. When such cells contract, the artery narrows. When they relax, it widens. Various factors may stimulate the smooth muscle cells of the coronary arteries to contract including mental stress, anxiety, exposure to cold and even a sustained hand grip. The latter effect was studied by Dr. Greg Brown who found that a hand grip sustained for a few minutes was followed by a 35 percent decrease in the vessel diameter. Since almost all heart disease patients receive drugs that relax the coronary vessels, and since the insertion of the tube into the groin and upward into the aorta is in itself a stressful experience—one that might cause the patient to clasp his hands in a sustained grip—changes observed through angiography can hardly have any value in the study of diet or drugs.

There are more uncertainties. Dr. Seymor Glagov and his colleagues from University of Chicago studied the hearts of 136 deceased individuals and found that when vessels become sclerotic, they actually widen to compensate for the narrowing brought about by the

deposition of cholesterol in their walls. In fact, this widening overcompensates for the deposition until the cholesterol deposits occupy about 40 percent of the area beneath the muscle wall. Only thereafter does the vessel become steadily narrower. In other words, an increase of vessel diameter may be due to better relaxation of the vessel wall or

disappearance of cholesterol in a highly sclerotic vessel; but it also could be due to a compensatory widening during the first stages of cholesterol deposition. Yet angiographic results are used to justify various cholesterol-lowering regimens, from lowfat diets to cholesterol-lowering drugs.

Other Theories Proposed to Explain the CHD Epidemic

Price	Deficiency of fat soluble vitamins

Yudkin	Refined carbohydrates
Kummerow	Trans fatty acids from hydrogenated fats
Hodgson	Excess omega-6 from refined vegetable oils
Addis	Oxidized cholesterol and oxidized fats
Shute	Vitamin E deficiency
Pauling	Vitamin C deficiency
McCully	Deficiency of folic acid, B6 and B12
Annand	Heated milk protein
Webb	Protein deficiency
Anderson	Magnesium deficiency
Huttunen	Selenium deficiency
Ellis	Microbial agents (viruses, bacteria)
Benditt	Monoclonal tumor theory
de Bruin	Thyroid deficiency
LaCroix	Coffee consumption
Morris	Lack of exercise
Stern	Exposure to carbon monoxide
Smith	Changes & fashions in reporting cause of death

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