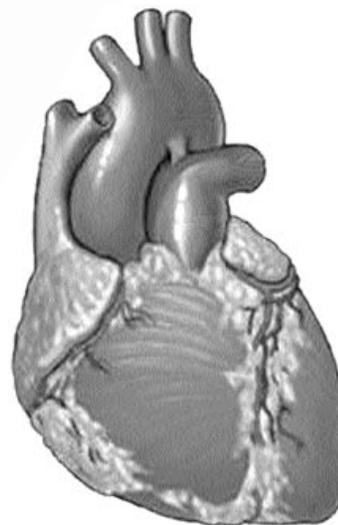
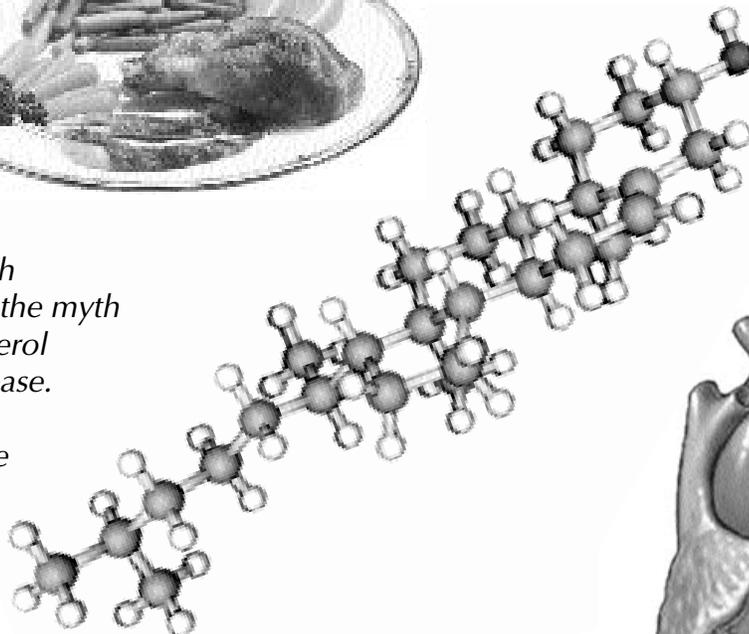


High blood cholesterol and heart attacks are co-symptoms of an unhealthy dietary regime—one with too much sugar and carbohydrates, the authors argue.

Two public health scientists dissect the myth that high cholesterol causes heart disease. But, take heart: they also propose a reasonable course of action for the health-conscious.



# The Role of Cholesterol and Diet In Heart Disease

by Alice Ottoboni, Ph.D. and Fred Ottoboni, M.P.H., Ph.D.

Shortly after World War II, the increasing incidence of coronary heart disease<sup>1</sup> prompted the medical community, with government support, to initiate a massive and long-term study of what might possibly be the cause. The study, known as the Framingham Study, enrolled a large num-

ber of families whose diets, lifestyles, and environments were surveyed and their medical and laboratory findings routinely recorded. After several years, it was noted that there was a positive association between blood cholesterol levels and incidence of heart attacks.

During this same period, nutrition pioneer Ancel Keys and his wife, Margaret, discovered a regional culinary tradition they named the Mediterranean diet. Convinced that this diet, which was considered low in animal fats, protected against

*The authors are long-time public health scientists, now retired. This article is adapted from their book, The Modern Nutritional Diseases: Heart Disease, Stroke, Type-2 Diabetes, Obesity, Cancer and How to Prevent Them, published in 2002 by Vincente Books, Sparks, Nev., P.O. Box 50704, Sparks, NV 89435. The cover price of the book is \$29.95. The publisher sells the book directly by mail order for \$19.95 with free shipping in the USA, with payment by check (made out to Vincente Books, Inc.).*

1. Heart disease is a catch-all for a wide and varied group of afflictions of the heart that compromise its ability to perform adequately its job of pumping blood to all parts of the body to deliver nutrients and remove waste products. The heart disease that is the most common cause of cardiovascular disability and death, better known as a heart attack, is referred to as coronary heart disease (CHD), and is the subject of this discussion.

coronary heart disease, they set out to obtain statistics on deaths from coronary heart disease and fat consumption from countries that had such statistics. They found strong associations between the two in six countries. These associations were taken as proof of cause and effect and, with support of the cholesterol-heart disease suggestion from the Framingham Study, gave rise to the *lipid hypothesis*, also referred to as the *cholesterol hypothesis*, for coronary heart disease.

The research of Ancel Keys and his colleagues became widely publicized and popular. The nutrition community enthusiastically adopted the lipid hypothesis, and clinical and epidemiological research in the field proliferated. The lipid hypothesis was accepted as fact by the medical community, and, in 1956, representatives of the American Heart Association presented the hypothesis on national television.

The public was informed that the cause of coronary heart disease was butter, lard, beef, and eggs. The diet recommended by the American Heart Association replaced these traditional foods with vegetable seed oils, margarine, chicken, bread, and cereals. This was the birth of the heart-healthy diet that soon became formalized in the government-sponsored low-fat, high-carbohydrate dietary policy that was adopted and followed by millions of Americans for over fifty years.

An in-depth examination of the body of literature on which the government-sponsored dietary policy was founded reveals serious flaws in experimental design and data evaluation. The most flawed of the studies that introduced the lipid hypothesis was that of Ancel Keys. Although Keys presented data from six countries that had statistics on heart attack deaths and fat consumption, there were actually over twenty that had such sta-

## Dietary Changes and Disease

Today, the United States is experiencing epidemics of cardiovascular diseases, obesity, type-2 diabetes, and cognitive disturbances, such as senile dementias and Alzheimer's disease. These chronic debilitating diseases are not new diseases, but old diseases that were uncommon prior to 1900, but have now become sources of great societal and economic concern for the health care community and the government. For example, heart disease was rare before 1892. Twenty years later, heart disease was responsible for slightly more than 10 percent of all deaths in advanced nations. By 2001, it was responsible for 31 percent of all deaths in the United States. With the exception of the age group 45-64 years, in which surgical intervention is most common, deaths from heart disease in the United States have continued to rise.

Since the late 19th Century, a gradual but dramatic change in the American diet also has taken place. The family farm has largely disappeared, and an agricultural industry based on mechanization has emerged. Better transportation and new methods of food storage, preparation, and preservation have reduced costs and brought a wider array of fresh and processed foods to the American table.

There is no argument that the American diet has undergone marked changes over past decades. It is also clear that attack rates of chronic debilitating diseases have increased significantly during the same period. But are dietary changes that have occurred over the past century the cause of these diseases?

The concept that large-scale changes in dietary patterns can result in disease is not novel or unreasonable. Considerable historical precedent for this concept can be found in the classic nutritional deficiency diseases. Pellagra and beriberi are just two of the long-forgotten nutritional diseases that once were scourges of mankind. The cause of pellagra is deficiency of the B-vitamin niacin. Pandemics of pellagra were rampant in poor countries where the traditional

diet of meat, eggs, and dairy products had been replaced by corn as the staple food. Corn was inexpensive and abundant but essentially devoid of niacin.

Beriberi occurred not because of food cost but because of food instability. Whole grains have short storage lives because oils in the germ readily become rancid. To solve this problem, machines were developed to remove the germ. Unfortunately, along with the germ, they removed the B vitamins. Substitution of refined grains for the rancid-prone whole grains caused mass epidemics of beriberi, a deficiency primarily of the B-vitamin thiamin.

### Dietary Changes in Last 100 Years

What are the large-scale dietary changes that have accompanied the current epidemics of chronic debilitating diseases? Before 1900, sugar was a luxury item obtained from sugar cane. Development of the sugar beet industry reduced the cost of sugar, and the sugar bowl became a fixture on the dining table. In addition, new processes to obtain sugar from corn made sweet commercial products, particularly soft drinks, widely available and affordable. The use of sugars has doubled in the last century.

After World War II, abundant supplies of grains, combined with advances in commercial production and distribution of bakery products and pastas, made refined wheat products common staples. About this time, prepackaged breads, cookies, and other ready-to-eat food products found growing public acceptance. The result has been a great increase in dietary use of foods high in sugars and starches.

Development of new sources of edible fats and oils, primarily from vegetable seeds, began in the early 1900s. Prior to that time, the major sources of dietary fat were butter, lard, coconut oil, and olive oil. The new oils required a process called hydrogenation to keep them from smoking during cooking and becoming rancid in storage.

tistics available at the time. Why did Keys exclude the latter data? The fact is that the countries excluded did not show the same association between fat consumption and deaths from heart attacks. In a thoughtful analysis in *The Cholesterol Myths*, Swedish physician Uffe Ravnskov clearly explained that if Keys had included data from all 22 countries, support for Keys's hypothesis would have been weak. Ravnskov went on to conclude that, based on a thorough review of all relevant scientific literature, the lipid-cholesterol hypothesis is a fallacy.

During the next two decades, many nutrition studies investigated the diet/heart connection. Unfortunately, the epidemiological competence of many investigators left much to be desired. In numerous studies, only fat consumption of participants was recorded, to the exclusion of all other diet components. To their credit, a few scientists reported that people who

consumed high levels of saturated fat also consumed high levels of sugar. They implicated sugar as the major dietary cause of coronary heart disease with convincing data correlating coronary heart disease to sugar consumption, but their data were ignored. By this time, the direction of research was firmly controlled by proponents of the lipid hypothesis, which already had been expanded to include dietary cholesterol as a culprit.

Even in face of the general acceptance of the lipid hypothesis, a number of scientists were skeptical, claiming that, despite the numerous studies of population groups in many countries, epidemiological methods had not identified the causes of cardiovascular diseases. An extensive review of the literature prior to 1977, including data from the massive Framingham Study, confirmed that the information available concerning the impact of diet on cardiovascular disease did not produce a clear picture of cause and effect. Thus, long after the public was urged to change its dietary habits, the true causes of cardiovascular diseases were still obscure.

Despite this confusing situation, it is apparent that sometime before 1970, those responsible for food policy in the United States had decided that dietary fat, particularly saturated fat and cholesterol, was the cause of high cholesterol, high blood pressure, and coronary heart disease. As a result, the low-fat, high-carbohydrate, "heart-healthy" diet sponsored by the government was adopted by millions of Americans. Thus, those who governed national dietary policy more than 30 years ago directed a whole nation, using a flawed road map, down a dietary path they promised would lead to a healthier life. The map seems to have been drawn using the simplistic but faulty logic that the fat and cholesterol that clogs arteries must have come from the fat and cholesterol in the diet. That this logic is not founded on scientific fact is easily demonstrated by the biochemistry of fat and cholesterol synthesis found even in college textbooks.

Proponents of the lipid hypothesis argue that some patients have success in lowering blood cholesterol on a very low-fat Pritikin-like diet that eliminates animal fats. This is true, but not because the diet is very low in fat, but because it is very low in calories. If a body has no calories in excess of its daily energy requirements, it cannot have calories to divert to cholesterol synthesis. Further, Pritikin-like diets are not useful as a long-term strategy because, being very low in fat, they tend to be deficient in protein and essential fatty acids and because lowering blood cholesterol, per se, does not prevent coronary heart disease.

### What Is Cholesterol?

Cholesterol is the principal sterol (steroid alcohol) in higher animals, and is an indispensable constituent of all cells and fluids of the body. It is a large, complex chemical present in the lipid fraction of the diet in combination with the true fats—the fatty acids and their triglycerides. Because the true fats are the most prevalent and familiar lipids in the diet, the term *fat* has become synonymous with *lipid* in nutrition vocabulary, with the result that cholesterol is often erroneously classed as a fat.

Cholesterol comprises up to 50 percent of cell membrane

Hydrogenation not only produces trans fats but also virtually destroys one of two essential fatty acids required for life and health. As a result, Americans are now consuming an average of 40 pounds a year of fats that never before in history were part of the human diet. These new fats have largely replaced animal fats that, unlike vegetable seed oils, contain a healthful balance of the essential fatty acids.

As radical as these dietary modifications were, even more profound changes resulted from acceptance in the late 1940s, by the nutrition and medical communities, of the lipid hypothesis originally proposed by Ancel Keys. Saturated fats and cholesterol were identified as the cause of cardiovascular diseases, with the result that red meat, eggs, and animal fats were demonized. The consumption of these foods declined markedly, and the void was filled with starches, sugars, and vegetable fats.

The dietary changes that have occurred over the past century have had a marked effect on the nutrition of the American public. What is the relationship between diet and nutrition, and why does a change in one influence the other? Diet refers to the foods that comprise the daily fare, and nutrition refers to the substances (nutrients) that the body requires for growth, maintenance, and repair, which are provided by foods.

Because all foods are composed of the same few basic nutrients (proteins, carbohydrates, fats and other lipids, vitamins, and minerals), except in different proportions, the nutritional quality of a diet is completely dependent on what foods are selected to be in it. Thus, if diet contributes to or causes a chronic debilitating disease, the fault must lie in the kinds and quantities of the foods that are selected for the diet. Heart disease, probably the best known and most feared of the modern nutritional diseases, can serve as an example for all such diseases of how diet can cause illness.

lipids, where its function is to regulate membrane flexibility. In addition to other vital biochemical and physiological functions, cholesterol is the starting point for the synthesis of several groups of very important biochemicals, including the male and female sex hormones, vitamin D, and the bile acids.

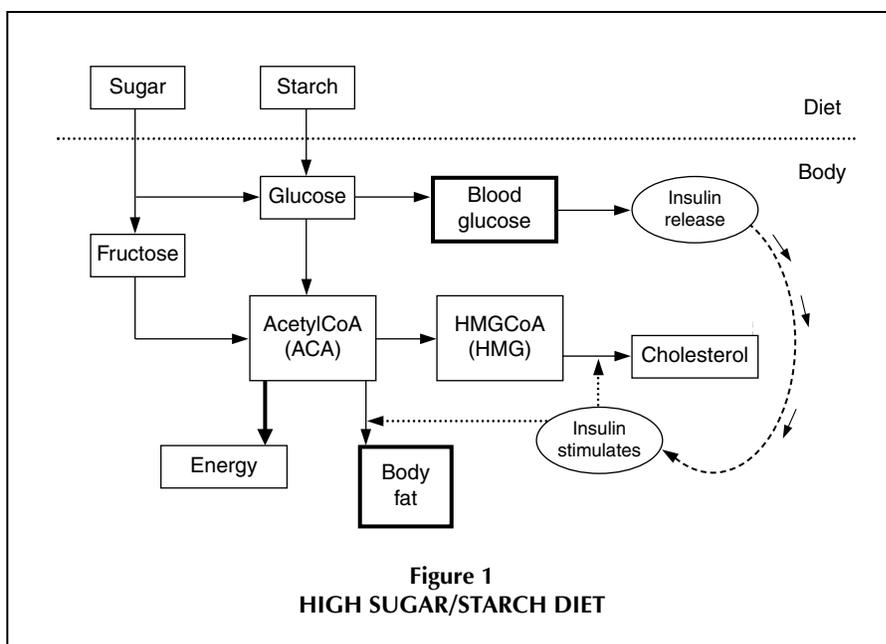
Cholesterol is transported in the blood in combination with specialized proteins. These combinations form lipid-protein molecules of varying density, hence the familiar LDLs and HDLs (low-density and high-density lipoproteins) that are routinely measured in medical examinations. In general, LDL transports cholesterol to all parts of the body where it is needed. Because one of these needs is to deposit cholesterol over inflamed arterial lesions, LDL has been labeled as “bad.” This designation is unfortunate because the deposition is a mechanism to protect against further damage, rather than a cause of damage. Conversely, HDL is labeled “good” because it carries cholesterol away from the body for eventual disposal.

Synthesis of cholesterol occurs in virtually all cells of the body, including the walls of arteries, but the major portion is synthesized by liver cells. Synthesis is controlled by mechanisms that turn the process on and off, depending on the body’s needs. Under normal circumstances, the body produces no more cholesterol than its life processes require. Excess cholesterol is excreted from the body by the liver, via the gallbladder, into the small intestine, and eliminated with the feces.

Despite considerable scientific data showing that high blood cholesterol and heart attacks do not have a causal relationship, but rather are co-symptoms of an unhealthy dietary regime, the nutritional and medical communities continue to insist that high blood cholesterol is dangerous and must be reduced. This fixation on cholesterol as a marker for coronary heart disease makes it essential that patients, and potential patients who are concerned for their long-term health, not only understand the significance of blood cholesterol, but also know how diet is responsible for high blood cholesterol levels.

### Diet and Cholesterol

Dietary cholesterol, which is present only in foods from animal sources, is poorly absorbed from the intestines. Cholesterol in foods does not cause a significant increase in a person’s normal level of blood cholesterol, despite misinformation to the contrary. Very high intakes of dietary cholesterol may increase blood cholesterol levels a few percent, but intakes below 800 milligrams a day have little impact. This is because healthy individuals maintain relatively constant levels of blood cholesterol regardless of the quantity of

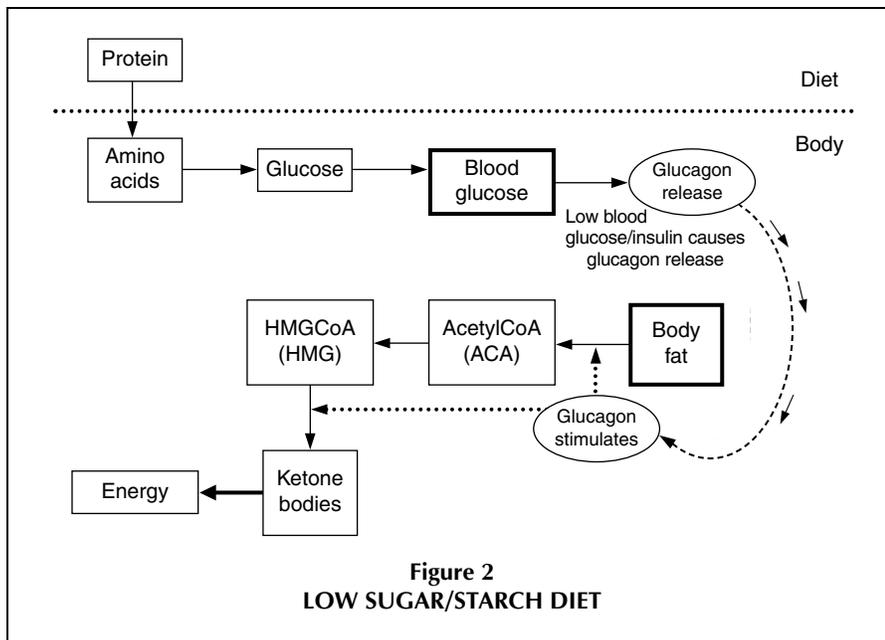


cholesterol in the diet. The amount of cholesterol the body synthesizes is reduced by whatever quantity of cholesterol is absorbed from the intestinal tract. The more cholesterol in the diet, the less cholesterol the body makes. The human body is a very energy-saving machine in all aspects of its biochemistry, not just in cholesterol synthesis. It does not waste energy in making what is provided by an outside source.

The biosynthesis of cholesterol is governed primarily by the hormone insulin, the secretion of which depends on blood glucose level. Figure 1 is a simplified diagram showing the pathways that dietary carbohydrates, which are largely sugar and starch, follow on their way to cholesterol. Starch is broken down to glucose, which is absorbed into the body where it causes release of insulin from the pancreas. Then, in a series of about 10 biochemical reactions called glycolysis, glucose yields acetyl CoA (ACA). Sugar is absorbed into the body where it splits in two, to yield glucose and fructose. The glucose half goes to ACA via glycolysis, but fructose bypasses glycolysis and goes directly to ACA.

A major function of ACA is to provide energy for life processes, but it is also a precursor of other important biochemicals, including cholesterol and fats. Figure 1 shows two multi-step pathways for ACA. One goes through HMG CoA (HMG) to cholesterol, and the other goes to body fat. Both of these pathways require insulin to proceed. In brief, the amount of glucose in the blood dictates the amount of insulin produced by the pancreas, and this insulin, in turn, directs ACA to go to cholesterol and body fat.

When the blood glucose level drops, as with low-carbohydrate diets, the insulin level falls and the pancreas secretes its complement hormone glucagon. Figure 2 shows this shift in metabolic pattern to one that spares glucose for its critical homeostatic role in blood. In this glucagon-governed pattern, the glucose required to main-



tain blood glucose levels is made from amino acids provided by protein. To further spare glucose from being diverted to provide energy, glucagon causes the release of fat from stored body fat, and converts it to HMG through ACA. Then, instead of HMG going to cholesterol, it is converted to ketone bodies, which are used to provide energy.

The metabolic pathway for a diet balanced in nutrients is essentially a composite of Figures 1 and 2. After a full meal, Figure 1 dominates, and several hours later, when dietary nutrients are not available and hunger pangs occur, Figure 2 dominates.

Even though dietary cholesterol does not cause a significant increase in normal cholesterol levels, imbalances of other nutrients in the diet can force the body to synthesize more cholesterol than it requires. Such dietary imbalances override the cholesterol-synthesizing control mechanisms, and cause the body itself to overproduce cholesterol by the pathway shown in Figure 1. This excess production of cholesterol results in an abnormal increase in blood cholesterol.

High levels of blood cholesterol are the result of long-term dietary excesses of sugars and starches. The sugars are provided by soft drinks, candy, and sweet bakery products; and the starches are provided by bread, cereals, potatoes, and pastas. The medical significance of high blood cholesterol for coronary heart disease in the absence of arteriosclerosis can be debated, but there is no doubt that high blood cholesterol is a warning signal that the diet is laying the foundation for coronary heart disease. A diet that causes high blood cholesterol promotes obesity, insulin resistance, and chronic inflammation, all of which are powerful risk factors for coronary heart disease, in addition to other chronic debilitating diseases.

Why is the public so ill-informed on the subject of cholesterol? Many studies published in scientific journals over the

past decades have been critical of the lipid hypothesis. These journals, unfortunately, are not usually read by the general public. The mass media, which is the principal source of medical and health information for most people, does not publish information that is counter to the dictates of the establishment. Thus, there has been little recognition by the average citizen that a cholesterol controversy exists in the scientific community.

The public owes a debt of gratitude to Uffe Ravnskov for his role in bringing serious questions about the need for, and dangers of, cholesterol treatment to public attention. Dr. Ravnskov, a Swedish physician and researcher, became concerned about the scientific inaccuracies and misstatements put forth by the anti-cholesterol campaign when it was introduced in Sweden in 1989. As a

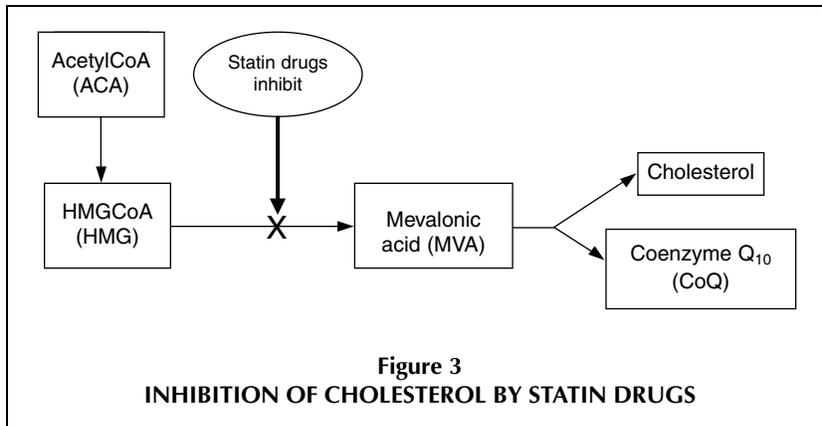
result, Dr. Ravnskov devoted himself to communicating the scientific facts about cholesterol to patients and potential patients through articles, books, and the Internet. Dr. Ravnskov's writings explain, in lay language, the myths concerning the relationship between cholesterol and coronary heart disease.

### The Role of Homocysteine

At this point, *arteriosclerosis* and *atherosclerosis* require definition. Arteriosclerosis refers to hardening of the arteries. It is observed as toughened areas that often contain calcium deposits called plaques. Atherosclerosis refers to an advanced form of arteriosclerosis that is characterized by deposits of cholesterol, fats, and blood clots within the arterial plaques. This distinction is basic to an understanding of the role of nutrients in the development and progression of coronary heart disease.

Arteriosclerosis appears to be a necessary precondition for atherosclerosis. When arteriosclerotic lesions occur, the body deposits cholesterol in the lesion to heal and protect it from further damage. Arteriosclerosis has no relationship to blood cholesterol level. Cholesterol deposition occurs in arteriosclerotic lesions regardless of how low the blood cholesterol level is, and it does not deposit in healthy vessels regardless of how high the cholesterol level is. This explains the seeming paradox of why some people with low cholesterol suffer heart attacks and some people with high cholesterol do not. The explanation can be found in the role of *homocysteine*.

The theory that homocysteine is intimately involved in cardiovascular disease was proposed by Kilmer McCully, a Harvard physician and research scientist, more than 30 years ago. Homocysteine, formed in the body from the amino acid methionine, plays a valuable biochemical role in normal, healthy metabolism; however, like many otherwise valuable biochemicals, homocysteine does damage



when its normal metabolism is disrupted. In the case of homocysteine, the metabolic disruption is caused by deficiencies of three B vitamins, B6, B12, and folic acid, which give rise to excessive levels in the blood. High blood homocysteine damages the walls of arteries and causes them to thicken, lose their elasticity, and form plaques and blood clots. This condition is the arteriosclerosis that predisposes to atherosclerotic diseases, including heart attacks and strokes.

There is ample clinical evidence to support the suggestion that homocysteinemia is a far more accurate predictor of coronary heart disease than is a high cholesterol level. However, because these findings are in conflict with the dogma that cholesterol and fats cause coronary heart disease, they are apparently unacceptable to the medical establishment. Today, clinical laboratories have the ability to measure homocysteine levels as part of routine blood analyses, yet such analyses are rarely requested in medical practice.

Why is it that medical practice essentially ignores the role of homocysteine in coronary heart disease and stresses the importance of cholesterol? Both biochemicals are amenable to being kept within normal values by diet; homocysteine with supplementation of the appropriate B vitamins and cholesterol with a low sugar and starch diet. Further, high blood homocysteine is a valid risk factor for coronary heart disease, whereas high blood cholesterol is no more than a questionable risk factor, except perhaps when accompanied by arteriosclerosis. Thus, the obvious answer to the question is that there are no drugs

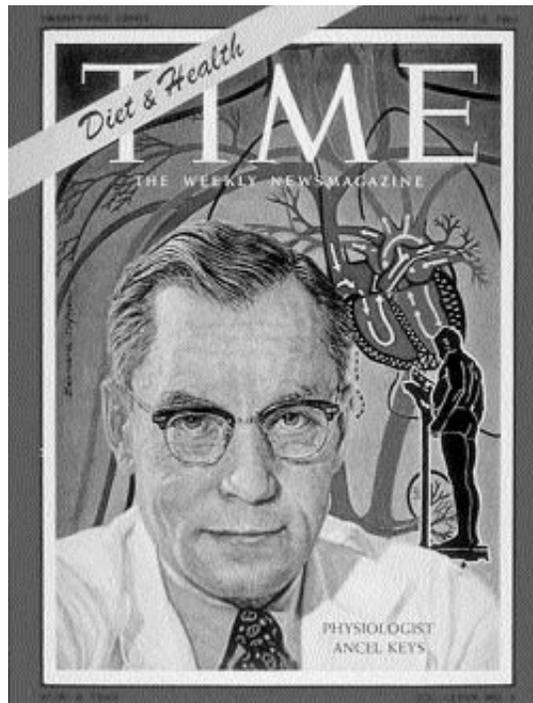
effects—unanticipated biochemical reactions incidental to the desired one. Side effects may or may not be harmful. One harmful reaction of the statins is the inhibition of the body's ability to manufacture coenzyme Q10 (CoQ). Figure 3 shows that MVA is not only a precursor of cholesterol but also a precursor of CoQ. Thus, by inhibiting biosynthesis of cholesterol, statins also inhibit biosynthesis of CoQ.

CoQ has a complex structure that can accept or donate electrons in biochemical reactions. This ability makes it an essential coenzyme partner for a number of enzymes that store or release energy in biochemical reactions. All cells

require CoQ to provide energy for metabolic processes. Heart tissues have a much greater energy demand and, hence, a much greater need for CoQ than most other tissues in the body. Thus, because of this great demand for energy, symptoms of CoQ deficiency are often related to the heart, primarily as congestive heart failure. It is ironic that statin drugs, which are medications prescribed to prevent coronary heart disease, can themselves cause heart disease by creating a deficiency of a biochemical essential for good heart health. Other important symptoms of CoQ deficiency are muscle pains, fatigue, and a general lack of energy.

#### The New Cholesterol Guidelines

Proof of the fact that the medical establishment is fully committed to the cholesterol hypothesis is the publication of the "New Cholesterol Guidelines" for the nation's doctors by the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH) in May of 2001. These guidelines are major clini-



*Manipulating selective data, Ancel Keys popularized the "Mediterranean Diet," which was low in animal fats, as a protection against heart disease. This later evolved into the cholesterol hypothesis. Its general acceptance was noted by Time magazine in this 1961 cover story.*

cal practice guidelines for the prevention and management of high cholesterol in adults. The stated goals of the new guidelines are to reduce the prevalence of high blood cholesterol, better identify people at high risk, and reduce the risk of coronary heart disease.

The impetus for the guidelines was the fact that, despite the decades-long pursuit of the heart-healthy diet by many Americans, coronary heart disease had finally become the number one killer in the U.S., striking down about 500,000 people each year—primarily by heart attack. The guidelines lower the laboratory value for blood cholesterol that would trigger statin drug therapy, and modify conventional dietary recommendations to urge more rigorous reduction of dietary cholesterol and saturated fats.

NHLBI estimated in 2001 that, under the new guidelines, the number of Americans on low-saturated-fat, low-cholesterol dietary treatment would increase from about 52 million to about 65 million, and the number who are prescribed cholesterol-lowering drugs would increase from about 13 million to about 36 million. The guidelines advise physicians that, because Americans at high risk for a heart attack are too often not identified and, as a result, do not receive sufficiently aggressive treatment, cholesterol-lowering drugs should be employed when diet and exercise do not sufficiently lower blood cholesterol.

These new guidelines were presented to the public as if



Courtesy of Uffe Ravnskov

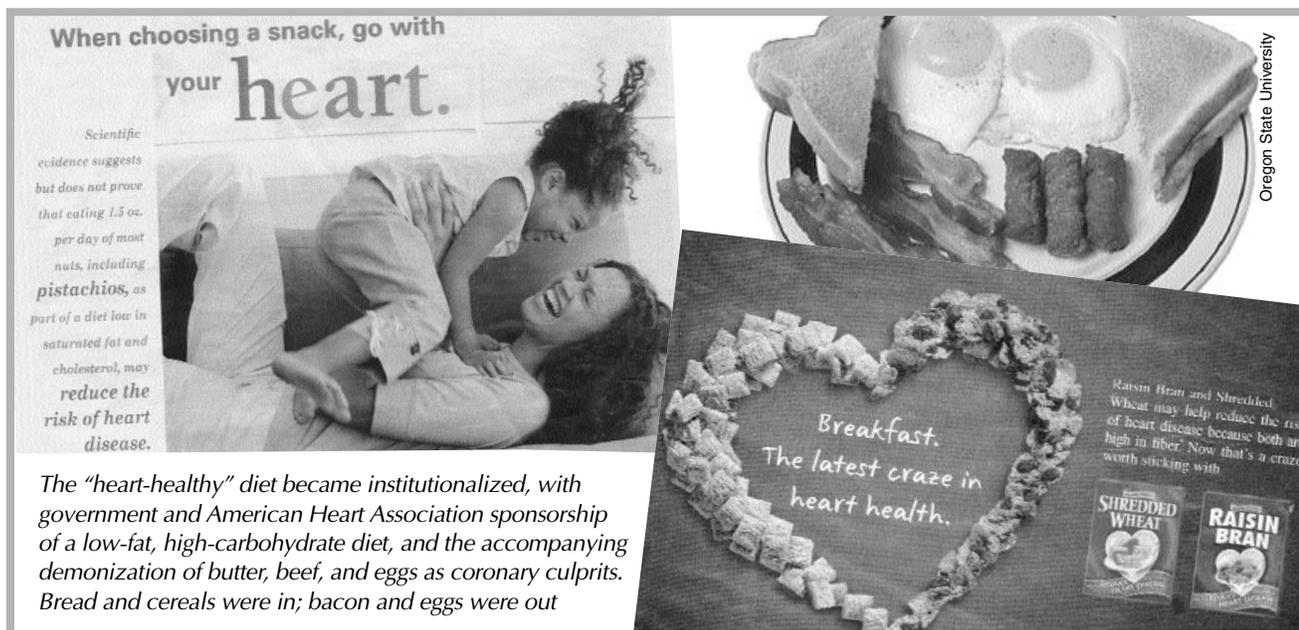
*Uffe Ravnskov, a Swedish physician, has challenged Keys's analysis, and calls the lipid-cholesterol hypothesis a fallacy. His research is summarized in The Cholesterol Myths: The Fallacy That Saturated Fat and Cholesterol Cause Heart Disease (published by New Trends Publications, Washington, D.C.)*

written and promulgated by NIH, a branch of the U.S. Government. But the facts are that the new guidelines were approved and issued by the National Cholesterol Education Program (NCEP), a nongovernmental organization operating under the aegis of, and with the support of, the NHLBI. A subgroup of the NCEP, called the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, actually wrote the new guidelines. It is noteworthy that the Expert Panel was composed primarily of experts from the drug industry.

Despite the fact that the process by which the guidelines were written may seem proper to most people, it was not. This process bypassed government codes aimed at ensuring that standards, guidance documents, and rules approved and promulgated by government agencies have been considered in open meetings and are free of bias. The approach used by NCEP suggests a strategy in which special interests used the stature and credibility of a government agency to promote faulty science that supports the sale of low-fat, low-cholesterol foods and certain prescription drugs. It is ethically wrong and

seemingly illegal for a private group, ostensibly sponsored by the federal government, to be given the responsibility for formulating and approving guidelines that will become standards of good practice for all of the physicians in our country.

Within days of the publication of the 2001 guidelines, pub-



Oregon State University

*The "heart-healthy" diet became institutionalized, with government and American Heart Association sponsorship of a low-fat, high-carbohydrate diet, and the accompanying demonization of butter, beef, and eggs as coronary culprits. Bread and cereals were in; bacon and eggs were out*

lic interest groups raised questions about the influence of drug manufacturers. It was estimated that the guidelines could put about 18 percent of the entire U.S. population on statin drugs, which would have the effect of tripling sales of these products to nearly \$30 billion per year. Aside from a potential windfall for drug companies, a grave concern is for the impact the guidelines will have on both the practice of medicine and the long-term health of Americans. Will physicians see the guidelines as the product of the latest government research? Will they prescribe more statin drugs and tell their patients to try harder to follow a low-saturated-fat, low-cholesterol diet? Will lawyers and judges use these guidelines as standards of good practice in legal controversies?

Some answers are now forthcoming. Since the promulgation of the guidelines, skyrocketing sales of statin drug constitute very good evidence that the nation's medical professionals are responding to them. This increase in statin drug sales indicates that doctors are promoting the heart-healthy diet, increasing blood cholesterol testing, and prescribing

statins. Thus, the folly of a half-century is continued and reinforced. But faulty science inevitably has its price. Heart disease will continue unabated, and better approaches to controlling coronary heart disease will very likely continue to be marginalized.

The "New Cholesterol Guidelines" ignore the adverse effects of excess dietary carbohydrates on heart health, despite the fact that, in reaching their conclusions, the authors acknowledged the fact that the 40-year use of the heart-healthy diet was accompanied by concurrent increases in the coronary heart disease that this particular diet was supposed to prevent. Then, in the face of these irrefutable facts, they recommended that Americans intensify their use of the heart-healthy diet. Such disregard for both facts and logic is not a rational outcome of scientific deliberation. When you see water running uphill, look for a pump!

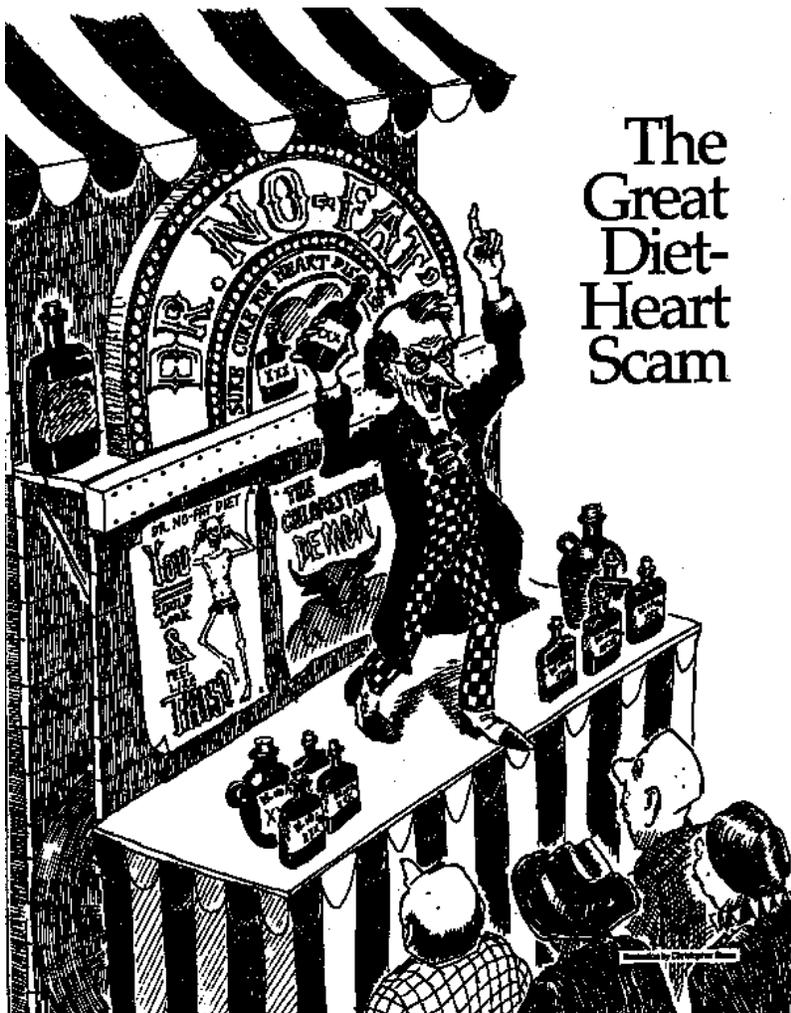
### What Do You Do Now?

As a beginning, accept the fact that your health is your personal responsibility. Do not assume, as many people do, that caring for your health is your doctor's task alone. Your doctor is concerned about your health, but you are only one of his many patients to whom he must devote his professional thoughts and concerns. He does not have time to be your alter ego, even if he could. Remember, no matter how much you depend on your doctor to provide you with good health, it is only you, not he, who will suffer the heart attack.

Make your doctor a partner in your health by becoming an informed health care consumer, and learn some basics of nutrition and its relationship to coronary heart disease. Reading and study will help you explain why an excess of glucose-releasing foods (sugar and starch) and an imbalance of essential fatty acids are two of the most important nutritional causes of modern nutritional diseases.

It is important for you to be informed, because your doctor is not an expert in nutrition and probably knows little more about it than an informed layman. His knowledge of nutrition most likely came from nutrition academia, which is the arbiter of nutrition knowledge and defender of the lipid-cholesterol hypothesis. Thus, your doctor may not be aware of the biochemistry presented in Figures 1, 2, and 3, and so, instead of recommending a low-carbohydrate diet, he will tell you to eliminate animal fats, and perhaps even red meat, as recommended by the "New Cholesterol Guidelines."

Along with your doctor, make your pharmacist a part of your health care team, especially if you are taking any prescription medications. As with nutrition, your doctor is not an expert in drug action. His knowledge of



*Conditioned by the mass media and a cult of statistics, the public's view of cholesterol and fat is not more scientific than that of the audience of yesterday for the snake-oil salesman.*

drug action comes from the pharmaceutical salespeople who explain how a drug works and what its side effects are. However, drug companies are not required to warn doctors about depletion of nutrients by a drug, or to include this hazard on drug labels. Thus, deficiency-caused health problems are often misdiagnosed because most doctors are not sufficiently aware of such problems.

On the other hand, pharmacists are knowledgeable not only in mechanisms of drug action, but also in mechanisms of action of natural and synthetic nondrug chemicals, including vitamins, minerals, herbs, miscellaneous supplements, and a wide variety of substances not intended for human consumption. Your pharmacist is an indispensable member of your health care team who, if you are taking statin drugs, can advise you about their depletion of CoQ.

Granted, reading and study will take effort, but it will be worth it because it is your health and that of your loved ones you are protecting. Be assured that nutrition is not an esoteric discipline; no one has a monopoly on knowledge of nutritional science, and no one has a monopoly on the ability to understand why the human body is not a simple furnace that will thrive on any fuel. It is a living organism dependent on countless complex biochemical reactions created over millions of years by natural evolution. It requires its own special fuel, a diet composed of a proper balance of specific proteins, fats, and carbohydrates, plus a host of other nutrients.

Finally, if you already have heart disease or any of the other modern nutritional diseases, take comfort in the fact that the human body is a very forgiving creature and will respond favorably to good nutrition, even after decades of abuse. And keep in mind as you study and learn—there are no drugs that can cure a nutritional disease!

#### Recommended Reading

Robert C. Atkins, *Dr. Atkins' New Diet Revolution* (New York, N.Y.: Avon Books, Inc., 1999).  
 This book contains a wealth of information on the causes and dangers of obesity. It describes a dietary program for safely returning to normal weight and health.

Robert C. Atkins, *Vita-Nutrient Solution* (New York, N.Y.: Fireside, Simon & Schuster, 1999).  
 Aptly subtitled "Nature's Answer to Drugs," this book presents detailed information on the use of nutritional supplements in the treatment of many chronic illnesses.

Mary D. and Michael R. Eades, *Protein Power* (New York: Bantam Books, 1999).  
 This book is based on the Eades's long and successful medical practice specializing in weight loss and control using a 40:30:30 (carbohydrate: protein: fat) diet plan

Mary G. Enig, *Know Your Fats* (Silver Spring, Md.: Bethesda Press, 2000).  
 An important source of accurate nutritional information on fats, oils, and cholesterol, this book contains difficult-to-find information for saturated, trans, and unsaturated fat composition of several hundred foods.

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