

## BOOK REVIEWS

**The Cholesterol Myths: Exposing the Fallacy that Saturated Fat and Cholesterol Cause Heart Disease** by Uffe Ravnskov, M.D., Ph.D. Published by the New Trends Publishing Co., Washington, D.C., 2000, xiv+297 pp. \$20.00 (p). ISBN 0-9670897-0-0.

With courage and care Dr. Ravnskov exposes the lack of experimental evidence for the diet-heart theory (DHT), which claims that eating less fat and cholesterol will prevent atherosclerosis. He shows how the abstract or conclusions of a number of papers are at odds with the actual data in the papers. He demonstrates how the use of one statistical method in preference to another can give a false impression that there is an effect of drug or diet in lowering total death rates, where there is, in fact, no significant effect.

“This book was written to give you and your doctor some facts about cholesterol and coronary heart disease (CHD). They are facts that even your doctor may not know because these facts have been misunderstood; or because many scientists, health authorities and representatives of the drug companies have suppressed them altogether” (p. 5).

Surprisingly, this author has included about 2 dozen cartoons to make this very wrenching expose more palatable. The chapters are called “Myths.” Here are the chapter titles, which certainly show the direction of this book:

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

In a striking graph from one of the papers of John Yudkin, M.D., Ravnskov shows that the number of deaths from CHD in England and Wales between 1910 and 1956 is closely correlated with the number of new radio and television sets purchased each year. This is a perfect example of a correlation without a cause. Another line on the same graph shows that the number of grams of animal fat consumed per day changed by only ~10% during this period. There is no correlation whatsoever between fat consumption and death rates from CHD, which increased 6-fold during this time period. He notes that in the US, coronary mortality increased about 10 times between 1930 and 1960, leveled off during the 1960s, and has since decreased slowly. During the decline of heart mortality the consumption of animal fat declined also, but during the 30

years of sharply rising coronary mortality, the consumption of animal fat also decreased.

In another example: “While the death rate from coronary disease increased in most countries after World War II, it decreased in Switzerland. If this decrease had been preceded by a decline in the intake of animal fat, Switzerland would have been a model for health care in other countries. But the diet-heart proponents never mention Switzerland because during the decline in heart mortality, the Swiss intake of animal fat increased by 20%” (p. 31).

In still another example: “The Masai [of Kenya] drink ‘only’ half a gallon of [whole] milk each day.... Their parties are sheer orgies of meat; on such occasions four to ten pounds of meat [eaten] per person is not unusual, according to Professor [George] Mann [of Vanderbilt University in Nashville, TN, USA]. If the diet-heart idea were correct, coronary heart disease would be epidemic in Kenya. But Professor Mann found that the Masai do not die from heart disease—although they might die from laughter if they heard about the campaign against foods containing cholesterol and saturated fat. But this was not the only surprise. The cholesterol of the Masai tribesmen was not sky-high as Mann had expected; it was the lowest ever measured in the world, about 50% lower than the value of [that of] most Americans” (pp. 32–33).

Major frauds are decried: “Most supporters of the diet-heart idea think that the increased risk of CHD is present at all cholesterol levels. Those who have a cholesterol level of 200 mg/dL, for example, are worse off than those with a cholesterol level of 150 mg/dL; and those who have a cholesterol level of 250 mg/dL are at even greater risk. The pharmaceutical companies love this concept for it implies that almost everyone should be treated, even those with normal cholesterol levels.... The truth, were it known, would send pharmaceutical stocks plunging. In most studies, the increased risk is present only above a level of cholesterol that includes just a small percentage of the total population. [These are the approximately 0.5% of people with a genetic defect called familial hypercholesteremia (FH).] And women can stop worrying immediately because high cholesterol is not a risk factor for the female sex.... In fact, it seems more dangerous for women to have low cholesterol than high. Dr. Bernard Forette and a team of French researchers found that old women with very high cholesterol live the longest... <1/5 times the death rate of... women who had very low cholesterol. In their report, the French doctors warned against cholesterol lowering in elderly women. But they could as well have warned against cholesterol lowering in any woman, or, to be more precise, in anyone at all” (p. 59).

Your reviewer checked one of the citations on MRFIT (Paul et al., 1982) to find that the summary noted honestly that the treatment group had less mortality from CHD and more total mortality than the controls did. That the former was not statistically significant was in the abstract; that the latter was not statistically significant was not in the abstract, but only in the body of the paper. Another problem with both this and some other studies is that the interventions included diet, anti-hypertensive drugs and smoking cessation all at once.

The authors thought that less smoking was beneficial and that anti-hypertensive drug therapy was harmful. But the diet for the treatment group called for lower saturated fat and cholesterol intake and higher polyunsaturated fat intake; the authors did not admit the possibility that this intervention could have been harmful. In an end note Ravnskov simplified a table in this paper and showed that the entire difference in death rates of certain sub-groups was due to quitting smoking, which cut the death rate in half.

Dr. Ravnskov went on to show that higher levels of high-density-lipoprotein (HDL, “good” cholesterol) are not protective against CHD and that lower levels of low-density-lipoprotein (LDL, “bad” cholesterol) are not beneficial, although the expected associations of each with CHD are present. Triglycerides were said to be even less correlated with CHD than cholesterol is; the assay for triglycerides is worthless unless the patient has been fasting 12 hours, and the assay is only accurate to  $\pm 50\%$ . Intimations in papers that there are “many” or “definitive” studies in reports and papers supporting the DHT were shown to be false by showing that citations often led to other reviews, each trusting the last, and ending in very few original studies, and that even those were often flawed statistically.

Dr. Ancel Keys was one of the main proponents of this myth that eating fat causes CHD. In a paper published in 1958, Keys showed a graph of the percent calories from fat in the food of various countries vs. the mean serum cholesterol levels. The data points fell on a smooth curve, showing an excellent correlation. Dr. Ravnskov added data points from a number of countries deliberately ignored by Dr. Keys. These fall nowhere near the curve. Furthermore, CHD death rates among subjects in Finland, Greece and Yugoslavia with similar serum cholesterol levels varied 5-fold depending on which area of the country the individuals lived in! Four studies in the US, one in the UK, one in Israel and one in Finland failed to show any correlation between diet and serum cholesterol levels.

“Numerous studies have shown that in people who eat a normal Western diet, the effect on blood cholesterol of eating 2 or 3 extra eggs per day over a long period of time can hardly be measured” (p. 108).

Ravnskov dealt with the smear campaign against eggs as follows: “To find out how egg consumption influenced my own blood cholesterol, I once used myself as a human guinea pig without asking the ethics committee at my university. Before and during the experiment I analyzed my [total serum] cholesterol. My usual egg consumption is one or two eggs per day, and my cholesterol value at the start of the experiment was 278 mg/dL, very close to a determination of [my] blood cholesterol made 10 years earlier.” On day 0, Dr. Ravnskov ate 1 egg; on day 1, 4 eggs; on day 2, 6 eggs; and on days 3–8, 8 eggs per day! “The data from my daring experiment showed that instead of going up, my cholesterol went down a little [to 246 mg/dL]” (p. 109).

Dr. Ravnskov explains that older people have higher concentrations of cholesterol in their blood than younger people. If the serum cholesterol is graphed against the degree of atherosclerosis with all age groups lumped together,

there seems to be a direct relationship. But if only people of about the same age and sex are considered, there is only a weak relationship with a correlation coefficient of 0.29. When the subjects with FH are left out, even the weak correlation vanishes.

Some early research was ignored: “The first study designed to demonstrate a possible correlation between blood cholesterol and degree of atherosclerosis was published by the pathologist Kurt Land, and the biochemist Warren Sperry of the Department of Forensic Medicine at New York University... [in] 1936. They studied large groups of individuals who had died violent deaths. To their surprise, they found absolutely no correlation between the amount of cholesterol in the blood and the degree of atherosclerosis.... Because Land and Sperry were cautious and methodical, their study should have nipped the DHT in the bud. Or, more accurately, if those who promoted the DHT later on had read Land and Sperry’s paper before beginning their research, they would probably have dropped the idea at once.... But the few who remember Land and Sperry misquote them and claim that they found a connection, or they ignore their results by arguing that cholesterol values in the dead are not identical with those in the living.... [To resolve this argument]... In the city of Agra in India, Dr. K. S. Mathur and his co-workers performed a similar study [in 1961]. Their first step was to measure blood cholesterol in 20 patients shortly before death and then a varying number of hours afterwards. They found that the cholesterol values were nearly the same if samples [were taken] before death and within 16 hours afterwards. Thus, blood samples taken very shortly after death are reliable—an important confirmation of the study done by Drs. Land and Sperry. Dr. Paterson’s group in Canada confirmed this.... Next Dr. Mathur and his colleagues studied 200 people who had died in an accident, without any preceding disease. Like Drs. Land and Sperry, and like Dr. Paterson, the Indian researchers could find no connection between cholesterol values and the degree of atherosclerosis. Those with low cholesterol had just as much atherosclerosis as those whose cholesterol was high” (p. 118–120).

A report from the Framingham Study found a weak correlation coefficient, 0.36. Dr. Ravnskov found what distinguished this report from all the others he studied: only 14% of the Framingham dead were chosen for autopsy, not close to 100%, as in the other studies. The risk of preferentially selecting subjects who probably had FH hypercholesteremia was said by Ravnskov to be great. To prove that high cholesterol is the villain—and not just an innocent bystander—demands that a change in the cholesterol concentration in each individual is followed by a change in degree of atherosclerosis in the same direction. Examination of all studies on this relationship showed no correlation.

Dr. Ravnskov reviewed the evidence presented earlier—that cholesterol levels in blood, or HDL or LDL levels, or the ratio of the latter are not correlated with either atherosclerosis or heart attack rates. It follows that forcible reductions of cholesterol levels by drugs (since diet alone does not change the levels much) would not be expected to change the rate of CHD by lowering cholesterol levels. However, two things are possible with drugs. First, some

unknown mechanism unrelated to cholesterol could lengthen lifespan. Second, some side effect unrelated to cholesterol could shorten lifespan. Furthermore, the pervasive misconceptions about cholesterol have made it nearly impossible to carry out a placebo-controlled trial of new drugs because it is mistakenly considered unethical not to treat people with high cholesterol levels!

“In the 1960s, Professor Jeremy Morris of London, England, led a team of physicians and scientists in an investigation to see whether the replacement of animal fat with soybean oil could have some preventive effect on CHD. This oil is rich in polyunsaturated fatty acids, those that are considered [erroneously to be] protective against atherosclerosis and CHD. Enrolled in the trial were about 400 middle-aged men who had previously been admitted to 4 London hospitals because of a heart attack; half of these received a diet containing large amounts of soybean oil. (This is one of the few trials sponsored solely by a government, and not by a drug company or any other vested interest.).... When the researchers analyzed the results 4 years later, they could find no beneficial effects from using soybean oil. Although, in this particular trial, blood cholesterol had decreased considerably in the treatment group, 15 had died of a heart attack. In the control group, 14 had died; and the number of non-fatal heart attacks was the same in both groups” (p. 144–145). Other trials gave the same result.

These trials on patients who already had symptoms of CHD are called “secondary prevention” trials. Now Ravnskov describes some of the “primary prevention” work, that is, trials with healthy or at least symptomless patients. Much larger numbers of subjects are needed in an attempt to obtain good statistical results, and compliance is always suspect because of the severe side effects of many of the treatments or drugs used in subjects who are basically healthy, and thus may not be compliant because they lack any fear of poor health. When you recall the conclusions in Myth 2, that high cholesterol does not cause CHD, you will not be surprised at the negative findings he has described.

In 1967 the Coronary Drug Project tested nicotinic acid, clofibrate, thyroid hormone, and estrogen to lower cholesterol levels in middle-aged men who had already had at least one heart attack. After 7 years the death rates were the same as those of the controls. Worse, all 4 drugs had severe side effects. The researchers fell victim to the “surrogate endpoint.” This is the use of an easily measured factor, such as total cholesterol level or blood pressure, as a surrogate or substitute for what is really important—increasing lifespan or the quality of life. In a later chapter Ravnskov calls this a “surrogate outcome.”

In 1970 the Upjohn Co. (Kalamazoo, MI, USA) sponsored a trial on 2000 men and women with high cholesterol involving its then-new drug colestipol. Two years later no effect was seen in the women. The number of heart attacks in the men in the treatment group was cut in half, a remarkable result never seen before or since. But Ravnskov found the snag: The selection of the patients to be in either the treatment or control groups was done by Upjohn’s scientists with the results of the participants’ blood assays in hand; it was any-

thing but random. Ravnskov noticed that there were too many control patients with FH. Your reviewer notes that, in the 1996 *Physicians Desk Reference* entry for this drug, there is not a shred of evidence for longer lifespan; moreover, there were no restrictions on prescribing this drug for women.

For the World Health Organization trial, researchers assayed blood cholesterol in 30,000 healthy, middle-aged men in Edinburgh, Prague and Budapest. The 10,000 men with the highest blood cholesterol levels were selected for the trial, half to receive clofibrate, half placebo. After 5 years there were more fatal heart attacks in the clofibrate group. There were 128 total deaths in the clofibrate group and 87 in the placebo group. "Yet clofibrate is still recommended in many countries as a useful drug" (p. 151)!

The National Heart, Lung and Blood Institute embarked on a new jumbo trial called The Lipid Research Clinics Coronary Primary Prevention Trial (LRC) to test the effectiveness of cholestyramine (Bristol-Myers Squibb). To find about 4,000 test subjects, the 0.8% of 500,000 middle-aged men with the highest cholesterol levels were selected. All were given a few weeks of dietary indoctrination to solve the supposed ethical dilemma of not otherwise treating the controls. Half received cholestyramine and half placebo for 7–8 years. Of those treated, 190 (10%) had nonfatal heart attacks, vs. 212 (11.1%) of the controls. For fatal heart attacks the figures were 1.7% and 2.3%, a difference of 0.6% absolute or 12 individuals. In the summary of the paper on this trial these unimpressive results were presented as a 19% lowering (relative risk) of nonfatal heart attacks and a 30% lowering of fatal heart attacks.

This is an example of scientific fraud among efforts to support the DHT. Ravnskov notes how the reporting of differences in fatality rates by percent reduction (say, a 50% reduction in relative risk) is actually misleading when the actual death rates are quite small in both the treatment and control groups of subjects in diet or drug studies. For example, a treatment that changes the absolute survival rate over a multi-year period from 99.0% to 99.5% represents a 50% reduction in relative risk, from 1% to 0.5% absolute. This is often described in papers as a 50% reduction in death rate. However, when the difference is barely significant statistically, as was often the case, Ravnskov points out that there is no real reason to recommend adoption of the treatment, especially if there are potentially serious side effects.

Ravnskov continues: "And this was not the only way in which the LRC figures were manipulated. In order to reach their 30% figure, the LRC directors included the uncertain cases, those who may or may not have died from a heart attack, and to reach their 19% figure, they excluded the uncertain cases. If it had been the other way around the results would have been 24% rather than 30, and 15% rather than 19. In other words, they selected data that gave them the results they were seeking." Even worse, the directors abandoned the 99% confidence level with a 2-tailed t-test and settled for a 95% confidence level with a 1-tailed t-test. [In an end note Ravnskov points out that scientists have agreed that a 1-tailed t-test should be used only when it is certain that the result

will go in just one direction. It is not supposed to be used when the drug (or other intervention) may do harm rather than good.] Very revealing is the absence of the number of deaths from all causes. More men in the treatment groups died by violence or suicide (11 vs. 4). In the misleading manner used by the LRC to present results, they could have said that violent death was 175% more likely in the treatment group. In order to achieve essentially nothing, the treatment group suffered gas, heartburn, belching, bloating, abdominal pain, nausea and vomiting; contrarily, the study's report assured readers that the side effects were not serious. Some promoters then claimed that "now that it had been proven that it is worthwhile to lower cholesterol no more trials were necessary!"

Ravnskov goes on to show that trials with a seemingly positive result, even for a surrogate endpoint, are cited much more frequently than trials with a negative result. This gives a positive feedback effect, reinforcing the dogma that reducing cholesterol level is beneficial, but this sort of misdirected effort actually does not produce better health.

A study showed that patients treated with lovastatin and colestipol had their coronary arteries narrowed as shown by X-rays. The title of the paper on this study indicated the opposite: "Regression of coronary artery disease as a result of lipid lowering therapy..." Ravnskov then presents the results of a meta-analysis of 26 cholesterol-lowering trials that met his standards. Result—no benefit.

Ravnskov presents the results of a number of trials of statin drugs in which total death rates are slightly lower than those of the controls. But in an early trial (EXCEL) of lovastatin (Merck's Mevacor) on 8,000 healthy subjects with cholesterol levels between 240–300 mg/dL, the absolute death rate from all causes after just 1 year was 0.5% vs. 0.2% in the placebo group. In another trial with lovastatin on healthy subjects, 5,000 men and 1,000 women, 2.4 % in the treatment group died after 5.2 years vs. 2.3% of the controls. Regarding studies carried out on lovastatin, studies lasting 10 years, Ravnskov found no reports on total death rates. Ravnskov queried Merck & Co. directly and was told that the trial was not designed to measure the total clinical outcome!

Deaths from heart attacks were significantly lower in some trials of other statin drugs, but total deaths were 3% absolute lower at best. In the CARE trial, Ravnskov showed that a 12% reduction in heart attacks (- 1% absolute) was overbalanced by a 1,500% increase in cases of breast cancer (+4% absolute). Total deaths were not given. Once again this shows that women should not be treated with statin drugs (or at all), and the benefit for men is quite limited at best with simvastatin and pravastatin.

The incidence of breast cancer was said to be a fluke, and was not observed in the LIPID trial lasting 6 years, in which overall mortality was said to be reduced by 22%, but this was relative risk; an overall drop in mortality of 3% absolute was achieved in subjects with a broad range of initial cholesterol levels (Tonkin et al., 1998).

In middle-aged men with hypercholesteremia treated with pravastatin for 5 years, death from all causes was reduced by 22%, but this was relative risk; an

overall drop in mortality of 1% absolute was achieved (Shepherd et al., 1995).

Ravnskov tries to explain what the polyunsaturated oils are chemically. His effort is one of the few weak points in this book. The degree of saturation actually refers to whether hydrogen can be added to the oil. If so, some of the carbon-carbon bonds in the fatty acid portion of the oil molecule must have been double bonds, which may take up 2 hydrogen atoms each. Olive and canola oils are the best examples of monounsaturated oils (a sole double bond in each fatty acid portion), and safflower, cottonseed and soybean are examples of polyunsaturates (2 or more double bonds in each fatty acid portion). If hydrogen cannot be added in the presence of a catalyst, the oil (or more likely the fat) is said to be saturated, meaning that it cannot take up any more hydrogen. Palm and coconut oils are the best examples. Tallow, lard and chicken fat have some saturated and some monounsaturated fatty acids in their molecules; they are not made entirely of saturated fatty acids by any means. The risk of eating *trans* fats is presented at some length.

Ravnskov gives examples of reports of interventions with little or no statistical significance being denied time for presentation at meetings and explains that offers to write minority dissenting reports on certain trials were being denied on the grounds that the conference was supposed to produce a consensus. Statements of DHT proponents and their recommendations are quoted followed by a Ravnskov's refutation of the claimed evidence. He reiterates that even drastic lowering of cholesterol levels with drugs (diet being ineffective) is of no benefit to women and of marginal benefit to men. Ravnskov presents arguments against trying to lower cholesterol levels in children.

If Ravnskov were a lone voice among the Philistines, his credibility would be lowered. In Myth 9 he gives the names of several of the scientists who support his position. This includes Mary S. Enig, President of the Maryland Nutritionists Association, whose research concerned the hazards of trans fats and who has written *Know Your Fats*, a book on the composition and effects of fats in the diet. Michael Gurr, Professor of Biochemistry, School of Biological and Molecular Sciences, Oxford University, pointed out the insufficient correspondence in vascular pathology between animal models and man, the selection bias in epidemiological evidence, the lack of correlation between CHD and fat consumption, and the lack of improvement in coronary mortality after dietary and drug intervention. George Mann, Professor of Medicine and Biochemistry at Vanderbilt University (Tennessee), realized from his studies of the Masai in Kenya that animal fat could not possibly be the cause of high cholesterol and CHD, and he has been open and fearless in his criticism of the LRC directors and has called the DHT "the greatest scientific deception of this century, perhaps of any century." Michael F. Oliver, former Professor and Director of the Wynn Institute for Metabolic Research (London, UK), has warned against campaigns for cholesterol lowering in the general population; criticized those who think that the increased mortality from non-medical causes in trials, such as suicide, is due to chance; and is uneasy about the link be-

tween low cholesterol and cancer. Edward R. Pinckney, editor of a number of medical journals, published a book in 1973 called *The Cholesterol Controversy*, which summarized all the inconsistencies in the cholesterol literature; he describes the dangers of lowering one's cholesterol and devotes an entire chapter to the political drama preceding an early anti-cholesterol campaign. Raymond Reiser is a former Professor of Biochemistry at Texas A&M University. He decried the practice of referring to other reviews, each taking the last on faith, which has led to the acceptance of a phenomenon (diet-heart) that may not exist. He reviewed work on fatty acids in the diet, found flaws in most of the studies, and concluded that the type of fat in the diet does not make much difference. He analyzed the references used by the American Heart Association in its rationale for dietary recommendations and found no supportive studies but found instead some that contradicted the recommendations. Ray Rosenman is the retired Director of Cardiovascular Research in the Health Sciences Program at SRI International in Menlo Park (California) and Associate Chief of Medicine, Mount Zion Hospital and Medical Center in San Francisco, CA. In a recent review he wrote that neither diet nor the identity of serum lipids (fats or oils) can explain wide national or regional differences in rates of CHD, or the 20th century variations in rates of CHD. He also pointed out that the CHD preventive effects of diets and drugs have been exaggerated by a tendency in trial reports, reviews, and other papers to cite and inflate supportive results while suppressing discordant data. The late Russell Smith was an American experimental psychologist with a strong background in physiology, mathematics and engineering. In his 1989–91 review of the diet-heart theory he wrote: "...studies are often poorly designed and data are often inappropriately analyzed and interpreted.... Much of the literature, therefore, is nothing less than an affront to the discipline of science...." He considered much of the work of the National Heart, Lung and Blood Institute and of the American Heart Association to be "incompetent" and "sloppy," and that their political and financial power is enormous and without equal, producing a juggernaut willing and able to suppress evidence and logic. "Equally culpable are the editors of the many journals who publish articles without regard to their quality or scientific import. It is depressing to know that billions of dollars and a highly sophisticated medical research system are being wasted chasing windmills." In an extensive 2-part review, William E. Stehbens, Department of Pathology and Molecular Medicine, Wellington School of Medicine, New Zealand, demolished the diet-heart theory and gave specific examples of misinterpretation of data, of the misleading use of relative risk rather than absolute death rates, and of the medical folly of trying to lower cholesterol levels in almost anyone (Stehbens, 2001). Now retired, Lars Werkö, previously Professor of Medicine at Sahlgren's Hospital, Gothenburg, Sweden, Scientific Director at the Astra Co. (now Astra-Zeneca), and head of the Swedish Council on Technology Assessment in Health Care, criticized the design of the Framingham Study and pointed out inaccuracies and sloppy data gathering in the MRFIT trial.

As it happens, a number of other physicians and scientists in addition to the ones in Myth 9 agree with Ravnskov's positions. Linus Pauling, in his 1986 book *How to Live Longer and Feel Better* noted that the Framingham Study showed no correlation between CHD and fat intake or with cholesterol intake.

Thomas J. Moore, a medical reporter based in Washington, D.C., wrote an article in the September 1989 issue of *The Atlantic Monthly* actually called "The Cholesterol Myth," in which he examined the literature, much as Ravnskov did. Moore's conclusions: "Lowering your cholesterol is next to impossible with diet, and often dangerous with drugs—and it won't make you live any longer." This review was also used in Moore's 1989 book *Heart Failure: A Critical Inquiry into American Medicine and the Revolution in Heart Care*.

William Campbell Douglass, Jr., M.D., in 1993 wrote a brochure called *Eat Your Cholesterol: How to Live Off the Fat of the Land and Feel Great!* (Atlanta, GA: Second Opinion Publishing). Many of the dietary studies and trials are the same ones evaluated by Ravnskov, but they are treated in a very popular tone.

John B. Allred came to nearly the same conclusions as did Dr. Ravnskov (Allred, 1993). An even more extreme view aired in the United Kingdom is that serum cholesterol level is a relatively poor predictor of CHD, that the many misclassifications may have damaging psychological effects, and that in most cases, even testing cholesterol level, as well as treating raised concentrations, is a waste of National Health Service resources (Sheldon, 1998).

Kilmer S. McCully, Ph.D., M.D., in technical papers (McCully, 2001) and in his book (McCully, 2000) wrote, "But no study anywhere has ever proven that lowering the amount of cholesterol in the diet reduces the risk of heart disease. And lowering cholesterol through drugs won't prevent arteries from hardening if homocysteine is high." McCully is the discoverer of the fact that the undesirable amino acid called homocysteine is an actual cause of atherosclerosis and CHD.

Charles T. McGee, M.D., wrote, "The cholesterol theory is a fraud on the American public... the theory will be exposed as a scam," in his book *Heart Frauds: Uncovering the Biggest Health Scam in History* (2001, p. 76).

The authors of 2 recent reports on the use of electron beam tomography (EBT) to detect calcified plaques in coronary arteries were very circumspect in interpreting their findings, especially in their abstracts, perhaps not fully aware of how completely their findings demolished the DHT. While EBT was very predictive of hard coronary events in both studies, the difference in hypercholesteremia between the patients with events and those without was not significant. There was absolutely no significance for HDL cholesterol (HDL-C) levels as predictive, and the spread in the values for LDL cholesterol, total cholesterol (TC), TC/HDL-C and triglycerides shows none of these to be usefully predictive despite the low p values (Hecht et al., 2001; Raggi et al., 2001).

Based on Ravnskov's meticulous analyses as well as the considerable support for his stance shown by others who have also studied the cholesterol data,

this book is recommended without reservation. Physicians and other health professionals as well as anyone threatened with cholesterol-lowering treatments would be enlightened and better able to resist worthless treatments. Health insurers might reconsider compensation for frequent (or any) clinical assays for cholesterol or triglycerides, let alone expensive treatments to lower cholesterol levels that reduce quality of life without prolonging it significantly.

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**The Placebo Response: How You Can Release the Body’s Inner Pharmacy for Better Health** by Howard Brody with Daralyn Brody. New York: Cliff Street Books (HarperCollins), 2000. 312 pp. \$25, cloth; ISBN 0-06-019493-6; HarperTrade, 2001, \$14, paper; ISBN 006093297X.

“The placebo response occurs when we receive certain types of messages or signals from the environment around us. These messages work in some fashion, at some level, to *alter the meaning* of our state of health or illness” (p. xvi; italics in original).