

An interview with William B. Parsons Jr., MD about his new book, Cholesterol Control Without Diet! The Niacin Solution

Please read our [disclaimer](#) if you haven't already.

For comment on **Bill Clinton's bypass surgery** and about the **danger of deaths from new (July 2004) cholesterol guidelines**, scroll to bottom of list of questions.



You say, "You don't have to diet to control cholesterol." What is the background for this statement?

Niacin, the first and still most successful cholesterol-control drug, controls blood cholesterol levels in the presence of an unrestricted diet. This has been known since the drug was first introduced in 1955. We used niacin in the large doses recommended by the Canadian originators without changing the diet in any way. The Canadians had done the same thing. Cholesterol levels dropped impressively. In the Rochester study, we also found that niacin reduced the "bad" cholesterol fraction and raised the "good" cholesterol fraction. This has been confirmed over the years. There still is not another drug that produces both these favorable changes.



Who should have his/her cholesterol level measured?

Every adult, really, and children in families who have had a heart attack, stroke, or other artery-narrowing event at a relatively early age. (One guideline says before 50 in a male relative or before 60 in a female relative.) Relatives of persons with known cholesterol problems should also be tested. But it is important that they do not just have the total cholesterol measured; the LDLC and HDLC fractions must be determined as well. If both bad and good cholesterol fractions are in the proper ranges, total cholesterol is really irrelevant. My book points out examples in which this is true.



Why is it important to know one's cholesterol level and treat it if abnormal?

In many cases the first symptom of coronary artery disease is **sudden death**. In these coronary victims, there are no preliminary warning symptoms, so we have no further opportunity to help these unfortunate persons. In an effort to see what can be done to prevent coronary deaths, research over the past several decades has determined what factors lead to heart attacks and what can be done to change them.



What factors have been found to lead to heart attacks?

Among the so-called coronary risk factors (which also apply to strokes) are age, male sex (or postmenopausal state in women), and strong family history of premature heart disease. These cannot be changed. On the other hand, factors which can be measured and corrected or treated are: smoking, high blood pressure, high blood cholesterol (hypercholesteremia), diabetes, physical inactivity, and obesity. The first three (smoking, high blood pressure, and hypercholesteremia) are the strongest predictors of vascular disease and, therefore, the most important to treat.



Does quitting smoking help to prevent heart attacks and strokes?

Yes. Smoking one pack of cigarettes per day approximately doubles the risk of heart attack and stroke. Quitting smoking reduces the risk within a short time to that of a lifelong nonsmoker! It cuts the risk in half by lowering that doubled risk to a normal one.



Does control of high blood pressure help to prevent heart attacks and strokes?

Yes. Many studies over the years show that proper blood pressure control reduces the risk of stroke significantly. A study reported in 1991 (Systolic Hypertension in the Elderly Program, or SHEP) showed that proper blood pressure control with inexpensive drugs not only lowered stroke risk but also resulted in 26% fewer heart attacks in older persons when the only abnormal part of the blood pressure was the upper (systolic) number.



Does reducing cholesterol levels help prevent heart attacks and strokes?

Yes. Niacin was the first drug for cholesterol control to offer such protection. This has been known since the middle 1970's. In recent years (1990's), studies have shown that a few drugs in another category for cholesterol control can also reduce the risk of heart attack and death. Regarding dietary treatment, it is difficult to give a short answer to this question, but the best short answer would be: Diet is not effective in preventing heart attacks and deaths.



Why is that?

Many answers are in an important book titled *Coronary Heart Disease: The Dietary Sense and Nonsense*. Published in 1993, it contains information presented at a meeting in Washington, DC, at which I had the privilege of being one of the eight

speakers. The scientists who preceded me on the program reviewed all the important studies over the years which had addressed the issues of diet, heart attacks, and deaths. They came to the conclusion that diet is not an effective method for controlling cholesterol or preventing heart attacks, strokes, and deaths.

My talk, the final one of the day, was titled Clinical Alternatives. In other words, my assignment was to answer the question, "If not diet, then what?" The talk covered a lot of material but spent more time on niacin than any other form of treatment. I pointed out that niacin is the only currently available drug with its long list of favorable characteristics. It is also the only inexpensive drug for controlling cholesterol.



What are these favorable actions which other drugs do not share with niacin?

If you were to make a "wish list" of the actions desired in a cholesterol-control drug, niacin has all of these actions. It is truly a "designer drug." No other drug has even the first two actions on the list:

- Reduces LDL ("bad") cholesterol and, therefore, total cholesterol
- Increases HDL ("good") cholesterol
- Reduces triglycerides
- Reduces Lp(a), an important risk factor
- Raises the ratio of HDL₂ to HDL₃, a protective effect
- Changes small, dense LDL particles (Pattern B) to larger particles (Pattern A), another favorable change



Can you simplify the language of LDL and HDL for us?

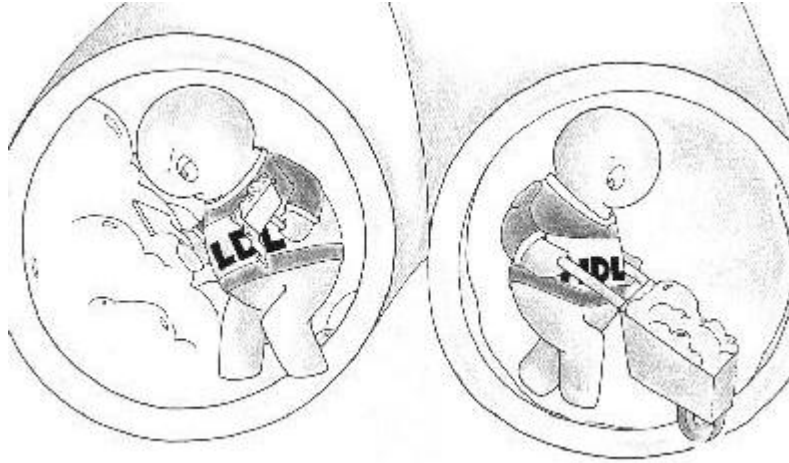
The cholesterol fractions and triglycerides collectively can be called "lipids," which just means "fat-like substances." They circulate in the blood serum (almost the same as plasma, the liquid part of the blood, as opposed to the blood cells), so we speak of serum cholesterol rather than blood cholesterol. These fat-like substances are attached to protein molecules, which is why we call them lipoproteins. Various lipoprotein types can be separated by density, or relative weight. The most important cholesterol fractions are low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol. We abbreviate these as LDLC and HDLC. There is another class called "very-low density lipoprotein cholesterol" (VLDLC), from which the body makes LDLC, but we seldom talk about it.



What makes some cholesterol "bad" and other cholesterol "good?"

LDLC gets into the linings of arteries and makes up a considerable part of the

deposits, or plaques, which narrow the arteries, eventually leading to blockage, which can result in a heart attack, stroke, or damage to another part supplied by the arteries. HDLC is not a part of the plaques but, in fact, removes material from plaques, leading to reduction in the narrowing and eventual blockage. This cartoon shows how these cholesterol fractions either deposit or remove cholesterol from the artery walls:



LDL ("bad") cholesterol is like a plasterer, heaping plaques onto the inner lining of arteries. HDL ("good") cholesterol works in the opposite direction, "unloading" cholesterol from arterial plaques.



Can you give us an easy way to remember which is which, the "bad" and "good" cholesterol fractions?

The book points out that H stands for "healthy," while L stands for "lousy." This memory device is not elegant, but it works. Let's always remember that niacin reduces bad cholesterol and increases good cholesterol. No other drug shares these characteristics.



Do we actually know that these changes reduce heart attacks and strokes?

Definitely! The Coronary Drug Project (CDP) was the first-ever nationwide cooperative study of drugs and heart disease. It was conducted between 1966 and 1974 in 53 centers from Massachusetts to Hawaii. The participants, 7,700 men between 30 and 65 who had already had one or more heart attacks, received either placebo capsules or one of four drugs capable of altering cholesterol and/or triglycerides. Two of the four drugs were dropped during the study because they caused adverse effects, including more heart attacks, than the placebo. Of the two drugs which finished the study (at least five years in every participant), the other drug did not improve the heart attack and stroke rate; only niacin had a favorable effect on all the endpoints.



How much did niacin reduce heart attacks, strokes, and other cardiovascular events?

Niacin reduced both heart attacks and heart attacks by about 25%. It reduced cardiovascular surgery by a whopping 46%! And it also reduced cardiovascular hospitalization and all hospitalization.



Did niacin also reduce the death rate from all causes?

Yes. This required a survey nine years after the end of the study, in which the statisticians contacted all 53 centers with a single question: who is still alive and who has died? When they sorted out these results according to drug groups, all of the other drugs had the same death rate as the placebo. Niacin had reduced the death rate by 11%, even in these high-risk patients who had already had at least one heart attack when they enrolled, an average of 15 years earlier. (The men were now between ages 45 and 80!) The statisticians calculated that niacin had added an average of 1.63 years to each man's life, which means that for some the added survival was much more than this average.



Your book says that in clinical practice a doctor should achieve even better results than in the CDP. Why is this so?

The CDP used a fixed dose of niacin, which was the usual starting dose, recommended by the Canadians and used in early studies at the Mayo Clinic and in Madison. In practice, we follow the cholesterol levels (which were kept from the doctors handling the CDP participants) and adjust the dose accordingly. Our earlier work had shown that at least 50% of patients required a larger dose as the CDP dose (sometimes twice as much) for the best cholesterol control. If the CDP dose had been even 50% larger, the results would have been even better. In a way, the cards were stacked against niacin in the CDP, but it had an outstanding record in spite of this.



Why has this good news not been publicized more widely over the years?

There are two important reasons. First, niacin has never had any "commercial push" behind it. Ever since I first worked with it, niacin has been a generic drug. No company can patent it and make large profits from its exclusive sale. This keeps niacin inexpensive. No sales representative comes to physicians' offices to tell of niacin's distinctive advantages, its long safety record, or its low cost. Other companies spend millions of dollars on advertising campaigns, aiming for larger shares of the multi-billion dollar market, even though their products have less impressive records and high price tags.

Second, for decades the cholesterol research establishment in the U.S. was dominated by workers mainly interested in dietary approaches to treating hypercholesteremia. All this began before niacin, when there was no effective

medication. Details of this domination are spelled out in the previously mentioned book, *Coronary Heart Disease: The Dietary Sense and Nonsense* and in my new book, [Cholesterol Control Without Diet! The Niacin Solution.](#)



How have the diet advocates managed to dominate medical opinion?

Until recently, diet advocates had always held positions of authority in opinion-making bodies such as American Heart Association and National Institutes of Health. Their bias in favor of diet thoroughly brainwashed the media, which in turn brainwashed the public, including the medical profession. Furthermore, the food industry has a huge vested interest in foods advertised as beneficial for cholesterol problems. I know researchers who believe that there has been a conspiracy between some dietary advocates and the food industry. I don't think they ever sat down around a table and conspired, but over the years they have trumpeted the party line about diet so loudly that the media and the public didn't realize there was any other way.

When most people hear "cholesterol," they automatically think "diet." My book points out why this is far from the truth. Diet is such a weak and ineffective for cholesterol management that it should be forgotten, except as a weight control measure. The only way to reduce excessive weight (which is an epidemic problem in the U.S.) is to control calories on an ongoing basis.



What do diet advocates say about cholesterol-lowering drugs?

The National Cholesterol Education Program (NCEP) of National Institutes of Health (NCEP) advises a trial of diet before adding a drug. But they are getting more realistic. Their 1988 guidelines specified a six-month trial of diet, and if not successful in the first three months, making the diet stricter. If any doctors really followed this plan, it must have made a lot of patients leave follow-up and never obtain effective treatment with a drug, because no one likes diet and no one likes an unsuccessful result. Two months is long enough to see whether diet will help. In their 1993 guidelines, NCEP changed the dietary recommendation to an unspecified (shorter) period, then using a drug when the diet fails to achieve the goals of treatment.



Your book states that many diet advocates complain that "all drugs have side effects and toxic effects," which implies that diet must be safe. What do you say about that?

A doctor must always know the expected side effects and any potential toxic effects of any drug he prescribes. He also needs to differentiate between nuisance effects and serious effects. The several cholesterol-control drugs on the market can be safely managed by physicians who know the necessary precautions and carefully supervise their use.

What bothers me is the diet advocates' implication that diet has no side effects or

toxic effects. It has both. The "toxic effects" are the heart attacks and strokes that occur when diet proves to be ineffective in cholesterol control. "Side effects" of diet include sentencing people to low-fat, high-fiber programs they neither want nor need. But the worst and most widespread "side effects" are the worry, concern, and guilt which people have been taught to feel when thinking about food, buying food, cooking and eating food. None of this is necessary! When you consider how many millions of people have been subjected to these "side effects," it would be no exaggeration to call the diet advocates' position a criminal conspiracy, as some do.



Has there been any landmark study of diet and heart disease, comparable to the CDP for drugs?

In the early 1960's, before the CDP, the National Institutes of Health (NIH) decided that there was plenty of evidence that it was better to have a lower cholesterol than a higher one. Proposing to find out whether treatment of high cholesterol levels really made a difference, they formed committees to determine the feasibility of a diet-heart study and a drug-heart study. The Drug-Heart committee, of which I was a member, decided that there were several drugs ready for such a trial and proceeded with their trial, the CDP. The Diet-Heart committee decided on a two-year feasibility trial at six centers.



What did the diet feasibility study show?

The two-year Diet-Heart Feasibility Trial really showed that most people would not follow any diet with significant fat and cholesterol restriction very well, even when special foods (not generally available then or now) were provided, along with repeated dietary counseling and what some one has termed "an enthusiastic team of exhorters." Furthermore, it didn't make much difference in cholesterol levels if they did. The study was too short to tell anything about rates of heart attacks, strokes, or deaths. They simply tried about six different diets to see whether people would follow them. Despite these negative findings, the diet advocates declared the study a success and advised a massive nationwide study. Fortunately, NIH did not accept this suggestion, and did not fund a larger study.



Getting back to niacin, what is your most important advice to patients?

The most important message of the book, repeated time after time, is this: *Niacin is not a do-it-yourself drug. It requires knowledgeable medical supervision.* In short, you need a doctor who is good at using niacin.



How can the patient know his doctor is good at using niacin?

The definition of a doctor good at niacin: one who has read my book. I advise the patient-reader to buy another copy of the book and present it to his/her doctor. The cost is not great; it is less than one saves in one month by using niacin rather than one of the best-selling but expensive drugs, the statins.



Does niacin have important side effects which limit its use?

Yes and no. It does have side effects, as all drugs do, but in most instances they do not limit its use if the doctor is good at niacin. Everyone who takes plain niacin experiences a flush, consisting of a warm feeling with redness of the face and upper body, sometimes accompanied by itching or a slight rash. At first this occurs with each dose lasting 20 to 30 minutes each time, but flushing usually decreases over the first several days of treatment. It disappears, for all practical purposes, in an average of three to four days.



Can the flush be lessened or eliminated in any way?

Yes, by taking a single aspirin tablet each morning, or by using a time-release (slowly absorbed) niacin preparation, or both. Plain niacin should always be taken with meals. It is also a good idea to avoid hot beverages at first, until the flush is no longer a problem.



Are there any other problems with niacin treatment?

There can be. But before going into specifics, let me remind everyone: although niacin is available over-the-counter as a "nutritional supplement," in doses used for cholesterol control, it is a drug which should not be taken without supervision by a knowledgeable physician. It is particularly important not to change from plain niacin to a time-release preparation in the same dosage. This is approximately the equivalent of doubling the dose, which can cause serious problems.



If not properly supervised, what sort of harm can niacin cause?

The most serious trouble would be liver damage, which can be detected by tests; then the drug can be stopped or the dose changed by a knowledgeable doctor. The physician must distinguish important liver trouble from a very common occurrence, slight alteration in one or more of the enzyme tests of liver function. My experience in the 1960's, along with that of other groups, including the Mayo Clinic, showed that slight increases above normal in one or more liver enzyme tests were to be expected as a normal part of niacin treatment in some patients and should not be the reason for

becoming concerned or stopping the drug.



Are there other side effects?

Yes, but not as serious as the liver effects, and most can be easily managed. Nausea may occur, but the doctor good at niacin handles this by changing the dose or the type of niacin preparation. The book discusses this and other side effects, which are less frequent and usually less significant.



With so many advantages and so few problems, why aren't more doctors prescribing niacin?

Again, the lack of "commercial push" and the extravagant promotional campaigns by manufacturers of other cholesterol-control drugs which have dominated the market in recent years. The competing companies brush off niacin as "hard to take because of side effects" and offer their expensive products, which lack niacin's distinctive advantages, at costs of \$50 to \$220 per month. Compare this with niacin's average cost of \$8 to \$10 per month. In a situation which usually requires continuing treatment for a lifetime, such a difference quickly multiplies into great financial savings for the patient whose doctor is good at using niacin.



Is niacin's status as a leading cholesterol-control drug recognized in any policy-making statement for physicians?

Yes. I don't mean to depict niacin as a total outcast. In its first detailed statement on diagnosis and treatment of cholesterol disorders, the NCEP (National Cholesterol Education Program) designated niacin and bile-sequestrant resins as the "first-line drugs" to use if dietary management proves inadequate. They selected these drugs because of their long safety records and proven ability to reduce not only cholesterol levels but also heart attacks. The 1992 NCEP guidelines reiterated the same choices and added one class to its list of "major drugs."



Can you briefly summarize the NCEP guidelines? What are the goals of treatment?

In 1993 they advised reducing LDLC to less than 100 in persons who have already had a heart attack, stroke, coronary bypass surgery, angioplasty (stretching a narrowed spot in an artery), or other arterial procedure, including surgery on neck or leg arteries. Levels this low have been shown not only to retard new cholesterol deposits but also to decrease existing plaques. For persons with no artery event but with two or more risk factors for coronary disease, they advised LDLC levels below 130. For those with no event and fewer than two risk factors, they accept LDLC below 160; I like to see it below 130 in everyone.

NCEP also pointed out that HDLC levels below 35 increase the risk of heart attack and other artery disorders and should be raised to higher levels. They did not give a specific target; I customarily aim for 45 or higher. As we said before, niacin is the only drug (or measure of any type, including diet and exercise) that both raises the HDLC and lowers LDLC.

With the advent of the statin drugs, the 2001 NCEP guidelines changed dramatically. The guidelines were flawed in several ways. They continued to emphasize diet, which doesn't work. If diet reduces LDLC at all, the reduction is negligible, about 5%. And diet also reduces HDLC just as much, a bad thing!

Worst of all, the 2001 guidelines strongly advised use of statins, almost to the exclusion of other drugs. When they do mention niacin, they call it "nicotinic acid," an old term that sounds bad and that readers might not recognize as a synonym for niacin. Each time they mention niacin, they couple it with fibrates and label them "triglyceride drugs." They do not mention that niacin reduces LDLC, raises HDLC, lowers triglycerides, reduces Lp(a), reduces VLDLC (very low-density lipoprotein cholesterol--which turns into LDLC and which they recommend as a target to be reduced), and favorably alters subfractions of both LDLC and HDLC. No other drug approaches this performance list. The panel also failed to mention that the statin drugs do only one of these things well (reduce LDLC).

Early in 2000 the NIH coordinator for the large NCEP panel (about two dozen members) told me that the panel was beginning to work on new guidelines. I sent copies of the first edition of *Cholesterol Control Without Diet* to each member. The 2002 guidelines showed that, as a group, they had not read or did not understand the distinctive advantages of niacin. Another factor, of course, might have been the strong activity of statin manufacturers in paying cholesterol experts. In a footnote to the 2002 guidelines, six members of the panel acknowledged that they had received moneys from statin companies. At least two others failed to acknowledge such support, although my files contain published papers in which they acknowledged financial support from drug companies that market statins. The current, second edition of the book discusses this matter in more detail.

In July, 2004 a smaller panel (nine, including the coordinator) issued an even more disgraceful set of guidelines. An Associated Press release reported that they had reviewed five recent studies (all statin-sponsored) and advised even lower LDLC levels as a goal of treatment. In 2001 NCEP had advised keeping LDLC below 130 in persons with no previous coronary or other event but below 100 for anyone with such a previous event. I was comfortable with those figures. In 2004 the small panel, through a convoluted reasoning process, used five recent studies to recommend LDLC below 70 for anyone with "high risk of coronary disease." More comments on these unwise and dangerous guidelines appear later in this report (after the Bill Clinton paragraph). Two days after the original AP story, another AP story revealed that eight of the nine panelists (all except the coordinator) had been paid by statin manufacturers.



What is Lp(a)? How is it treated?

Lp(a) -- pronounced "LP little a" -- is an abbreviation for "lipoprotein (a)," a part of the LDL family which has been found to be especially related to heart attack and stroke when the body produces too much of it. The only measure which reduces it effectively (if you exclude use of estrogenic hormones in postmenopausal women) is niacin. It is not necessary to do the expensive measurement of Lp(a) in everyone if the doctor uses niacin as his drug of choice for most lipid disorders. He just knows that this level, if too high, is being reduced by niacin as it corrects other problems with cholesterol and triglycerides.



Do you ever use diet as a cholesterol-control measure?

That's up to the patient. If he/she needs to lose weight, the only way to do so is by limiting calories. One part of reducing calories is to reduce intake of fats, higher in calories than either carbohydrate or protein. I do not restrict eggs because studies have shown they have no effect on levels of cholesterol or lipoproteins, and they are a good source of protein.

I emphasize that the main factor in determining your blood cholesterol is what the body factory is doing, not what you eat. Many people had heard that the body manufactures its own cholesterol (about 80% of it) but had never heard it put that way. The setting of the body factory is inherited and, if unsuitable, can best be changed by drugs for cholesterol control. The drug that does everything right is niacin.



How do you present the diet vs. drug question to your patients?

If one wishes to try a careful diet for a couple of months, I have no objection. However, the patient should understand that whatever he/she eats during this trial period must be the diet he/she is willing to use for a lifetime. Diet may lower total and "bad" cholesterol (the largest fraction), but in many patients it does not. Or it may lower these only during periods of weight reduction. When persons learn that since the late 1950's there has been a drug, niacin, which reduces total and "bad" cholesterol while raising "good" cholesterol (which diet does not do), most people prefer to take medication and continue to enjoy eating as they please.

Over the years there have been convincing studies showing that persons will not follow sufficient changes in diet to meet the goals of treatment. These are discussed in the book. An excellent community study in California showed that diet reduced total cholesterol by a trivial amount (about 5%) and lowered HDLC ("good" cholesterol) as much as LDLC ("bad")--not a desirable change.

In a nutshell: diet just doesn't work. If any part of your cholesterol profile is in an undesirable range, you have inherited a body factory that is responsible. Treatment must change the body factory. This requires medication. The book explains, for doctors and patients alike, why niacin is far preferable to the billion-dollar statin

drugs.



How important is the long-term safety record of niacin, compared to drugs introduced more recently?

Anyone who thinks about carefully will realize that it is quite important. Of the best-selling "statins," the first one was marketed in 1987, giving it a record of clinical use for just more than 10 years. Others were introduced in 1991, 1993, 1997, and 1998. Early in 1997 one of the drugs introduced in 1991 was found to have a serious interaction with several commonly used drugs, including antidepressants and erythromycin. This hazard applies to some, but not all, of the statins.

Doctors at Stanford and University of California at San Francisco have raised the question of cancer from long-term use of statins. They point out that these drugs are carcinogenic (cancer-producing) in rodents at doses 2 to 30 times those used in humans. They also point out that known cancer-producing agents, like tobacco and asbestos, take 20 to 40 years of exposure before the cancers appear. They specifically advise against use of statins in younger people, but it seems wise to consider their reasoning when choosing a drug for cholesterol control in every patient.



BILL CLINTON'S CORONARY BYPASS SURGERY

Bill Clinton's coronary bypass surgery has heightened interest in coronary surgery. A radio talk show host asked Dr. Parsons how he would have handled Clinton's health problems if the ex-president had been his patient. The author replied that the book takes the position that ***heart attacks are preventable***. He would hope that, if Clinton had been under his care over the years, all of his risk factors would have been so well controlled that he would have not reached the brink of a heart attack, requiring surgery.

He reminded listeners that *diet has so little to do with your blood cholesterol level that we might as well say that it has nothing to do with it*. He then went on to say that Clinton's much-publicized penchant for fast foods had more to do with his weight than with his cholesterol level. The cholesterol level was inherited, as evidenced by his relatives' having premature heart attacks.

Regarding the ex-president's stopping his statin drug when his cholesterol level came down, that was an unwise move. His doctors say Clinton will be taking a statin now. Parsons cited the new (July 2004) guidelines, written by a panel of experts who have been paid by the statin drug manufacturers in the past or currently. He pointed out that if the guidelines are implemented (and they will be, since most doctors are clueless on this matter), they will cost American consumers billions of dollars and will cause deaths.

See next question and answer, **Why are the new (July 2004) cholesterol guidelines unwise and dangerous?**



Why are the new (July 2004) cholesterol guidelines unwise and dangerous?

In July 2004 a panel of experts, meeting in Bethesda, Maryland, issued a new set of guidelines for cholesterol control. The previous guidelines (2001) had been forged by a panel of a couple dozen experts. This time there were just eight such experts, plus the NIH coordinator.

The 2001 guidelines had advised controlling the LDL (bad) cholesterol to **below 130** if you have not had a heart attack or other artery event and **below 100** if you had already had such an event. After reviewing five new studies, done in recent years and sponsored by manufacturers of statin drugs (Lipitor, Zocor, Pravachol, several others), they issued a new edict that recommended LDL **below 70!** And this was not just for those with a previous heart attack but for everyone "at high risk of heart disease." They advised using statin drugs to reduce LDL cholesterol to these levels.

Treating all or most of these people with statins would start the drugs for millions of people and increase the dosage for millions already taking statins. This would deplete wallets by more billions of dollars and would cause deaths. Keep reading.

That is a huge number of people! It includes smokers, those with high blood pressure (whether treated or not), those with abnormal lipid patterns other than too-high LDL cholesterol, diabetics, obese persons, and those without adequate physical exercise.

Four days after the AP news story about the new guideline, another AP story appeared, revealing that **all of the expert panel (excepting only the government coordinator) have received moneys from the manufacturers of statin drugs.** Such payments take many forms: consulting or speaking fees, research money, or other support from companies producing statins, drugs that sell billions of dollars each year. The 2003 update of Cholesterol Control Without Diet! The Niacin Solution contains a chapter with more details of how FDA (and in this case National Institutes of Health) repeatedly ignore federal law which states that experts with conflicts of interest may not serve on guidelines committees or advise regarding approval of new drugs.



What are the dangers of the new guidelines?

Let's go back. In May 2001, Bayer withdrew from the market its entry into the statin drug field, Baycol, which had been on the market for about two years. They withdrew it because it had caused **31 deaths** in the US and more worldwide. At that time the Washington watchdog group, Public Citizen, reviewed FDA records and found that the five statin drugs remaining on the market (Lipitor, Zocor, Pravachol, Lescol, and Mevacor) had caused **81 deaths**. Later Bayer admitted that the figure of 31 deaths had been inaccurate; the actual number was **at least 100. That's at least 181 deaths until mid-2001!**

The author does not know why there has been no further tally of deaths since

then.. He notified FDA that it is really their responsibility to tally deaths since 2001 and bring that total to the attention of the medical profession and the public. Their reply was a bureaucratic response, saying that they will not do this and offering the desired data under the Freedom of Information Act--by jumping through many hoops (such as a separate request for each drug about which information is requested) and spending money to reimburse the agency for the time their employees spend in developing the information. Two later letters emphasizing FDA's responsibility to protect the public, have met equally negative responses.

The author has tried to persuade Public Citizen to tally deaths since 2001 (from CD's of FDA records). To date their point man for statin matters has explained their failure to follow up with a current tally by saying, "The CD's are six months behind." [Well, then: Why not tally to July 2004 or January 2005 and publicize the number of deaths till then?]

Here's how deaths occur. Many, perhaps most, persons who take statins suffer from muscular aches (myalgia). In some, this becomes more severe and is accompanied by muscle tenderness (myositis). In a relatively small percentage, the process goes on to actual **dissolving of muscles**. The medical term for this is "**rhabdomyolysis**"--pronounced "rab-doe-my-ol-us-us." Doctors often shorten this in conversation to "**rhabdo**." Just remember that it means **dissolving of muscles**.

Myoglobin, like hemoglobin, was meant to be in cells, not floating free in the plasma. When myoglobin is in plasma, the kidneys filter it out of the blood. The problem is that this can block kidney tubules, which can lead to kidney failure and death. Nephrologists (kidney specialists) state that they see patients with "rhabdo" quite frequently and sometimes have to use dialysis to save their lives. Some are not so fortunate; then deaths occur.

No one knows how many persons have "rhabdo," placing their lives in danger. Nephrologists say that *they see "rhabdo" so frequently that they do not bother to report it unless the patient dies!*

Here's another unfortunate situation: certain drugs, if taken along with statins, can trigger the muscle-dissolving and its potentially fatal consequences. This happens because the statins are mostly removed from the body by an enzyme system in the liver, called P-450. So are a number of other drugs. The book talks about this.

Let's look at one possible scenario. Suppose you see a nose and throat specialist because of sinusitis, and he/she says, "The best drug for this is erythromycin. I'll give you a prescription." Maybe the doctor doesn't even know that you are taking a statin, or doesn't know that erythromycin is removed from the body by the same liver enzyme. If you take these two drugs together, the statin level in your blood goes sky-high. This may dissolve your muscles, shut down your kidneys, and cause death.

A fair number of drugs are removed by the P-450 system. Strangely, so is grapefruit juice! I have recently read that taking a Zocor tablet with a glass of grapefruit juice is the same as taking 10 to 15 tablets with a glass of water! Not only may doctors be unaware of this P-450 enzyme interference; but other drugs will come along in the future with the same potential for serious harm.

For a few weeks after the Baycol withdrawal, newspaper stories warned people, "If you have muscle pains while taking a drug in this group, *stop the drug immediately* and notify your doctor." Then those warnings no longer appeared. Of course, the fine print at the bottom of advertising pages (and fast talk at the end of TV commercials for statins) mentions muscle aching and advises, "Tell your doctor what other medications you are taking." In the author's view, these are not adequate warnings regarding a possible fatal complication. Public Citizen agrees. They recently pointed out that the FDA approved Crestor, the first new statin in several years, despite the fact that pre-clinical investigations (before the drug's approval for marketing) had shown several cases of muscle-dissolving and kidney failure (no deaths, fortunately). Crestor was the first statin drug to show these effects in pre-marketing studies. Public Citizen felt that Crestor should not have been approved, but FDA approved it. It is now being advertised heavily.

In June 2005 FDA held hearings on the charge that Crestor is the most dangerous of all statin drugs and should either be taken off the market or accompanied by stern warnings about its hazardous potential. The author does not know the entire content of the hearings except that Sidney Wolfe, MD (Director of the Public Citizen Health Research Group) did testify. Obviously his evidence and recommendations were ignored by FDA, which took the position that the risk of rhabdomyolysis and death from statin drugs is small *and Crestor was no more deadly than any of the others!* (Sure, hundreds deaths from drugs taken by millions of people is a small percentage--*but those hundreds of people are still dead!*)

Whether it is true or not, it appears that FDA has decided to take the side of the pharmaceutical companies, who desire to sweep hundreds of deaths under the rug, rather than the interest of the public--whom we had always believed it was FDA's mission to protect. A Public Citizen representative has told the author that each year FDA receives \$150 million from the pharmaceutical industry, as part of their operating expense. Each person and organization must decide for themselves whether this sets up a conflict of interest or not. The fact that FDA uses experts to advise on approval of drugs that are paid by pharmaceutical companies and uses authors of guidelines that are also paid by drug companies is addressed in the book (pages 135-136).

It was a better world when pharmaceutical companies were not permitted to advertise directly to the public. Cholesterol Control Without Diet! The Niacin Solution discusses the huge expenditure of pharmaceutical companies on direct advertising--and its effect on drug prices, a very important issue in all of our lives.

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