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STATIN STUPIDITY AND CHOLESTEROL CONFUSION

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Don't get me wrong. "Statin" drugs like Lipitor, Zocor, Pravacol, Lescol and Mevacor are clearly effective in reducing the incidence of heart attacks and deaths due to coronary heart disease. Unfortunately, they can have insidious side effects that either did not surface during clinical trials or were not reported. They have also been skillfully suppressed in subsequent promotional efforts so that few physicians are aware of these potential problems.

What we are told is that statins are contraindicated during pregnancy and lactation. Because they can cause abnormalities in liver function tests, periodic monitoring should be performed and statins should be used with extreme caution if there is a history of liver disease. Patients must also be warned about the possibility of developing a rare muscle disease known as rhabdomyolysis. This is a disorder in which massive muscle cell destruction floods the system with waste products that can cause complete and irreversible kidney shutdown.

Baycol (cerivistatin) was approved in 1997 after it was demonstrated to be effective in lowering cholesterol and no serious problems had been encountered in over 3,000 clinical trial participants. It was withdrawn last August after 31 patients died from rhabdomyolysis. The Baycol incident illustrates the type of communication failure that has happened before and will undoubtedly continue to occur with other drugs unless the present warning system is revised.

A similar problem occurred with Rezulin, a diabetes drug **approved by the FDA in 1997 over the objections of its own physicians** because of evidence it could cause fatal liver failure. After these fears came to fruition, the agency and the manufacturer sent four separate warning letters to doctors alerting them to the problem and the need to monitor liver function tests monthly. However, the vast majority of doctors either didn't get or heed the message. Five months after the last warning, only 5 percent of physicians were regularly performing the recommended liver tests. Rezulin was withdrawn in March 2000, but only after more than 60 deaths due to liver disease had been reported.

ALSO INCLUDED IN THIS ISSUE

A review of governmental correspondence revealed that **senior FDA officials had collaborated closely with the manufacturer during the approval process as well as later**, when a whistle-blowing consumer watchdog group exerted pressure to ban the drug. **The extent of this collusion and connivance is alarming.** Th

Why FDA Failures Threaten Our Safety

The Rezulin experience provides important insights into why there are concerns about statin safety. Much of this information came from investigative reporters who secretly obtained documents and e-mail communications showing that the FDA helped the manufacturer play down the potentially fatal risks of Rezulin during the approval process and provided the company with inside information and favors. According to the *Los Angeles Times*, Dr. John Gueriguian, a FDA medical officer assigned to examine Rezulin, told the company in 1994 that he was concerned about "potential toxicities". His boss, Dr. G. Alexander Fleming, reassured the manufacturer in 1995 that he would "ease Dr. Gueriguian out" if they were displeased with him, according to one memorandum. Sure enough, Gueriguian was removed from the case in 1996.

Dr. Fleming e-mailed a copy of Dr. Gueriguian's unflattering medical review to the company but withheld it from the Advisory Committee that examined the drug. Two days before the committee meeting, Fleming e-mailed the manufacturer that "the drug looks like it ought to be on the market. Loosen up and put on a good presentation. Call if you need help."

After the drug was approved it became clear that it posed a significant health hazard with respect to liver failure. Rhabdomyolysis was also reported as a complication. However, the FDA continued to drag its feet despite pleas from its own officials to withdraw the drug several months before those in charge were forced to make this decision. The action was finally taken a few days after a FDA physician sent a letter to Congress imploring its help in persuading his superiors to withdraw the drug. Only a few hours before announcing the Rezulin ban, FDA officials had assured news organizations and the public that the agency had "plenty of weapons" to control and reduce the problems associated with the drug!

As pointed out in previous Newsletters, many believe that this and similar problems, such as the weight loss drug Redux fiasco stem from lowered safety standards that have led to approving drugs too rapidly and withdrawing them too slowly when a problem is uncovered. This is due to a lack of personnel to evaluate and monitor adverse reactions and a disturbing increase in the influence drug companies have over FDA approval and regulatory activities. The approval time for a new drug dropped from an overall average of about 30 months in 1988 to approximately 13 months in 1999. **During the two-year period from 1998 the FDA has had to recall six drugs they had previously certified as safe.** The FDA is dependent on drug company funding and despite obvious conflicts of interest; many FDA committee members have strong financial ties to the companies whose drugs they review.

The Baycol withdrawal may be just the tip of the statin safety iceberg. There have been 81 deaths from rhabdomyolysis linked to other statin drugs and the figure is undoubtedly much higher since many possible cases were excluded. Because doctors and hospitals are not required to report adverse reactions, academic and government statisticians estimate that **there are probably at least 10 times the number of patients who experience statin side effects for each case officially reported.** And new side effects are starting to surface.

How Safe Are Statins?

Last August, the Public Citizen's Health Research Group petitioned the FDA to add a "black box" warning to all statin packaging that would be in bold type surrounded by a black box to make it stand out. The agency should also require that additional warnings in bold type be added to the package inserts of these products. In addition, they should require that a medication guide be distributed to all patients filling statin prescriptions advising them to immediately stop using the drug if they experience muscle pain, tenderness,

weakness or tiredness. Finally, drug companies should be required to send "dear doctor" letters to all health care professionals about the risk of muscle damage due to statins.

According to the group's director, **"labeling on statins is inconsistent and dangerously inadequate. Most people taking these drugs aren't aware that they could sustain serious muscle damage and could even die from taking these drugs."** These warnings are particularly necessary in light of the recent government report recommending that 23 million more people take cholesterol-lowering drugs, including many with normal cholesterol and LDL values. One day after Baycol was banned The European Medicines Evaluation Agency announced they would establish an Advisory Panel composed of one representative from each European union member state to conduct a safety review of other statin drugs. This panel has no authority to enact any changes and can only make recommendations.

There is evidence that statin side effects are increased and are more severe in older patients as well as those who also take other drugs to lower cholesterol and triglycerides, like Lopid (gemfibrozil). It also seems quite clear that side effects are directly related to dosage, which could prove to be a serious problem. The goal of therapy is to lower LDL to an arbitrary level despite overwhelming evidence that statin cardioprotective benefits are not related to effects on any lipid concentrations. This means that the dosage and duration of treatment will be steadily increased if the desired result is not achieved.

The following letter was published in the November 21st issue of the *Journal of The American Medical Association*.

> **To the Editor:** The NCEP guidelines focus on lowering LDL-C and also expand the population to be treated to include individuals at increased risk for coronary heart disease. This implies that those with clinical evidence of atherosclerosis elsewhere, multiple risk factors (eg, hypertension, cigarette smoking), and all patients with diabetes should be taking cholesterol lowering drugs, primarily statins, perhaps perpetually.
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> There is little doubt that statins can significantly reduce coronary mortality but using LDL levels to determine dosage and duration of treatment may be inappropriate. The atherosclerotic plaques of coronary artery disease are the result of an inflammatory response with foam cells, macrophages, smooth muscle cell proliferation, and neovascularization. Perhaps 80% to 90% of acute coronary thromboses are due to fissuring and rupture of these plaques. Statins have significant stabilizing effects on plaque in patients with elevated LDL as assessed by a reduction of C-reactive protein to safe levels in 70% to 80% of patients after only 6 weeks. However, no correlation has been found between lowering CRP and LDL concentrations. This suggests that the cardioprotective effects of statins are related more to their anti-inflammatory properties than their lipid effects. In fact, the ability of statins to prevent recurrent coronary events following myocardial infarction also results from reducing inflammation rather than any lowering of lipid levels, since at least half of patients with myocardial infarctions have normal LDL levels and CRP measurements may be a more predictive marker for atherothrombotic events.
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> The adverse effects of statins are also a concern. In addition to liver disease, patients taking statins also experience muscle pain due to rhabdomyolysis, unusual fatigue, emotional disturbances, confusion, memory loss, and amnesia. These improve when statin therapy is discontinued. Adverse effects will undoubtedly increase under the new guidelines as more people are treated. It might be advisable to determine the minimum statin dosage that provides cardioprotection. As with aspirin, this may be much lower than for other indications.
>
> Paul J. Rosch, MD
> Yonkers, NY

Since its publication in the November 21 issue, I have received numerous reports from patients and physicians suggesting there may be many other statin side effects.

Cancer, Retinopathy And Shingles?

Once the statin bandwagon started rolling it began to take on the appearance of a steamroller with claims that these wonder drugs could not only prevent heart disease but cancer, osteoporosis and Alzheimer's disease. There is not only little rationale for this based on lipid lowering actions but evidence suggesting the exact opposite in some instances.

There have been numerous reports in the literature that low cholesterol and/or the administration of cholesterol-lowering drugs

is associated with an increase in malignancy. A 1995 review of the subject found that all statin and fibrate drugs used to lower lipids caused cancer in rodents, in some instances, at dosages equivalent to those commonly prescribed for patients. **Noting that it may take decades to demonstrate such effects in humans, the authors concluded that "in the meantime, the results of experiments in animals suggest that lipid-lowering drug treatment, especially with the fibrates and statins, should be avoided, except in patients at high short-term risk of coronary heart disease**."

In one statin study there were 13 cases of breast cancer in the treated group compared to only one in placebo controls, but this is never mentioned in advertisements or the guidelines. In another, the active group had more non-melanoma skin cancers. These relatively short-term findings are somewhat scary and since they are not well known, the development of cancer in patients on statins may be viewed as pure coincidence, especially in the majority who are also taking other drugs.

Cancer could result from depletion of Coenzyme Q10, a well-documented effect of statin therapy. In one study in which 14 patients with recurrent prostate cancer were given Q10 for 300 days, ten had an average reduction of 73 percent in elevated PSA levels and prostate size shrunk to almost half. The four who did not respond as favorably had the largest prostates and the greatest number of metastases. If Q10 can reverse prostate cancer and statins inhibit Q10 biosynthesis, is it possible that statins stimulate prostate cancer growth?

Recent research reveals that statins stimulate angiogenesis, or growth of new blood vessels, which could potentially promote cancer growth. Tests in human cell samples and in rabbits show that Zocor mimics VEGF, a natural growth factor that helps to regulate blood vessel development. VEGF may also help to spread colorectal cancer. In one report, survival time was significantly less in patients whose tumors tested positive for this growth factor. If statins share this VEGF effect it could also explain a cancer connection.

Another very recent study shows that VEGF can contribute to the development of diabetic retinopathy. It would be difficult to detect if statins act in a similar fashion since the occurrence of this complication is hard to predict. This is of concern since many diabetics have heart disease and are likely taking statins. In addition, the new guidelines recommend that all diabetics should be on statins, even if their cholesterol and LDL are normal.

Following my *JAMA* letter and another to the *British Medical Journal*, I received an e-mail from a 57-year-old lady who had been in excellent health and was started on 10 mg. of Lipitor because of "high cholesterol". Four weeks later her gums became sore and soon after she developed shingles. She believed that Lipitor was responsible and asked for my opinion about this and if I knew of similar cases. I was not aware of any but explained that statin induced Coenzyme Q10 depletion could impair immune system resistance to herpes and other viral infections. She had developed gum disease, which like memory loss with statins, improves rapidly with Q10 supplements and I suggested she take 100 mg. daily. **Drug companies are well aware of this problem but never mention it. Merck has had patents for a statin-Q10 combination capsule since 1990!**

Q10 is a powerful immune system stimulant and has been shown to increase T4/T8 lymphocyte ratios, which tend to be low in AIDS and cancer patients. I recently learned from the group studying statin safety that they had reports of patients who had developed shingles and other viral infections. They also confirmed the benefits of Q10 supplements.

The Hazards Of Low Cholesterol

Regular readers of this Newsletter must be tired of hearing me rant about this for the past two decades. However, it is important to revisit this issue since recent research studies support this view. In addition, others expose the numerous fallacies of the diet-cholesterol-heart attack hypothesis. Some of the dangers associated with lowering cholesterol include:

CANCER

Researchers thought that the Framingham and other large studies would demonstrate that elevated blood cholesterol levels were associated with an increased incidence of malignancy. However, the data revealed just the opposite. Of the more than twenty studies published over the past two decades most also confirm an association between cancer and low blood cholesterol. While critics claim that the low cholesterol is likely due to cancer rather than vice versa, this is refuted by one study in which cholesterol values over time were studied in patients with colon cancer. Researchers found that there had been an average thirteen percent decline in the ten years prior to the diagnosis of cancer compared to an average increase of two percent in controls. Both groups had comparable cholesterol levels at the beginning of the study.

STROKE

A very large Japanese study covering two decades concluded that low cholesterol levels were associated with a significantly increased incidence of stroke. Further support comes from a follow-up of 350,000 screened for the MRFIT study in the U.S. showing that **deaths from hemorrhagic stroke were six times greater in those with low blood cholesterol levels.** Although the Framingham study found no relationship between cholesterol levels and stroke, it did show that every three percent increase in fat intake was correlated with a fifteen percent reduction in stroke.

SUICIDE, DEPRESSION AND VIOLENCE

One study reported that male psychiatric **patients with cholesterols under 160 had twice the rate of suicide as controls with normal values.** Another showed that men with similar low cholesterol measurements had a three fold greater incidence of depression and were also at greater risk for suicide.

In a survey of 121 healthy young college women, those with cholesterol levels under 160 were also much more likely to score high on measures of depression as well as anxiety than controls with cholesterol values over 180. Violent behaviors have been significantly linked to low cholesterol levels in a variety of studies. All these problems are believed to result from low levels of brain serotonin. Cholesterol is crucial for the production of serotonin.

PREMATURE DEATH IN CHILDREN

Governmental recommendations are that children over two adopt a low-fat, low-cholesterol diet to prevent heart disease in adult life. This is based on a correlation between fat and cholesterol intake and blood cholesterol found in seven to nine-year olds from six countries. What was not revealed was a much stronger correlation between average cholesterol and death rates/1000 in children under 5 years old.

Country	Cholesterol	Childhood Deaths
Finland	**190**	**7**
Netherlands	173	9
USA	**166**	**12**
Italy	158	12
Philippines	**146**	**72**
Ghana	**127**	**145**

BAD NEWS FOR THE ELDERLY

In one study, senior citizens with blood cholesterols over 250 had less than half the mortality of those whose level was around 200. In the 30-year Framingham follow-up, high cholesterol did not predict coronary death after age 47 and a falling cholesterol was associated with increased death rates. For each 38 mg. drop in cholesterol, coronary and total mortality increased 11 percent. Another study in the elderly showed that each 38 mg. rise in cholesterol corresponded to a 15 percent decrease in mortality. The Honolulu Heart Study that followed 8000 men for 35 years just reported a 50 percent increase in death rates for those with cholesterols 167 or lower compared to others whose average was around 200. Men whose cholesterols had been low for twenty or more years were at greatest risk for premature death. This would seem to refute the claims of critics who argue that low cholesterol is the result of illness rather than its cause.

The Real Reason Statins Work

As noted originally, there is little doubt that statins can provide significant benefits for patients with coronary heart disease and possibly other disorders as well. However, this does not appear to be related to their ability to lower total cholesterol or LDL but rather their anti-inflammatory and possibly other effects. Evidence for this appears to be overwhelming.

The obstructive lesions of atherosclerotic plaque are very different than the atheromatous deposits produced by force feeding experimental animals high cholesterol diets both in appearance and location. Plaque lesions have all the hallmarks of an inflammatory reaction, many of which are reminiscent of responses to physical trauma or infection. The vast majority of heart attacks result from rupture and fissuring of plaques. Since the ability of statins to reduce coronary events can be demonstrated within two months, it is doubtful that this is due to any significant lowering of LDL or cholesterol. **The most likely explanation is that any benefits result from their anti-inflammatory and stabilizing effects.**

If this were true, then one would expect to see a reduction in indices of inflammation, such as CRP (C-reactive protein) and that is precisely what occurs. CRP is lowered to safe levels in seventy to eighty percent of patients with coronary artery plaque after six weeks of statin therapy without any appreciable change in elevated LDLs. The benefits of statins in preventing recurrent coronary events following a heart attack are also probably due to their anti-inflammatory effects since at least half of these patients have normal LDL levels.

Stent implantation to relieve coronary artery stenosis can be an effective alternative to bypass procedures but postoperative complications can occur, including a recurrence of the stenosis. Statins reduce the risk of complications not by lowering LDL but because they reduce inflammation. A study in the December issue of the *Journal of the American College of Cardiology* followed 388 consecutive patients who underwent coronary stent implantation, 249 of whom received statins.

An elevated CRP was present in 207 patients. Of this group, those who did not receive statins were 2.37 times more likely to experience a major adverse cardiac event compared to treated patients with normal CRP measurements. Complication rates in the statin group with high CRP values were significantly reduced to about the same degree as that seen in untreated patients with normal measurements. There was no correlation with any changes in cholesterol or LDL, which were minimal. The researchers concluded that CRP levels may be the best way to determine which patients will benefit most from statin therapy.

There are claims that statins can help to prevent other disorders such as Alzheimer's. Statin manufacturers would have you believe increased cholesterol may favor the production of amyloid deposits thought to be responsible for Alzheimer's disease. The fact is that half of the brain is composed of fats and according to one authority, treatment should consist of "strategies for increasing the delivery of cholesterol to the brain". In one study, statin treated patients showed problems with memory and attention span after six months, probably from CoQ10 depletion. If statins do help prevent Alzheimer's, it is likely due to their anti-inflammatory properties rather than any lipid-lowering effects. The same is true for claims of benefits in senile macular degeneration, another multibillion-dollar market.

Determining dosage and duration of statin therapy by its ability to lower LDL to an arbitrary level that has nothing to do with its clinical effects is inane. It can only lead to raising the dose, which will increase the incidence of adverse side effects. Recommendations are also that all diabetics should be on statins even if LDL is normal and there is no evidence of coronary disease. How will these dosages be determined? How long will we continue to be hoodwinked by the cholesterol cartel and the minions under their control?

Uffe Ravnskov's *The Cholesterol Myths* expands on this. Because of limited space, My Newsletter comments on this book could not do it justice. However, the following review by Steve Byrnes is right on target.

The Cholesterol Myths by Uffe Ravnskov, MD, PhD,
New Trends Publishing; Washington, DC; 2000. $20.00. 300 pp.
Available from New Trends Publishing, 877-707-1776; Stephen Byrnes, ND, RNCP

Would you buy a book that was literally set on fire by its critics on a television show about it in Finland? I would and so should you. The long-awaited English version of debunker extraordinaire Dr. Uffe Ravnskov's notorious Cholesterol Myths is now available from New Trends Publishing.

Ravnskov, a medical doctor with a PhD in Chemistry, has had over 40 papers and letters published in peer-reviewed journals criticizing what Dr. George Mann, formerly of Vanderbilt University, once called **"the greatest scam in the history of medicine": the Lipid Hypothesis of heart disease - the belief that dietary saturated fats and cholesterol clog arteries and cause atherosclerosis and heart disease.**

If one thing comes through as you read the book, it is this: Ravnskov has done his homework. In painstaking detail, he critically analyzes and demolishes the nine main myths of the Lipid Hypothesis: 1) High-fat foods cause heart disease; 2) High cholesterol causes heart disease; 3) High fat foods raise blood cholesterol; 4) Cholesterol blocks arteries; 5) Animal studies prove the diet-heart idea; 6) Lowering your cholesterol will lengthen your life; 7) Polyunsaturated oils are good for you; 8) The cholesterol campaign is based on good science, and 9) All scientists support the diet-heart idea.

Equipped with a razor-sharp mind, an impressive command of the literature, and a deadly, needling sarcasm, Ravnskov methodically slaughters the Sacred Cow of modern medicine and the most profitable Cash Cow for assorted pharmaceutical companies. Sparing no one, Ravnskov again and again presents the tenets of the Lipid Hypothesis and the studies which supposedly prove them, and **shows how the studies are flawed or based on manipulated statistics that actually prove nothing**. Ravnskov then answers the objections or rationalizations offered by diet-heart supporters, desperate to explain away inconsistencies and contradictions in their own data.

For example, Ravnskov opens with an analysis of the study that kicked off the Lipid Hypothesis in the 1950s: Ancel Keys' Six Countries Study (and later, the more famous Seven Countries Study). As most health professionals know, Keys' study showed that countries with the highest animal fat intake have the highest rates of heart disease. Keys' conclusion was that there was a cause and effect relationship because the country with the lowest animal fat intake (at that time, Japan) had the lowest rates of heart disease. Sounds convincing, right? Not so, says Dr. Ravnskov. And in a few pages the reader is informed how **Keys hand-picked the countries he included in his studies, namely, the ones that supported his hypothesis, and conveniently ignored all of the other countries that didn't.**

And this is just the beginning! Ravnskov approaches true brilliance in his review of the studies that supposedly showed benefit from the current wonder-drugs pushed by the pharmaceutical industry: the statins. **Hailed as miracle substances that "significantly reduce cholesterol and incidence of heart attacks," Ravnskov shows that these substances are probable carcinogens (women on the drugs had a much higher incidence of breast cancer) and that the overall statistical reduction of heart disease in the drug trials was negligible. Nevertheless, despite the dismal results of the very first trial (the EXCEL Trial which Ravnskov soberingly describes to the reader), the industry and its well funded doctors urge their use, even in people who do not have heart disease.**

Ravnskov warns: "**Because the latent period between exposure to carcinogen and the incidence of clinical cancer in humans may be 20 years or more, the absence of any controlled trials of this duration means that we do not know whether statin treatment will lead to ... cancer in coming decades. Thus, millions of people are being treated with medications the ultimate effects of which are not yet known."**

If there is one weakness of the book, it is its lack of explanations of what does cause heart disease. Ravnskov comes close to fingering a few factors such as high stress, excessive polyunsaturated fat intake, trans-fatty acids, and smoking, but he never offers his own theory as to what causes the Western world's number one killer.

This is, however, a minor glitch. Ravnskov has done the world a major service in presenting his findings. All health professionals need to listen to this scholar and **listen very carefully, for the advice offered by the medical establishment for the last 50 years to beat heart disease has failed miserably.** It is time to turn away from cholesterol-lowering drugs that have frightening side effects. It is time to turn away from low fat diets that harm children and deprive people of fat-soluble vitamins. And it is time to turn away from the junk science that characterizes the Lipid Hypothesis and its supporters. It is time to listen to reason and to view all of the evidence against a failed hypothesis and discover the true and varied risks and causes of heart disease. It is time to listen to Uffe Ravnskov.

Certain highlighted statements deserve emphasis, starting with the amazing fact that opposition to Uffe's book was so intense that it was actually burned on a television show when it first appeared. However, despite their violent objections, critics have been unable to refute any of his statements. Since then, other doctors and scientists have increasingly joined Uffe in his debunking campaign. Our ranks have swelled to about 50 and our voices are starting to be heard.

The Times They Are A-Changing And The Tide Is Finally Starting To Turn

As Thomas Huxley noted, "*The tragedy of science is the slaying of a beautiful hypothesis by an ugly fact.*" My interest in the cholesterol controversy was kindled by Sir John McMichael, Professor Emeritus of Medicine, University of London and a highly respected cardiologist. In a 1979 *British Medical Journal* article he wrote, *"All well-controlled trials of cholesterol-reducing diets and drugs have failed to reduce coronary heart disease mortality and morbidity. Nevertheless, commercial, professional, and even government-sponsored propaganda continues . . . Official medical endorsement of these cholesterol reducing measures should be withdrawn".* This was in response to a clofibrate cholesterol-lowering trial that had ***"unacceptable risks which could also apply to diets."***

Other distinguished physicians subsequently protested, including George Mann, "Pete" Ahrens, Stewart Wolf, William Stehbens, Michael Oliver and Ray Rosenman. They were no match for the might of the cholesterol cartel who manipulated the media as well as influential physicians with vested interests. Journals often rejected contributions critical of the prevailing dogma even when they were supported by solid scientific data. Even when letters pointing out flaws in a previous article were published, those being criticized rarely responded to the specific questions that were raised, as was the case with my recent *JAMA* letter. We have regularly included sessions devoted to debunking the diet-cholesterol heart attack hypothesis at our International Congress on Stress featuring presentations by most of the above individuals and several Newsletters have also dealt with various aspects of this topic over the past two decades.

Many of these contributions emphasized the role of stress in coronary heart disease, as well as how stress can contribute to conventional risk "factors" like elevated cholesterol, hypertension and cigarette consumption, although these are only markers. Most of my colleagues already thought I was a little far out because of my preoccupation with the role of stress in disease, but when I attacked the sacred cholesterol cow they became convinced I was off my rocker. While writing this Newsletter I received a call from a close friend confirming this. After retiring as Director of Surgery he moved away and although I have not seen him for several years, we talk every few weeks. He called to say that based on a previous Newsletter, he decided to take CoQ10 and was astounded to find that within a week or two there was no longer any blood on his toothbrush. I have heard many similar stories, particularly with respect to the dramatic effects of Q10 in patients with heart disease and it is very reassuring to see that the tide is changing.

Other organizations have joined Uffe's campaign and a group of international scientists recently sent a strong letter to the FDA warning that "It is possible that the recently reported statin-related deaths are the tip of a side effect iceberg and the magnitude of the potential problem cannot be overstated." There are strong concerns that statins may interfere with cholesterol production in the brain needed for neuronal connections. For more on this and the role of stress - stay tuned!

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