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STATINS, CHOLESTEROL, STRESS & CORONARIES

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There have been so many conflicting and confusing reports about statin safety and efficacy that I wanted to revisit this topic for those who "stayed tuned." In many, if not most instances, these have confirmed the concerns originally expressed about the ability of powerful vested interests to preserve their exorbitant profits by using a variety of nefarious strategies.

Also Included In This Issue

Pharmaceutical companies are a prime offender. As emphasized in previous Newsletters, promotional TV ads hype the benefits of statins for everyone by using deceptive relative risk statistics, while minimizing or ignoring significant adverse side effects. The facts are , save for patients at very high risk, statins do not provide any benefits for senior citizens, or females of any age.

Lipitor is a good illustration. Although not significantly superior to or safer than other statins, **it has raked in approximately $130 billion during its 14 years on the market**, making it the world's best selling drug ever. Most patients spend $115 to $187 for a month's supply of Lipitor, but now that a much less expensive generic version is available, the price will plummet. The co-pay for a generic is usually $10/month, and to combat this, Pfizer is offering a free card that will reduce costs to **$4.00/month, and it is still making a profit.** Watson Laboratories currently has the only approved generic Lipitor, and in return, Pfizer has crafted a deal that will allow it to

receive 70% of their profits for the next five years. Other companies will be allowed to market a generic version in six months and Pfizer will likely also have their own generic, so the price will probably drop even further.

Why Cholesterol Is Merely A Marker And Stress Is Much More Important

It is now quite clear that elevated cholesterol does not cause heart attacks, and that lowering it in healthy people not only fails to provide any benefits, but can also be dangerous. Despite this, the statin juggernaut rolls on with a multimillion dollar propaganda machine that now promotes statins not only to prevent and treat coronary heart disease, but also degenerative and a dozen or more other disorders. That's not surprising, since having found such a lucrative cash cow for treating coronary disease; it is only natural that statin manufacturers would attempt to find other fertile fields to plow. But there were several problems that stood in their way, the most important being how to explain the efficacy of statins in conditions where cholesterol was normal or low and no rationale to support any value from lowering it. In addition, some of the benefits of statins in coronary disease occurred much too rapidly for them to be due to lipid lowering, they were seen whether cholesterol was high, normal or low, and at least half of the patients admitted for heart attacks had perfectly normal lipid levels. To circumvent this, it was proposed that in addition to lowering cholesterol and LDL, statins had other "pleiotropic" activities that could counter all the above criticisms and justify their use in other diseases.

In genetics, pleiotropy refers to a phenomenon in which one gene results in several different physical characteristics. The beneficial pleiotropic effects of statins include anti-inflammatory and antioxidant properties that protect the arterial wall from being damaged by cholesterol, and vasodilatation that allows widened arteries to deliver more blood to the heart and other tissues. Statins also help to prevent clot formation by inhibiting platelet aggregation and reducing blood viscosity, which would facilitate the flow of blood through partially blocked arteries. There were also adverse pleiotropic responses that affected the liver, muscles, and other organs and functions, but these side effects were generally dismissed as being uncommon, usually mild, and reversible. Statin manufacturers maintained that the virtues of statins would also outweigh such hazards in other disorders, and that these conditions could be readily identified. There were numerous statin trials involving tens of thousands of patients containing massive amounts of data that had not been completely exploited because the studies had primarily concentrated on lipid measurements, coronary events and adverse side effects and complications. There were anecdotal reports of improvement in a wide variety of non-cardiac disorders that needed to be followed up. One way to do this was by scrutinizing all the records to see if these or other diseases were less frequent in statin takers than controls. This is the same fallacious

reasoning, which failed to recognize that association does not mean causation, by labeling them as "risk factors" instead of "risk markers."

The Framingham study established elevated cholesterol, hypertension and smoking as the major risk factors for heart attacks and deaths due to coronary disease. The higher the cholesterol, blood pressure and amount of smoking, the greater the risk, and the effects of these three risk factors were also additive. The fact that these were considered to be causative agencies is evidenced by the massive, long-term MRFIT (Multiple Risk Factor Intervention Trial). Conducted at leading U.S. medical centers it was designed to normalize cholesterol and blood pressure and curtail smoking in middle-aged men at increased risk for heart attack because of these presumed causes. Seven years and $115 million later, although all these goals were achievedm there was no reduction in heart attacks. Not only did the treated group fare no better than their matched controls, **deaths due to coronary disease were highest in the group being treated for hypertension.** While those whose cholesterol and saturated fat consumption had been cut by 42% and 28% respectively had slightly lower heart disease death rates, this modest benefit was far outweighed by significantly increased total mortality rates, especially from hemorrhagic stroke, cancer, suicide, accidents and violence. **The risk of dying from a cerebral hemorrhage was 500% greater in those with low cholesterol compared to those with high levels.**

That was over 30 years ago and we are still making the same mistakes. As I pointed out at the time in a letter to *Lancet*, "Why we smoke, eat rich foods, have high blood pressures and serum cholesterol levels may be much more important than the mere observation of such stress-related statistics." If you get people to stop smoking, you will reduce rates of emphysema and lung cancer, because smoking causes these. Similarly, normalizing elevated blood pressures will help prevent stroke. Lowering high cholesterol (or LDL) will not accomplish anything because they do not cause any diseases other than xanthelasma, harmless deposits of cholesterol under the skin that often have a hereditary component. Like over 200 other risk markers such as a pot-belly, premature vertex baldness, a deep earlobe crease, or peripheral artery disease, cholesterol, hypertension and smoking are merely associated with heart attacks. A tummy tuck, hair transplant, plastic or other surgery may correct these problems, but they will not prevent heart attacks.

Coronary atherosclerosis is a multifactorial disorder, which is another way of saying its cause or causes are unknown. Some likely contributors include irritation due to chronic asymptomatic infections, homocysteine, increased clotting tendencies and hemodynamic factors. Genetic influences may raise

or lower the threshold for each of these and stress can also play a pivotal role. For example, it is not generally appreciated that stress can:

- Increase homocysteine, C reactive protein and fibrinogen, all of which promote inflammation or coagulation
- Decrease resistance to infections that have been increasingly incriminated in the development of atherosclerosis and vulnerable plaque due to obstruction of the *vasa vasorum* by lipoprotein aggregates and microbial remnants
- Cause coronary vasoconstriction, spasm and increased platelet adhesiveness and aggregation that facilitate clot formation
- Cause increased visceral fat deposits that contribute to insulin resistance, diabetes, elevated triglycerides and other manifestations of metabolic syndrome
- Produce myocardial necrosis in the absence of coronary occlusion by increased secretion of catecholamines at nerve endings in the ventricle
- Cause sudden death due to ventricular fibrillation in healthy young patients with no evidence of coronary atherosclerosis

Depression, anxiety, anger, hostility, major life change events, and especially job stress have all been linked to increased coronary events and deaths in scientific studies. Type A behavior is as significant a risk factor for heart attacks as elevated cholesterol, hypertension and smoking. For what it's worth, stress elevates cholesterol far more than dietary fat intake and also contributes to smoking and hypertension. In contrast to cholesterol, some studies have shown that reducing coronary prone Type A behavior, depression, hostility and anger reduced cardiac morbidity and mortality, and that beta-blockers blunt the harmful effects of catecholamines.

Pleiotropic Effects Of Statins – A New Panacea Or The Same Old Snake Oil?

Armed with this new ammunition that statins also had antioxidant, anti-inflammatory, vasodilating and blood thinning properties, the spin-doctors had little difficulty promoting their expanded use for almost everything. It is important to emphasize that once a drug is approved, it can be prescribed for any disease, disorder or complaint, **unless** it has been contraindicated. What follows are excerpts from just one article with quotes that allegedly explain the justification for adding additional diseases that statins might benefit. Some, such as congestive failure, infectious diseases and cancer have been highlighted because they require additional discussion.

BEYOND THE CORONARY ARTERIES

- Stroke and Peripheral Vascular Disease – since statins protect coronary arteries from cholesterol induced atherosclerosis, they should prevent blockage in the carotid and other arteries. One study of

patients with peripheral artery disease reported "improved pain and walking performance after just three months of statin therapy."

- Aneurysms – when the wall of an artery weakens, it can balloon out and become an aneurysm. If an aneurysm bursts, it can be fatal. Studies "suggest that statins may slow the enlargement of abdominal aortic aneurysm."
- Deep vein thrombosis (DVT) – "Although DVTs are not caused by atherosclerosis, studies have reported that statins reduce the risk of DVTs by 22% to 67%, presumably because the drugs fight blood clotting."
- Aortic stenosis – impairs the flow of blood from the left ventricle to the aorta and weakens heart muscle. Although correctable by surgery, "there are no proven noninvasive treatments," cites one study suggesting that statins can slow down the progression of early aortic stenosis and another that might also do this in mitral stenosis.
- Hypertension – results with statins are "mixed", but one study of two different statins found that they "produced a small but significant reduction in blood pressure. If these results are confirmed, they could help explain how statins protect against aneurysms and strokes."
- Atrial fibrillation (AF)– a rapid irregular heartbeat that can cause strokes. "Although the evidence is mixed, some research suggests that statins may protect against AF and its complications, not by lowering cholesterol but by improving vascular function, fighting inflammation, and protecting against oxidative stress."
- Heart failure – often caused by all of the above and manifested by fatigue, shortness of breath, and fluid accumulation. "Although some studies have been negative, others suggest that statins reduce the risk of hospitalization for heart failure. **High-dose statin therapy may be particularly beneficial**."
- Sudden death – an analysis of 10 randomized clinical trials "linked statin therapy to a 19% reduction in the risk of sudden death. The researchers speculated that the apparent benefit is related to a reduction in life-threatening abnormal heart rhythms."

NEUROLOGICAL AND PSYCHIATRIC DISEASES

Although the brain accounts for less 2% of body weight, it contains 25% of all the cholesterol in the body, most of which is in the myelin sheaths that protect nerve cells and facilitate speedy communication between them. Because of the blood-brain barrier that blocks blood cholesterol from entering, the brain must produce it's own cholesterol, so that blood levels do

not reflect those in the brain. The blood-brain barrier also prevents water-soluble statins like Pravachol from entering, but Mevacor and Zocor can.

- Mood And Behavior – "**have not identified any adverse effects on cognitive function or psychological well-being**. In fact, the long-term use of statin drugs has been associated with reduced risk of anxiety, hostility, depression, and suicide, perhaps because of the improved physical health that results from these medications."
- Stroke - because it shares some of the same risk factors as coronary disease, " one of the most important 'side benefits' of statin therapy is a reduced risk of stroke. A meta-analysis of 42 trials involving 121,285 people found that statin therapy reduced the risk of stroke by 16%; nearly all the benefit resulted from protection against strokes caused by blocked arteries (ischemic strokes), and **the study also refuted earlier worries that statins might increase the risk of strokes caused by bleeding. . . . each 40-milligram per deciliter drop in the cholesterol level reduces the risk of stroke by 20%**."
- Dementia –"In 2000 and 2001, observational studies raised the hope that statin therapy might reduce the risk of Alzheimer's disease and vascular dementia. But randomized clinical trials — the same type of stringent studies that established the value of statins for heart disease and stroke in the first place — have failed to confirm these hopes." (It was surprising that studies, which claimed that statins could delay the onset of Alzheimer's or improve symptoms, were not cited.)

GENITOURINARY, PULMONARY AND OTHER DISEASES

- Kidney Disease - "Statin therapy appears to reduce urinary albumin excretion in patients with chronic kidney disease, an important benefit. Statins may also protect against the development of kidney dysfunction and slow progression of established kidney disease. . . a meta-analysis of 26 studies involving 25,017 patients with chronic kidney disease found that statins reduced these patients' risk of cardiovascular death and lowered their overall mortality rate without an increased risk of side effects."
- Benign Prostatic Hyperplasia (BPH) – enlargement of the prostate and symptoms of BPH affect most men middle-aged and older. "A Minnesota study of men between the ages of 40 and 79 linked statin use to a 56% reduction in the likelihood of prostate enlargement and a 52% reduction in BPH symptoms. The researchers credited the anti-inflammatory effects of statins for this apparent benefit.
- Erectile Dysfunction (ED) – Elevated cholesterol is also a risk factor for ED, so it is not surprising that a small study linked statin use with a 27% reduction in risk of developing ED. Other studies of men with ED

and high cholesterol found improved function after taking statins as well as a more satisfying response to Viagra. Statins also accelerated recovery of erectile function after nerve sparing prostate surgery.

- Chronic Obstructive Pulmonary Disease (COPD) - is the fourth most common cause of death in the U.S and studies here and in Norway show that statins can slow its progression and reduce risk of death. "The investigators credit these benefits to the statin drugs' ability to reduce inflammation and oxidative stress, which may also contribute to the statins' potential role in asthma."

INFECTIONS, CANCER AND OTHER DISEASES

- Infections - One meta-analysis of 16 COPD studies found that "patients on statins were **43% less likely to contract infections and 45% more likely to respond to treatment than patients who were not taking statins. The apparent benefit may depend on these drugs' ability to regulate the immune response, reduce inflammation, and prevent excessive blood clotting.** . . . Laboratory experiments show that statins are very effective in protecting mice from bacterial infections."
- Cancer - The approach used here was to grow cancer cells in test tubes with or without a statin drug in the culture medium. Researchers found that, "statins are able to slow the growth of cells taken from individuals who had prostate cancer, bladder cancer, breast cancer, or other malignancies." Some of the possible mechanisms suggested were: altering the expression of cancer genes, affecting the growth of blood vessels, the ability of cancer cells to adhere and spread, and promoting apoptosis, cell death by suicide. In addition, some results suggest that statins may "strengthen the effects of standard cancer drugs or radiation." . . . The most hopeful results are for prostate cancer. Although three careful studies found no benefit, eight others linked statin use to a reduced risk of prostate cancer. The possible benefits ranged from a 10% reduction in the overall risk of prostate cancer to a 76% reduction in the risk of aggressive prostate cancer."
- Reduced risk of cataracts, macular degeneration, rheumatoid arthritis, gall bladder disease, vitamin D deficiency and gastrointestinal bleeding due to alcoholic liver cirrhosis are some other benefits of statins.

This list of amazing claims comes from a Harvard Medical School Newsletter that provided no references or information about the author(s) or possible conflicts of interest. Supportive meta-analyses are cited to provide a patina of authority, but the preponderance of publications with opposing views are dismissed with caveats such as "others do not agree" or "more research is

needed." It would take too long to discuss all the errors of commission and omission, some of the most egregious errors that have been highlighted, but those familiar with the literature and past Newsletters will cringe at claims that statins have no cognitive side effects and may protect against Alzheimer's. There are numerous reports of amnesia and personality changes and books have been written about this as well as the likelihood that statins can cause dementia. Statin depletion of Coenzyme Q10 is believed to be partly responsible for the progressive rise in congestive failure, which mirrors its increased use. Q10 supplementation can prevent or reverse congestive failure and some statin trials now exclude patients with signs or symptoms of this disorder. Some studies suggest that statins could contribute to or worsen COPD, and as will be seen, a high cholesterol and liporoteins increase resistance to infections. One study that followed over 100,000 healthy people for 15 years found that those with low cholesterol at the start had significantly more hospital admissions for infectious diseases, including influenza and pneumonia. Low cholesterol also increases HIV and AIDS mortality due to postoperative infections and sepsis. Kidney failure is another complication of statin therapy in patients who develop muscle disease and especially rhabdomyolysis. Claims that statins prevent or are effective in treating cancer are dangerously deceptive since it is more likely that the reverse is true and that contrary information has been suppressed.

Although Statins Could Cause Cancer, Few Journals Will Publish This

The argument for using statins to prevent or treat cancer that is presented is not only weak, but also blatantly biased, because it completely ignores the wealth of evidence that the reverse is true. **All statins are carcinogenic** in animal studies using dosages that produced blood levels comparable to those seen in patients taking statins. If statins prevent cancer, then **why are cancer patients excluded from statin trials?** Statin enthusiasts claim that meta-analyses of numerous statin studies fail to show any increased risk of cancer, but this is not surprising, nor is it true. It is not surprising, since the vast majority of these only have relatively short follow-ups that would also have failed to find a link between smoking and lung cancer. This can take decades to detect, in contrast to skin and breast tumors that surface much sooner. In that regard, consider the following:

- In trials with Zocor, which is now available in the U.K. without a prescription, there was an increase in non-melanoma skin cancers.
- A Pravachol study reported that **12 of 286 women developed breast cancer compared to only 1 of 290 in the placebo control group.** The majority of these were recurrences.
- Another Pravachol study of elderly people, who are at higher risk for malignancies, found an increase in cancer after only 1 year, which progressively increased and was statistically significant in 4 years.

- Lymphoid malignancy patients on statins had a 13.3% rate of cancer compared to 7.3% of matched controls with non-malignant diseases.
- In 1,261 patients who had undergone radical prostatectomy, those on statins were more likely to have elevated blood tests indicating a recurrence, and also more aggressive cancers than non-statin controls.
- Two statin studies similarly showed a higher incidence of prostate cancer compared to controls, which increased with longer follow-up.
- In patients with bladder cancer, the tumor became more aggressive in 53% of those who took statins, in contrast to only 8% of non-users.
- A very recent study reported that taking statins for three years was associated with a 54% increase in pre-cancerous colon adenomas.
- Numerous studies show that low cholesterol alone is a significant risk factor for cancer, and the goal of statin therapy is to lower cholesterol and especially LDL, to the lowest possible levels.

Uffe Ravnskov, Kilmer McCully and I expanded on the above and also discussed possible mechanisms of action that might explain these and other adverse statin and/or low cholesterol effects. All our statements were supported by over 40 references from peer-reviewed publications, and although this is a potential public health problem for 40-60 million, these are the responses received when we submitted it in succession to six journals.

1. *Archives of Internal Medicine*: I regret to inform you that its priority rating is not sufficiently high to warrant our considering it further for publication. Based on our initial review, we will not be sending the paper for additional outside editorial review.

2. *CA: A Cancer Journal for Clinicians*: Thank you for submitting your proposal for an article on "Low cholesterol, cancer and the role of lipoproteins" to CA: A Cancer Journal for Clinicians. It is our editorial policy to concentrate on articles that address cancer more broadly (treatment modalities used for many cancer types, current treatment of common types of cancer, public health issues relevant to several cancer types, etc.). For these reasons, we cannot consider your article for publication in CA. However, you may want to consider submitting your article to CANCER, another peer-reviewed American Cancer Society journal, which publishes more focused papers such as the one you have described.

3. *Cancer*: Thank you for your recent manuscript submission of "Low cholesterol, cancer and the role of lipoproteins" (CNCR-11-2485) to Cancer. Your paper has undergone initial review. I am sorry to report that it was not deemed to be of broad enough interest to our readership to merit further evaluation.

4. *Journal of the American Medical Association*: Thank you for your inquiry. However, JAMA is not able to consider your manuscript for publication.

5. *Journal of the National Cancer Institute*: I am sorry that we shall not be able to use the above-titled manuscript. After careful evaluation, the Editorial Board did not accord it a priority sufficient for further consideration.

6. *Scandinavian Cardiovascular Journal*: Thank you for submitting the manuscript # SCAR-2011-0151 entitled "Low cholesterol, cancer and the crucial role of lipoproteins" to the *Scandinavian Cardiovascular Journal*. The questions raised are important, indeed, and deserve a thorough analysis and discussion. Admittedly not being an expert on this field, my impression is that the present manuscript is polemic in style, and biased. This view was shared by one leading cancer epidemiologist; he/she finds the present selection and interpretation of the literature superficial and subjective. Hence I choose not to forward your manuscript to our reviewers.

We retitled our paper **"The statin-low cholesterol-cancer conundrum"** and it was published earlier this month in the *Quarterly Journal Of Medicine*, a monthly and highly respected British journal that "publishes peer-reviewed articles which promote medical science and practice." As can be seen, it is almost impossible to get anything critical of the lipid hypothesis or which disputes the efficacy and safety of statins published in the U.S., because journals do not want to jeopardize the lucrative advertising and reprint revenues from manufacturers of cholesterol lowering drugs and low fat foods. The *Scandinavian Cardiovascular Journal* rejection was somewhat surprising, since they had published several similar critical papers submitted by others and us in the past. I strongly suspect that the reviewer who accused us of being biased and argumentative had strong drug company ties. Last month's issue actually featured an invited editorial I co-authored with three distinguished Swedish scientists entitled **"The Cholesterol hypothesis: Time for the obituary?**" It presented what we considered to be indisputable evidence, if not proof, that statins provided no preventive or other benefits to healthy people, that clinical trials in patients with coronary disease showed no reduction in total mortality, and that serious side effects had been suppressed or ignored in company sponsored clinical trials. It concluded **"In summary, we have now an overwhelming amount of scientific data that falsify the cholesterol myth. So, it is time to say goodbye to this old, ill-founded and fallacious lipid hypothesis."** With respect to being "polemic" and prejudiced, much more could have been added to these two papers. Both have been attached so you can judge for yourself. Neither referred to other safety problems, such as growing concerns that **statins cause diabetes,** a risk factor for coronary disease, **as well as a fatal neurological disorder similar to amyotrophic lateral sclerosis**. Peripheral neuropathy can surface within days of starting statins, and biopsy proven muscle disease may occur without abnormal blood tests used to detect this. In addition, over 90% of adverse drug side effects, especially from statins, are either not recognized or reported to the FDA.

When Should Statins Be Prescribed And What Determines Optimal Dosage?

Passionate proponents of the theory that lowering cholesterol prevents coronary atherosclerosis contend that statins are so safe and provide so many other benefits, that they should be added to the drinking water or given daily to everyone over 55. Equally staunch and stubborn statin skeptics maintain they should be banned because they can be dangerous at any dose. Discussions designed to find some compromise or common ground generates more heat than light because of the fervent and sometimes furious feelings on both sides. As my mentor, collaborator and close friend Hans Selye often reminded me, theories don't have to be correct, only facts do. Faulty theories are often valuable for their heuristic merit, in that they stimulate others to find new facts that then lead to better theories. (This turned out to be prophetic, since it proved true for some of Selye's theories.)

From my perspective, the following observations are particularly pertinent, and have been confirmed so many times they may be considered to be facts.

- Lowering cholesterol or LDL does not prevent coronary atherosclerosis or heart attacks.
- Statins do reduce the incidence of coronary deaths and future events in patients with proven heart disease.
- This cardioprotective effect is not due to lowering cholesterol or LDL, since it occurs regardless of lipid levels, does not correlate with their degree of reduction and there is no dose-response relationship.
- Statins do not lower total death rates in people without heart disease.
- Statin side effects are more numerous and serious than generally appreciated and their long-term consequences are unknown.

Based on these facts, current statin therapy guidelines, which mandate continued administration until LDL is lowered to an arbitrary level of 70 mg/dl, should be abolished. Few can achieve this goal, which means that statins will be given in increasing doses for longer periods of time, or perpetually. That's great for drug companies, but will only insure a higher incidence of adverse side effects and complications for patients. It has been suggested that the benefits of statins are due to their "anti-inflammatory" effects, and that measuring markers of inflammation such as C-reactive protein (CRP), would be preferable. There are not only no studies to support this, but Vioxx, a powerful anti-inflammatory drug, was taken off the market because it was associated with an increased incidence of heart attacks. And there is no such thing as "good" HDL or "bad" LDL. Clinical trials of drugs to prevent heart attacks by raising HDL have been halted because of increased rates of heart attacks. As emphasized in the attached, lipoproteins may have unappreciated but powerful effects on the immune system that increase resistance to infections and provide other rewards. This does not negate the possibility that statins could be effective in certain

situations. For example, it has been suggested that statins might benefit patients with early HIV infection because a covering of cholesterol obtained from the host protects the AIDS virus. This protection also makes it difficult to detect as a foreign threat. However, if the patient goes on to develop clinical AIDS with opportunistic infections and Kaposi's sarcoma, statins would now have detrimental effects. Low cholesterol has also been shown to interfere with the efficacy of antiretroviral therapy for AIDS. The bottom line is it will never be possible to accurately determine the indications and contraindications of statins until we know how they work and develop tests that can measure their mechanisms of action and risk for harm much more precisely than methods currently available. It is also likely that the lipid hypothesis will prevail until this has been accomplished. It is unlikely that this will happen very soon since there is just too much money and too many reputations at stake. Consider the following thoughts on this subject:

> All truth passes through three stages. First, it is ridiculed. Second, it is violently opposed. Third, it is accepted as being self-evident.
>
> Arthur Schopenhauer

> A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it.
>
> Max Planck

> The mind of the bigot is like the pupil of the eye; the more light you pour upon it, the more it will contract. Oliver Wendell Holmes Sr.

Nevertheless, eventually, as Shakespeare noted, "The truth will out." Chinks are increasingly appearing in the lipid hypothesis armor as doctors as well as patients are confused about the growing LDL vs. CRP controversy, and questions related to statin safety – so stay tuned to see what happens next!

Paul J. Rosch, MD, FCP
Editor-in-Chief

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PAUL J. ROSCH, M.D., F.A.C.P.
EDITOR-IN-CHIEF
www.stress.org
e-mail: stress124@optonline.net