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STRESS, CHOLESTEROL, FATS & HEART DISEASE

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It's not difficult to understand why most Americans, including physicians, are convinced that high blood cholesterol is the major cause of heart disease and that elevated cholesterol is due to eating saturated fats. It's easy to visualize how fatty foods raise blood cholesterol, which, despite being a large inert molecule, somehow precipitates out to infiltrate the inner lining of the coronary arteries, where they form fatty atheromatous plaques.

Also Included In This Issue

These deposits slow the flow of blood and eventually completely obstruct the artery, resulting in a myocardial infarction. This sequence is so well accepted that the terms coronary occlusion, myocardial infarction and heart attack are often considered to be synonyms and interchangeable. What is hard to believe is that there is no proof to support this entrenched fatty food⇒cholesterol⇒heart attack hypothesis.

In point of fact, it has been completely refuted by numerous scientific studies. Consider the following half dozen examples:

Why Saturated Fat And Cholesterol Do Not Cause Coronary Heart Disease

1. Almost two-dozen studies have reported that coronary heart disease patients ate less or the same amount of saturated fat as healthy controls. The huge World Health Organization project MONICA (Monitoring of Trends and Determinants in Cardiovascular Disease)

that collected data from 21 countries for over 10 years failed to find any correlation between heart attacks and fat consumption or cholesterol. **Every single country with the lowest fat consumption had the highest mortality rates from heart disease and those with the most fat consumption had the lowest.** The French consumed three times as much saturated fat compared to Azerbaijan but had one-eighth the rate of heart disease. The heart disease death rate in Finland was three times greater than in Switzerland, even though the Swiss ate twice as much fat.

2. No dietary cholesterol lowering trial has ever shown a reduction in lowering coronary disease or total mortality. In the "Prudent Diet" study of 49 to 59 year-old men, one group substituted margarine for butter, cold cereal for eggs, and chicken and fish for beef. Controls ate eggs for breakfast and meat three times a day. After ten years, there were eight deaths from heart disease in the low fat diet group, compared to none for the meat eaters. Ancel Keys also fed middle-aged men a very high cholesterol diet but found that their cholesterol levels were no different than a control group who consumed less than half as much. Decades later, he admitted "**There's no connection whatsoever between cholesterol in food and cholesterol in blood**. And we've known that all along. Cholesterol in the diet doesn't matter at all unless you happen to be a chicken or a rabbit."
3. In the Framingham study, which was responsible for establishing cholesterol, hypertension and cigarette smoking as the three major risk factors for coronary heart disease, a 26-year follow-up report found that 50% of cases occurred in people with below average cholesterol. There was a direct association between falling cholesterol levels over the first 14 years of the study and increased mortality rates over the following 18 years. For men above the age of 47, those with low cholesterol had greater mortality rates than those with high cholesterol. Subjects whose cholesterol had decreased spontaneously over 30 years were also at greater risk of dying from heart disease than those whose cholesterol had increased. In addition, **the more saturated fat and the more cholesterol people ate, the lower their serum cholesterol was. Those who ate the most saturated fats weighed the least.**
4. No association between cholesterol levels and the severity or extent of atherosclerosis has ever been found in autopsy studies of the general population. No clinical or imaging study has ever found any relation between the degree of cholesterol lowering and less atherosclerosis. In one angiography study, in which blood cholesterol had been reduced by more than 25% in 24 patients, atherosclerosis was increased in 18 and unchanged in eight. A Mayo Clinic study similarly found that in all patients whose cholesterols had decreased by more than 60 mg., there was a significant increase in coronary atherosclerosis.

5. High cholesterol does not increase risk for heart attacks or coronary events in people over 65, women of any age, as well as patients with diabetes or renal failure. **Senior citizens with high cholesterols have significantly fewer infections and live longer than low cholesterol controls.** In familial hypercholesterolemia, there is no association between the very high cholesterol and LDL levels and a corresponding increased incidence or prevalence of coronary disease.
6. The huge and lengthy MRFIT study (Multiple Risk Factor Intervention Trial) was designed to prove the links between diet, cholesterol, and other risk factors with heart disease. Cholesterol consumption was cut by 42 percent and saturated fat consumption by 28 percent. While those on this diet 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.** In most other studies, the incidence of stroke was also higher in those who ate less saturated fat.

Why Has The Cholesterol→Coronary Disease Dogma Persisted?

It must seem curious to some that the lipid hypothesis is still regarded as gospel by physicians despite overwhelming proof that it is flawed, if not totally fallacious. For example, a scholarly review published in *Science* several years ago showed that cholesterol levels up to 240 were normal and that **women with a cholesterol over 240 had a decreased risk for coronary deaths.** Belief in this lipid doctrine was originally based on experiments in which cholesterol was force fed to animals that do not eat meat, and the results are not relevant to humans. Ancel Keys subsequently demonstrated a correlation between fat intake, cholesterol and heart disease deaths in seven countries. This was so convincing that his views and suggestions became official NIH policy and a massive campaign to lower cholesterol was launched in 1961. The problem was that while Keys had 22 countries to choose from, he "cherry picked" only those that had both high fat intake and coronary death rates to support his theories. Had he analyzed the data on all countries, he would have come to a very different conclusion. For example, Mexico, which had the same high fat consumption as Finland, was not included because coronary deaths were seven times higher in Finland. **Also omitted were Sweden, Germany, France and Israel, where the higher the saturated fat and cholesterol intake, the lower the incidence of coronary deaths.**

This demonization of fats was magnified and perpetuated by the cholesterol cartel of manufacturers of cholesterol lowering products, low fat foods, lipid testing equipment and others who stood to reap huge profits. It reached its peak with the advent of statins, which quickly became and remain the widest

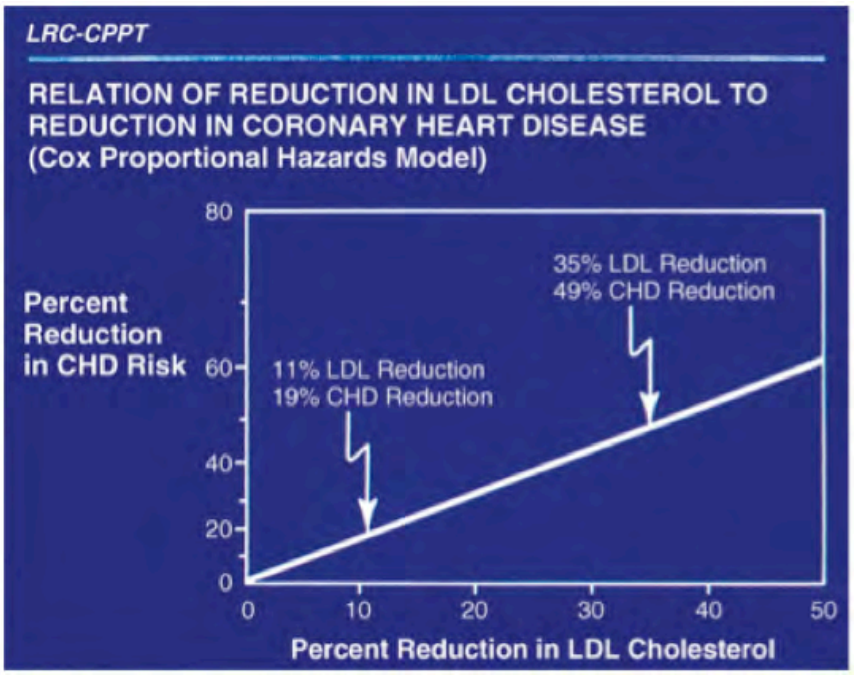
selling and most profitable drugs ever. Pharmaceutical company efforts were aided and abetted by the FDA, Congress and other regulatory agencies, prestigious academic institutions, organizations and physicians, all of whom were handsomely reimbursed for their support and endorsements. Medical journals as well as the lay media were reluctant to publish anything that promoted a contrary view for fear of losing lucrative advertising revenue. Any opponents were viciously persecuted, as happened with Kilmer McCully, who demonstrated that lowering homocysteine might be more important than lowering cholesterol to prevent heart attacks, and was not only much safer than statins, but only cost pennies a day rather than dollars.

Like a deep earlobe crease, pot belly, premature vertex baldness and hundreds of other items, people with elevated cholesterols are at greater risk for heart attacks. However, that does not mean that plastic surgery, liposuction or a hair transplant will make this less likely, and the same holds true for cholesterol. **Association never proves causation.** It would be much more accurate to refer to these as risk markers that are merely statistical associations due to genetic factors, rather than risk factors that have a causal relationship. To get around this, statins are advertised as reducing the risk of heart attacks by implying that lowering cholesterol will result in a corresponding reduction in heart attack rates. What the ads don't say is that this is relative risk, not absolute risk, which is quite different. Nor do they indicate how many people need to be treated for one person to benefit. For example, your doctor tells you that there is a new statin drug with no side effects and that one study showed that if you take it every day for the next five years your risk of heart attack will be reduced by **34%**, but this is relative risk. What you are not told is that after five years, 2.7% of patients taking this drug had a heart attack compared to 4.1% taking a placebo, an **absolute risk reduction of only 1.4%**, which is much less attractive. Nor are you told that 71 people would have to take this every day for five years to prevent one person from having a heart attack, but it is not known if that one person will be you. In addition, all the people in this study were at increased risk because of elevated cholesterols, and there is no evidence that statins would benefit anyone with a normal cholesterol.

One of the earliest attempts to deceive the public by using relative risk was the NIH sponsored Coronary Primary Prevention Trial, which claimed that taking cholestyramine (Questran) would lower cholesterol by 28% and result in a 50% reduction in major coronary events after seven years. Bile acids, which are made from cholesterol, aid in the digestion of fats in the small intestine. Cholestyramine binds to bile acids and since the drug is not absorbed, it and the bile acids are excreted, and more cholesterol has to be extracted from the blood to replenish the supply. After three years of screening several hundred thousand individuals, for elevated cholesterol, 3,800 men in the 40 to 60 heart attack prone age group with very high

cholesterols were recruited to participate in the study. Although the 50% drop in cholesterol was not achieved, researchers triumphantly reported that there was a 19% reduction in the study group, as illustrated below.

The Cholestyramine Coronary Primary Prevention Trial



But **19%** was a **relative risk reduction** from lowering LDL cholesterol. The **actual risk reduction** was **1.1%** for all coronary events, and for fatal heart attacks, it was only **0.6%.** The graph to the left just shows the reduction in relative risk of coronary disease that was predicted to be associated with a progressive lowering of LDL. **Every 1% drop in LDL there would result in almost a 2% reduction in heart disease initially**. But as can be seen, a 35% drop in LDL only reduced CHD risk 49%, not 70%. And a 30-year Framingham follow-up found that for each 1% drop in cholesterol, there was an 11% increase in coronary and total mortality.

In addition, the cholestyramine study dealt only with primary hypercholesterolemia, a lipid disorder affecting less than one in five hundred. It was uncertain that lowering cholesterol would be beneficial for men in other age groups or women of any age, or whether cholestyramine would be safe, or even tolerated. Some men stopped taking the foul tasting 4 to 5 packets of cholestyramine after a few days, many complained of severe constipation and other gastrointestinal complaints due to the lack of bile acids. Most were unable to take the full 24 grams daily, so that relatively few stayed on the required regimen for seven and a half years. With respect to safety, cholestyramine interferes with the absorption of fat soluble vitamins and numerous common drugs, including Coumadin, Digoxin, Inderal phenobarbital, thiazide diuretics and thyroid. And little was mentioned about the 21 cases and 8 deaths from gastrointestinal cancer in those taking the drug, compared to 11 cases and only 1 death in the control group.

Small wonder that statins seemed to be a much better option, although their significant side effects have also been skillfully concealed. Perhaps the most egregious abuses have come from statin ads, especially on TV. These consistently cite relative risk reduction statistics from a specific demographic group, such as middle-aged men who have had a heart attack, and imply that they will prevent heart attacks in men of any age or women.

Advertisements were initially required to state that "Statins have not been shown to prevent heart attacks", and even though this was in tiny print that was difficult to read, drug companies successfully campaigned to have this removed. Statins do not provide benefits in senior citizens or women of any age and several class action suits have been filed for false advertising by those who have suffered significant side effects. More importantly, it is increasingly clear from JUPITER and other studies that **statin benefits are unrelated to their cholesterol lowering effects and are more likely due to anti-inflammatory or antithrombotic effects.** As I pointed out almost a decade ago in the *Journal of The American Medical Association,* "It might be advisable to find the minimum statin dosage that provides cardioprotection. As with aspirin, this may be much lower than for other indications." The important lesson to learn from all of the above is that association never proves causation. For example, the Japanese eat very little fat and suffer fewer heart attacks than the British or Americans. The French eat a lot of saturated fat and also suffer fewer heart attacks than the British or Americans. The Japanese drink very little red wine and suffer fewer heart attacks than the British or Americans. The Italians and French drink excessive amounts of red wine and also suffer fewer heart attacks than the British or Americans. The Germans consume huge quantities of beer, eat lots of sausages and fats and suffer fewer heart attacks than the British or Americans. **Conclusion**: **Eat and drink whatever you like. Speaking English is apparently what causes heart attacks**.

Why Stress Is Much More Important Than Cholesterol Or LDL

The appreciation that emotional factors can have a powerful influence on the heart, and the acknowledgment of some intimate, although poorly understood, heart-mind connection, is certainly not new. Aristotle, and later Virgil, actually taught that the heart rather than the brain was the seat of the mind as well as the soul, and a similar belief can be found in ancient Hindu scriptures and other Eastern philosophies. Almost 2000 years ago, Celsus commented on this mind-heart relationship, noting that “fear and anger, and any other state of the mind may often be apt to excite the pulse.” Our earliest uses of the word clearly indicate its conceptualization as the seat of one’s inmost feelings, temperament, or character. Broken hearted, heartache, take to heart, eat your heart out, heart of gold, heart of stone, stouthearted, are words and phrases we still use to vividly symbolize such beliefs. William Harvey also recognized that the heart was much more than an automatic pump, when he noted in 1628, "every affection of the mind that is attended either with pain or pleasure, hope or fear, is the cause of an agitation whose influence extends to the heart." John Hunter, who during the 18th century elevated surgery from a mechanical trade to an experimental science, suffered from angina, and being a keen observer complained, "my life is in the hands of any rascal who chooses to annoy and

tease me." He turned out to be somewhat of a prophet, since in fact an argument did precipitate his death from a heart attack.

Napoleon's favorite physician, Corvisart, wrote that heart disease was due to the "passions of the mind", among which he included anger, madness, fear, jealousy, terror, love, despair, joy, avarice, stupidity, and ambition. One hundred and thirty years ago, von Dusch, a German physician, first called attention to the fact that excessive involvement in work and similar types of behavioral patterns appeared to be the hallmark of people who developed coronary heart disease. Toward the end of the 19th century, Sir William Osler, an astute clinician, succinctly but accurately de- scribed the coronary-prone individual as a "keen, and ambitious man, the indicator of whose engines are set at 'full speed ahead.'" In the 1930s, the Menningers suggested that coronary heart patients tended to have strongly aggressive behavior, and a decade later, Flanders Dunbar, who introduced the term "psychosomatic" into American medicine, characterized such individuals as being authoritarian with an intense drive to achieve unrealistic goals. Fierce ambition and compulsiveness to achieve power and prestige were emphasized by many subsequent investigators, and over 50 years ago, the rising incidence of coronary heart disease in England was attributed to increased stress, especially in the workplace.

All of these 19th and 20th century physicians were describing various aspects of what is now called Type A behavior, a term coined by Meyer Friedman and Ray Rosenman over 50 years ago. Type A individuals are apt to be very competitive and are usually in a hurry, so they eat, talk, and do most other activities as quickly as possible. They generally try to do too many things at the same time, are frequently concerned with what they are going to do next, and are often so preoccupied with work that they tend to have few other interests. Around the same time, Stewart Wolf independently but noted that coronary disease was often due to the constant striving to achieve unrealistic goals, adding that even when successful, such individuals were unable to relax and enjoy the satisfaction of their labors. He called this the "*Sisyphus Syndrome*", since in Greek mythology, Sisyphus had been condemned to roll a huge boulder up a hill, which, as soon as it reached the top, always rolled down to the bottom, and he repeatedly was forced to start all over again. Type A was acknowledged by the NIH to be as powerful a risk factor for heart attacks as cholesterol or anything else. In that regard, the large MRFIT study designed to demonstrate that lowering the standard risk factors of cholesterol, hypertension and cigarette smoking failed to show any reduction in heart attacks or coronary deaths in those who achieved these goals. In contrast, two other intervention trials conducted during this same period were so successful that they had to be halted prematurely so controls would not be denied their benefits. One was a trial designed to modify and

lessen Type A traits and the other was administration of propanolol (Inderal) a drug that blocks the damaging effects of stress hormones.

The Pathophysiology Of Stress Induced Coronary Heart Disease

Attempts to study the mechanisms whereby emotional states could produce cardiovascular damage and sudden death received tremendous impetus as a result of the investigations of Walter Cannon at Harvard University in the early part of the last century. Cannon' s studies showed that the response to the stress of acute fear resulted in a marked increase in sympathetic nervous system activity and an outpouring of adrenaline, which assisted the animal in life saving "fight or flight" activities. His subsequent studies of voodoo deaths and lethal spells due to "bone pointing" by witch doctors in primitive societies also implicated a flooding of the system with adrenaline as the most likely cause of a fatal arrhythmia. In the late 1940s, Hans Selye's formulation of his General Adaptation Syndrome, and Diseases of Adaptation provided further insights into stress induced heart disease in animals that he believed also applied to humans. Selye's subsequent research in the 1950s included the experimental production of cardiac necrosis due to stress, in which direct biochemical injury to heart muscle rather than occlusion of a coronary vessel was the most important factor. He demonstrated the important influence of sodium, potassium, magnesium, and calcium in modulating this response. His results were subsequently corroborated in humans and now form the basis of various therapeutic strategies and pharmacologic approaches.

Selye's emphasis on the pituitary-adrenal cortex dominated stress research in the 1950s and 1960s, but subsequent advances led to a recognition that human responses to stress in humans involved a vast repertoire of hormonal secretions including other pituitary and target gland hormones, the renin-angiotensin system, prostaglandins, and brain peptides such as serotonin, dopamine, melatonin, prolactin, and the endorphins. Attention was also being focused on central nervous system mechanisms that initiate and transmit the stress signal, with evidence suggesting that both heart rhythm and force of contraction are regulated by the same centers in the frontal cortex of the brain that stimulate sensory receptors during acute fear. Experimental animals that would normally succumb to ventricular fibrillation due to severe psychological stress are protected if the nerve pathways from the frontal cortex to the brain are cut or temporarily blocked by freezing.

As indicated, the terms coronary occlusion and myocardial infarction are still used as synonyms by those who continue to view this sequence of events as the cause of all heart attacks. This concept was widely accepted because of James Herrick's 1912 autopsy studies showing that a myocardial infarction was due to occlusion of a coronary artery by a clot or thrombus. We now recognize that myocardial infarction can occur in the absence of significant

coronary atherosclerosis or obstruction due to the excessive release of norepinephrine at myocardial nerve endings. This produces a distinctive "contraction band" necrosis under the microscope that is identical in animals humans following some severely stressful incident, but without the marked white cell infiltration and inflammation usually present in most other myocardial infarcts. Conversely, it is not unusual to find extensive and even obstructive coronary atherosclerosis in elderly patients who have never had any signs or symptoms of heart disease, and died following an accident or some other cause unrelated to the cardiovascular system.

A half dozen of the numerous other mechanisms that link stress to coronary heart disease include:

1. Stress also contributes to other risk factors such as smoking, hypertension, diabetes and obesity. For those who still believe in elevated cholesterol, stress has a much greater effect than fatty food intake, as demonstrated in tax accountants as April 15 approaches, and students on the eve of an important exam.
2. Stress can cause constriction of the coronary vasculature and increased platelet stickiness and clumping that promote clot formation.
3. Stress increases homocysteine, CRP and fibrinogen, all of which are risk factors or risk markers for coronary heart disease.
4. Stress causes increased deep abdominal fat deposits that contribute to insulin resistance and metabolic syndrome with its varied and sometimes deadly cardiovascular consequences.
5. In addition to Type A behavior, hostility, excessive anger, stressful life events, depression, and anxiety, have all been demonstrated to cause coronary heart disease.
6. It has been proposed that unstable atherosclerotic plaque might actually represent a "microabscess" that resulted from an infection. There is surprising support for this theory, and it is well established that stress can increase susceptibility to infections.

There is much more that could be said about all of the above. With respect to stressful life events, one has only to look at the startling statistics showing that senior citizens have a 20 percent chance of dying, usually from a heart attack, in the 12-18 months after the loss of a spouse. During the month following the 9/11/2001 terrorist attacks on the World Trade Center in New York, the rate of defibrillator firings was two to three times normal even in patients living far from the catastrophe. In one study of work-related stressors, upcoming deadlines were associated with a six fold increase in myocardial infarction, and other studies suggest that chronic work-related stress could carry a two to three times higher rate of coronary events, especially when employees perceive little control over their work environment. In women with established coronary disease, marital stress was associated with a risk of recurrent events three times higher than in

controls with little marital discord. Caring for a sick spouse at home nearly doubles heart disease death rates. There is also stress cardiomyopathy or "Broken Heart Syndrome" in which middle-aged or older individuals are admitted with severe chest pain and ECG changes suggestive of an impending massive infarction, but who have no angiographic abnormalities or enzyme changes indicating muscle damage. This usually follows some acutely stressful event that results in myocardial "stunning" from increased stress hormones, and most patients recover spontaneously within 72 hours.

Uffe Ravnskov and Kilmer McCully propose that atherosclerosis and unstable plaque may be due to infection rather than cholesterol deposits. They point out that lipoproteins are part of a nonspecific immune defense system that binds and inactivates microbes and their toxins by the formation of complexes. This would explain why vulnerable plaque contains lipids and microbes in the arterial wall, why neutrophils are seen in the myocardium following an infarct, as well as the frequent occurrence of fever, diaphoresis, elevated inflammatory markers and even bacteremia in acute myocardial infarction. There is considerable supportive evidence. The presence of cholesterol in human atheroma was first described in 1856 by Rudolph Virchow. He termed it *endarteritis deformans* to emphasize it resulted from an inflammatory process that injured the intimal lining of arteries, as follows

> We cannot help regarding the process as one which has arisen out of irritation of the parts stimulating them to new, formative actions; so far therefore it comes under our ideas of inflammation, or at least of those processes which are extremely nearly allied to inflammation.

In other words, atherosclerotic plaque in humans was a response to injury or inflammation. The cholesterol deposits came later. A variety of microbes have been identified in atherosclerotic plaque and *Chlamydia pneumoniae* has been cultured from the atherosclerotic plaque of patients with unstable angina. This organism is a fairly harmless pathogen that causes a mild flu like illness, but unlike most other bacteria, it lives not on the surface of cells but within them, and is frequently found in the coronary arteries of heart disease patients, but not others. While this might not produce any signs or symptoms, it can continue to stimulate immune system components designed to attack foreign microorganisms. These chemicals are attracted to sites on the inner lining of blood vessels where *Chlamydia pneumoniae* is frequently located, which can produce a smoldering, low-grade inflammatory response that results in the steady growth of plaque. One study reported that a protein on chlamydia cell walls was almost identical to one found in heart tissue and suggested that when the immune system attacks chlamydia, it accidentally attacks the heart protein as well. In experimental animals, the resultant microscopic changes are very similar to those seen in human myocardial infarctions. Most people probably harbor chlamydia, as well as *Helicobacter pylori,* which causes peptic ulcer and

many with the tubercle bacillus also have no signs or symptoms of their presence. It is only when stress reduces resistance to these pathogens that clinical disease surfaces.

Numerous stress studies have demonstrated that depression significantly increases risk for coronary events, congestive failure and premature death following a heart attack. Some of the reasons for this include: an increase in sympathetic tone as evidenced by elevated levels of circulating catecholamines and reduced heart rate variability; elevated levels of cortisol, free fatty acids and inflammatory cytokines that promote atherosclerosis; elevated levels of platelet factor 4 and beta-thromboglobulin that promote platelet aggregation that leads to clots and thrombi. Prinzmetal's or variant angina, is characterized by severe chest pain occurring at rest. It is often seen in young women and is caused by vasospasm of the coronary arteries, rather than atherosclerotic interference with blood flow. While not associated with any acute stressful event, it is much more common in depressed patients and may be related to increased platelet aggregation. Depressed patients may also be less likely to exercise or eat well, or to adhere to medication or other therapies designed to prevent heart disease.

Recent research has also started to focus on anxiety. An article in the May 2010 issue of the *American Heart Journal* that reviewed the medical records of 97,000 U.S. veterans reported that "When the researchers accounted for a number of other factors related to heart attack risk, **people with anxiety disorders were anywhere from 25 percent to 43 percent more likely to suffer a heart attack than those with no anxiety disorders**." Two papers in the June issue of the *Journal of the American College of Cardiology*, also firmly established anxiety as an independent predictor for future coronary heart disease. The first was a meta-analysis on the association of anxiety with coronary heart disease in initially healthy people using data from the US, Europe, and Asia. Even after adjustment for other influences, **anxious people had a 25% greater risk of coronary disease and an almost 50% higher risk of cardiac death over a mean follow-up period of 11.2 years**. In the second study, 50,000 Swedish men who were thoroughly examined for military service were followed for an average of 37 years. Sweden has universal health coverage and maintains detailed ongoing records, and all the men classified as having anxiety or depression had been diagnosed by a psychiatrist. Although depression was not a predictor for subsequent coronary disease as in most studies, **those having an anxiety disorder, were twice as likely to suffer from coronary disease or to have had a myocardial infarction**. No females were included, but prior studies have shown that women are much more likely than men to suffer from anxiety and depression. An accompanying editorial emphasized that these new risk factors for coronary disease "need to be carefully scrutinized for clinical utility….Anxiety disorders are as prevalent as

hypertension and are a major affliction of the young, with a lifetime prevalence of around 28%, and when anxiety coexists with depression, the corresponding impact on quality of life is even worse." The editorial also noted "Physicians are frequently timid about assessing emotional symptoms. It is odd that we thread catheters, ablate lesions, and give rectal exams but are uncomfortable asking patients about their lives."

Despite this growing confirmation of the important role of anxiety, depression and stress in the etiology and course of coronary heart disease, and proof that the lipid hypothesis is erroneous, little is likely to change for several reasons. The first is that we tend to believe something when it is repeated more than three or four times, especially by different sources. As William James noted, "There's nothing so absurd that if you repeat it often enough, people will believe it." Lenin and Hitler knew that "A lie told often enough becomes truth" and the cholesterol cartel has capitalized on this in their promotional campaigns. One example is the Jarvik fiasco, in which the creator of an artificial heart, that never worked, is portrayed as a caring and authoritative cardiologist in superb physical condition who takes Lipitor because it is superior to other prescription and generic statins. He is also shown jogging with his son and skillfully sculling on a sunny and serene mountain lake. The facts are that Dr. Robert K. Jarvik does not treat patients since he is not licensed to practice medicine, does not know how to scull and doesn't take Lipitor. The shots demonstrating his sculling expertise were of an athletic, late middle-aged accomplished rower who resembled him. The close up frames that actually showed Dr. Jarvik were taken while he was in a rowing apparatus on an elevated platform to conceal that it was on dry land. Nevertheless, wearing a white coat with a stethoscope draped around it, Jarvik tells viewers that Lipitor can lower "bad" cholesterol by up to 60% to achieve a "36% reduction in heart attacks*". "I'm glad I take Lipitor, as a doctor and as a dad," he says, before a final shot shows his double rowing with vigorous, muscular strokes in the distance.

When *Consumer Reports* showed the Jarvik ad to almost 1,000 patients who had been advised by their physicians to lower their cholesterol, they received the following reactions:

- Sixty-five percent said the ad conveyed that leading doctors prefer Lipitor.
- Forty-eight percent said Dr. Jarvik’s endorsement made them more confident about Lipitor.
- Twenty-nine percent had the definite impression from the ad that Dr. Jarvik sees patients regularly.
- Thirty-three percent of those taking another prescription statin said they were likely to speak to their physician about switching to Lipitor.

- Forty-one percent said the ad conveyed that Lipitor is better than generic alternatives. (In fact, the vast majority of people taking statins can get the same results from a generic for less than half the cost.)
- Over 90 percent believed that the ad was credible and accurate.

The message for most was that Lipitor could reduce heart attacks in more than one out of three healthy people, regardless of their cholesterol. Few paid any attention to the asterisk after the claim that Lipitor resulted in a "36% reduction in heart attacks*". It referred to a statement in tiny print at the bottom of the screen explaining that there were 2 heart attacks out of 100 patients on Lipitor, compared to 3 heart attacks for controls taking a placebo. Thus, there was **only a 1% actual risk for those with "multiple risk factors for heart disease" who took Lipitor daily for over a decade. How many people would take Lipitor if they knew that its likelihood of preventing a heart attack was one in 100 if they took it for over ten years?** And this is only for those at high risk.

It may be true that "figures don't lie", but as demonstrated in the Ancel Keys and cholestyramine reports, "liars can figure." Disraeli wrote that "There are three kinds of lies: lies, damned lies and statistics." As someone wisely said, "Statistics are a highly logical and precise method for stating a half-truth inaccurately." The First Law of Statistics is that if the statistics don't support your view, you need more statistics. The Second Law is that given enough statistics, you can prove anything." Statin makers have taken advantage of this by confusing the public with deceptive relative risk statistics that have little significance, illustrating Harry Truman's contention that, "if you can't convince them, confuse them". In contrast to cholesterol, stress is difficult to define, much less measure, so that research demonstrating how it can contribute to heart disease is apt to be much less convincing than company sponsored statin study statistics. Nevertheless, it would be wise to remember Albert Einstein's advice that "**Not everything that can be counted counts, and not everything that counts can be counted**."

Paul J. Rosch. MD, FACP
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