

HEALTH AND STRESS

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FATS, CORONARY DISEASE, LDL, STATIN SIDE EFFECTS & Co Q10

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Regular readers of this Newsletter will recall that prior issues have emphasized that although statins are useful drugs, current prescribing recommendations are not only inappropriate but may also be dangerous. This problem stems from the mistaken belief that elevated cholesterol and/or LDL are the prime cause of coronary heart disease. A corollary of this is that a high fat diet causes elevated cholesterol levels. Unfortunately, powerful vested interests have been successful in perpetuating these myths which are still accepted by the public and most physicians as dogma.

Drug companies, low fat food manufacturers and other constituents of the cholesterol cartel have been able to accomplish this by spending hundreds of millions of dollars on direct media advertisements and promotions. They also have considerable covert

control over Congress, the FDA and other regulatory agencies as well as influential medical institutions, journals and physicians.

Over the past six months, several research reports and studies have provided strong support that debunks these dietary fat → elevated cholesterol → coronary heart disease myths. These confirm our prior allegations that:

Σ **Diets high in meat, dairy products or natural fatty acids do not increase serum cholesterol nor do they contribute to coronary heart disease or obesity.**

Σ **Elevated serum cholesterol, LDL and other lipid components are not the cause of heart attacks.** They are statistically linked with an increased incidence of coronary events as are premature baldness, deep earlobe creases, abdominal obesity and hundreds of other items. **These are simply associated risk markers rather than causative risk factors.**

Σ Most heart attacks are not caused by obstructive atherosclerosis that occludes a coronary artery but rather rupture of a piece of plaque.

Σ Statins reduce coronary morbidity and mortality because of their anti-inflammatory activities rather than by reducing blood lipids.

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- Σ The goal of statin therapy is to lower LDL to an arbitrary level that is often difficult to achieve. This end point is not only inappropriate but also dangerous, since it leads to continually increasing the dosage of the drug and a higher incidence of adverse effects.
- Σ Statin side effects are more numerous and severe than previously suspected and many may be due to depletion of Coenzyme Q10.

Could A Diet High In Fats Be Healthy?

In his *Diet Revolution* introduced 30 years ago, Dr. Robert Atkins stated that if people avoided carbohydrates they could eat as much fat and protein as they wanted to and still lose weight. He provided numerous examples of success in patients unable to lose weight with caloric restriction or other regimens and sold millions of books. However, nutritionists and cardiologists were not only skeptical, but denounced the Atkins diet as dangerous. They had been campaigning to convince the public that fats, and particularly the saturated fats in meat and dairy products caused high cholesterol, the leading cause of heart disease,

Atkins stated that fats are harmless and that the real coronary culprits are carbohydrates like sugar, pastas, bagels and rice. His book encouraged eating "truly luxurious foods without limit", such as "delicious lobster with butter sauce, juicy steak, bacon cheeseburgers". Starches, refined carbohydrates, sugar, fruit juices and anything made from flour were prohibited but moderate amounts of vegetables were allowed. Many who were lured by the promise of being able to eat all the steak, lobster and dairy products they wanted to and still lose weight did not realize that it was really avoiding carbohydrates that mattered.

Those who faithfully adhered to this admonition found that the pounds kept falling off. Atkins' huge following caused considerable concern and his diet was derided by the NIH, American Heart Association, American Dietetic Association and most establishment organizations. Others like Nathan Pritikin and Dean Ornish, who recommended avoiding all fat like the

plague, also attacked it as being ineffective, unhealthy and a sure prescription for a heart attack.

At the annual meeting of the American Heart Association last November, Duke researchers reported on their study of 120 overweight volunteers who were randomly assigned to either the Atkins diet or the Heart Association's widely used Step One low fat diet. Those on the Atkins diet were limited to less than 20g of carbohydrates daily with 60% of their calories coming from fat. At the end of the six-month study, the Atkins diet group had lost an average of 31 pounds compared to 20 pounds on the Step One diet. Total cholesterol fell to a small degree in all participants but the Atkins group averaged an 11% increase in HDL (good cholesterol) and a whopping 50% drop in triglycerides. There was no change in HDL and only a 22% decline in triglycerides in those on the American Heart Association low fat diet.

This was the third formal study reported during the past year confirming that the Atkins diet resulted in weight loss and had no adverse side effects. There was not only no increased risk of heart attack based on a rise in cholesterol, but lipid changes suggested just the opposite, which generated a great deal of media attention. In response, the Association's spin-doctors stated that reports of the research had created "the erroneous impression that the American Heart Association has revised its dietary guidelines. This is not the case." They emphasized that the study was "very small and short-term" and "did not actually compare the Atkins diet with current AHA dietary recommendations", although these were not terribly different. This might have been expected, and confirms the old adage that **those who do not learn from the mistakes of history are doomed to repeat them.**

"There Is No New Thing Under The Sun"

So states the Bible (*Ecclesiastes* 1:9) and the high fat diet-obesity-heart disease controversy seems to bear this out. **The Atkins diet is hardly new and was utilized 140 years ago** by William Banting, a London carpenter and coffin maker to the rich and famous. Although there was no

family history of obesity he was disturbed about being so overweight in his thirties and asked a physician friend how this problem could be corrected. As Barry Groves explained in a fascinating essay, he was advised to "increase bodily exertion before any ordinary daily labours began". Banting began rowing his heavy boat on a nearby river for two hours a day but all this accomplished was to markedly increase his appetite. He put on so many pounds that he was forced to stop.

He was then told to simply eat "moderate amounts of light food" but was not sure what this meant. This not only produced no weight loss but left him feeling constantly fatigued and "caused many obnoxious boils to appear". Two rather large carbuncles necessitated hospitalization for surgery but during his stay he was fed several times a day to improve his resistance and he became increasingly obese. Over the next two decades "he tried swimming, walking, riding and taking the sea air. He drank gallons of physic and *liquor potassae*, took the spa waters at Leamington, Cheltenham and Harrogate, and tried low-calorie, starvation diets; he took Turkish baths at a rate of up to three a week for a year but lost only 6 pounds in all that time, and had less and less energy." During this period, he tried every slimming treatment that had been suggested without success and had to be hospitalized 20 times. Disillusioned and depressed, he finally decided to give up all attempts to lose weight.

In 1862, at age 66, he was 5' 5", weighed over 200 lbs. and wrote that:

I could not stoop to tie my shoes, so to speak, nor to attend to the little offices humanity requires without considerable pain and difficulty, which only the corpulent can understand. I have been compelled to go downstairs slowly backward to save the jar of increased weight on the knee and ankle joints and have been obliged to puff and blow over every slight exertion, particularly that of going upstairs.

In addition to a large umbilical rupture, his vision was failing and he had become increasingly deaf. He was particularly concerned about his hearing problem but since his doctors had no remedy, he was referred to William Harvey,

a distinguished ENT specialist. Dr. Harvey had recently returned from a symposium in Paris where he had heard the famous Claude Bernard discuss his new theory that the liver manufactured a sugar-like substance (glycogen) from circulating blood elements that influenced the course of diabetes. This had stimulated Harvey to study how fats, sugars and starches affected the body and might lead to other disorders.

He felt that Banting's obesity was contributing to his deafness and told him to give up bread, potatoes and all starches since these tended to create fat. Banting wasn't very happy with this since he thought it gave him little to eat. However, he agreed to give it a try after Harvey explained his meals could include:

- Σ six ounces of bacon, beef, mutton, venison, kidneys, fish or any form of poultry
- Σ the "fruit of any pudding" but not the pastry
- Σ any vegetable except potato
- Σ two or three glasses of good claret, sherry or Madeira but no port or beer
- Σ tea without milk or sugar + one ounce of toast

When he began this diet in August 1862, Banting weighed 202, was down to 184 at Christmas and stated in April that he had not felt this well in the past 20 years or more. He weighed 156 the following August, a loss of almost a pound a week. As Groves notes:

By the end of the year, not only had his hearing been restored, he had much more vitality and he had lost 46 lbs. in weight and 12 1/4 inches off his waist. He suffered no inconvenience whatever from the new diet, was able to come downstairs forward naturally with perfect ease, go upstairs and take exercise freely without the slightest inconvenience, could perform every necessary office for himself, the umbilical rupture was greatly ameliorated and gave him no anxiety, his sight was restored, his hearing improved, his other bodily ailments were ameliorated and passed into the matter of history.

Banting was so delighted that he promptly prepared a small booklet entitled *Letter on Corpulence Addressed to the Public* so that others could benefit from his experience.

Is History Repeating Itself?

Excerpts from this booklet, which appeared in 1863 clearly reflect his enthusiasm:

I can conscientiously assert I never lived so well as under the new plan of dietary, which I should have formerly thought a dangerous, extravagant trespass upon health.

I can confidently state that quantity of diet may safely be left to the natural appetite; and that it is quality only which is essential to abate and cure corpulence. These important desiderata have been attained by the most easy and comfortable means, by a system of diet, that formerly I should have thought dangerously generous.

He wrote that this "present dietary table" was far superior to what he was eating before:

It is more luxurious and liberal, independent of its blessed effect, but when it is proved to be more healthful, the comparisons are simply ridiculous . . . I am very much better both bodily and mentally and pleased to believe that I hold the reins of health and comfort in my own hands . . . It is simply miraculous and I am thankful to Almighty Providence for directing me through an extraordinary chance to the care of a man who worked such a change in so short a time.

Banting charged nothing for the first two editions of his booklet because he did not want to be accused of writing for profit. These 2,500 copies were snapped up and a third edition, still published in 1863, was sold for a pittance to cover his costs. In addition to paying Harvey's fees Banting gave him 50 pounds to be distributed to his favorite charities but still felt obligated to do more to repay his debt. In 1868, he endowed a new institution, the Middlesex County Convalescent Hospital for people who had to return to work after being hospitalized to make ends meet and often had relapses because they couldn't afford to rest at home.

Banting's booklet was so contrary to the established dietary doctrines that physicians were incensed and it was ridiculed. Nobody was able to deny that the diet worked, but since it had been published by a layman, doctors were fearful that this would undermine their position in society. As a result, they felt obligated to attack and denounce it and tried to do so at every opportunity, but **the only criticism they could come up with was that it was considered to be "unscientific".**

Dr. Harvey also suffered because, although he had an effective treatment for obesity, he could offer no convincing theory to explain why it worked. Although a noted Fellow of the Royal College of Surgeons, he was an easier target for the other members of his profession to attack and ridicule and his referral practice began to suffer. Nevertheless, so many people found that the diet worked that physicians could no longer ignore it and searched for some explanation. Everybody agreed that protein was not fattening and a compromise was offered by Dr. Felix Niemeyer of Stuttgart. He interpreted protein to mean only lean meat from which the fat had been trimmed and the "Banting" diet became a high protein-low fat-low carbohydrate diet. **Banting lived a healthy life and maintained a normal weight until his death in 1878 at the age of 81;** but he always maintained that this new diet was far inferior to the one that had so changed his life.

Other variations and modifications have appeared over the years. In 1955, Dr. Albert Pennington also found that "weight loss appeared to be inversely related to the amount of glycogenic material in the diet. Carbohydrate is 100%, protein 58% and fat 10% glycogenic." In other words, foods that increased insulin production put weight on and in this regard, carbohydrates were the worst and fats were the best. Pennington's diet was so successful that it was published in *Holiday* magazine and became known as "The Holiday Diet". Subsequently, Teller's *Calories Don't Count*, Stillman's *The Doctor's Quick Weight Loss Diet* and Tarnower's *The Complete Scarsdale Medical Diet* and other imitations also became best-sellers.

Despite his book's title, the Atkins diet was hardly a "Revolution" but it made the *New York Times* best seller list for four years. So many million copies were sold preaching the opposite of what the government was promoting that he was forced to defend his views in Congressional hearings. Attempts were even made to revoke his license based on allegations of other "unconventional" practices. His recent *New Diet Revolution* is also hardly new, but is similarly selling millions and receiving increasing support from respected authorities.

Coronaries, Cholesterol And LDL

So, if eating lots of fat doesn't cause weight gain, raise cholesterol, or increase risk for heart attacks, why do these beliefs persist? Does diet have anything to do with coronary artery disease? Does what you eat have a significant influence on your "cholesterol count", which TV ads constantly remind us is so important to monitor? Are heart attacks caused by an increase in cholesterol, low density lipoproteins (LDL bad cholesterol), apolipoproteins or other lipids? If not, then why are statin drugs that lower cholesterol and LDL so effective in reducing coronary events and mortality?

To address these in order, low fat, low cholesterol foods and beverages as well as cholesterol lowering drugs and products are a multi-billion dollar business. It is impossible to go into a supermarket without being bombarded by a plethora of products from soups to candies and cakes that are presumably healthier or have less calories because of their no fat or low fat content although this is often not true. Professional reputations are also at stake and nobody wants to rock the boat.

What you eat can very likely contribute to coronary heart disease, obesity, metabolic syndrome and diabetes. However, it is not fat but carbohydrates that are the culprits, particularly foods like pasta, rice and bread at the base of the famous Food Guide Pyramid promoted by Federal guidelines as a healthy low-fat diet. In addition, sugar or corn syrup in soft drinks, fruit juices and sports drinks are often consumed in copious amounts because they are fat free. These stimulate the production of insulin that triggers a cascade of events, which, when repeatedly invoked, have been shown to have a variety of deleterious effects.

Dietary fat intake has little influence on cholesterol or other lipid levels. In one very well documented report, an 86 year-old man who had consumed two-dozen eggs every day for 15 years had a normal cardiogram and normal cholesterol! In contrast, stress can cause a prompt and dramatic rise in cholesterol, although its contribution to coronary disease is mediated by other mechanisms.

The notion that high cholesterol caused coronary heart disease stemmed largely from the Framingham Study and Ancel Keys' Seven Country study showing statistical associations. Both of these have been shown to have serious flaws due to self-serving selection and data distortion. In the first Framingham report, the risk of dying from a heart attack with a cholesterol over 260 was 413% higher compared to men with cholesterol under 170. However, this disturbing disparity was partially due to the inclusion of subjects with very high values due to familial hypercholesterolemia.

A review of the data on 30-year follow-up showed that for men over 47, those with low cholesterol died just as frequently as others with high levels. Most studies show that high cholesterol is not a risk factor for women and that it is associated with reduced mortality in the elderly. Had Keys selected data from some of the other 15 countries available to him he would have concluded that high cholesterol reduced coronary deaths. Even in the countries he chose, there is a wide disparity in death rates from heart disease at the same cholesterol level that can't be explained.

To get around these and numerous other discrepancies we were then told that the source of the problem was really LDL "bad" cholesterol. "LDL has the strongest and most consistent relationship to individual and populations risk of CHD (coronary heart disease), and LDL-cholesterol is centrally and causally important in the pathogenetic chain leading to atherosclerosis and CHD" according to *Diet and Health*, published in 1989. This is the largest official review of heart disease, and although authored by the prestigious National Research Council, the four references they cited to support this statement failed to do so. In one study, women over 70 women with low LDL had a greater CHD risk. Similarly, Scott Grundy, a leading proponent of the diet-cholesterol-heart attack hypothesis wrote that **"Evidence is abundant that elevated LDL is a major cause of CHD and that lowering LDL levels reduces CHD risk." This is also not only unsupported and erroneous, but as will be seen, could lead to serious problems.**

Atherosclerosis, Inflammation And CRP

Since cholesterol is insoluble, it is transported in the blood inside spheres called lipoproteins, because they are composed of lipids and proteins. These easily dissolve in water since the outer lining consists largely of water-soluble proteins and the inside is composed of lipids, with room to contain insoluble molecules like cholesterol. Lipoproteins act like submarines to transport cholesterol from one part of the body to another and are characterized by their density. HDL (High Density Lipoprotein) carries cholesterol from artery walls to the liver, where it may be utilized or excreted along with bile. LDL (Low Density Lipoprotein) submarines ferry cholesterol from the liver to peripheral tissues and vessel walls to deliver it to the interior of cells when needed.

These natural substances are labeled "bad" and "good" because studies show that a low HDL or a high LDL is associated with a greater risk of heart attack with the reverse being true for a high HDL or low LDL. The lower the HDL/LDL ratio the greater the risk, but this hardly means that either of these cause atherosclerosis. They are simply statistical associations. Smoking causes a low HDL/LDL ratio but is probably associated with higher rates of heart attacks by other effects on blood vessels. Stress and hypertension are other risk factors that may be associated with a low HDL/LDL ratio but coronary events are more likely related to prolonged vasospasm. Conversely exercise increases HDL but also protects against coronary disease via other mechanisms. No studies show that sedentary, smoking, hypertensive and stressed individuals with high LDL's are at greater risk than others with low LDL's. Association never proves causation.

The proposal that the smaller LDL submarine can more readily infiltrate vessel walls to form a big cholesterol deposit also does not hold water. The single cell arterial wall is impervious to LDL molecules and can only be penetrated if it is damaged by injury or inflammation. If LDL was able to breach this barrier, then one would expect to see uniform rather than specific atherosclerotic deposits, much as sunlight affects all exposed tissues.

In addition, atherosclerotic plaque has all the characteristics of an inflammatory reaction, with macrophages and foam cells similar to the response seen following injury due to trauma or infection. The atherosclerotic lesions due to hypercholesterolemia that result from force feeding of fat or cholesterol to experimental animals are quite different both in their appearance and their distribution. **There is abundant additional evidence to support the contention that atherosclerotic plaque in humans is due to an active inflammatory process rather than the passive precipitation of cholesterol resulting from increased blood lipid concentrations.**

The process begins when the blood vessel wall is injured by chronic damage such as infections due to gum disease or seemingly minor bacterial and viral infections that produce few symptoms as well as hypertension, smoking and seemingly minor upper respiratory infections due to bacteria or viruses. Fatty tissues that line blood vessel walls become inflamed as white blood cells invade them and churn out inflammatory proteins in a misguided defense response. Heart attack patients have been shown to have much higher antibody titers to *Chlamydia pneumoniae* compared to patients hospitalized for other disorders. Chlamydia, which causes mild flu-like symptoms for a day or two, has also been cultured from atherosclerotic plaque.

Inflammation is commonly assessed by measuring C-reactive protein (CRP) a chemical liberated in response to injury and infection. An eight year study of 28,000 women followed for eight years reported two months ago in the *New England Journal of Medicine* showed that elevated CRP levels were twice as likely as high cholesterol to predict future heart attacks by as much as 15-25 years. About half the heart attacks and strokes observed were in patients with normal or low LDL levels. It is now clear that most heart attacks and coronary events are due to inflammatory processes that cause disruption of plaque that breaks off into the circulation rather than progressive occlusion of a vessel due to a steady but slow deposition of lipids as the cholesterol cartel constantly preaches.

More Nails For The Cholesterol Coffin

Σ In the Framingham Heart Study at least one third of all coronary heart disease events occurred in individuals with a cholesterol under 200. More recent figures have revealed that almost half of all heart attack and strokes occur in patients whose lipid levels are below average.

Σ **If elevated cholesterol and/or LDL levels are the cause of atherosclerosis, why is there no correlation with rates of stroke or peripheral arteriosclerosis?** One reason may be that many such complications often attributed to atherosclerotic occlusion really resulted from an unstable thrombus or clot that was a consequence of inflammation. Further support comes from the observation that the relative risk of such problems steadily increases as CRP values go from low-normal to high-normal and are markedly different at the ends of this scale.

Σ Prospective studies of healthy middle age men show that CRP levels are higher at baseline among those who subsequently developed either significant peripheral vascular complaints or required peripheral arterial surgery over the next 6 to 8 years.

Σ In the Women's Health Study of some 38,000 predominantly post-menopausal American women who were free of cardiovascular disease at study entry, the relative risk of future vascular events increased as the level of C-reactive protein went from low-normal to high-normal. This was true for all vascular events as well as for the specific combined endpoint of heart attack or stroke. Neither cholesterol and/or LDL measurements had any predictive value.

Σ The same observation holds for subgroups of middle-aged women who would be considered to be at very low risk because they have no evidence of any lipid predisposition, (high cholesterol, high LDL, high triglycerides, low HDL), no hypertension, no diabetes, don't smoke and have no family history of coronary or atherosclerotic disease. As the level of CRP increased, so too did the future risk of having some adverse vascular event.

Σ Just because you give a disorder a name does not mean that you have

accurately defined it, much less understand its cause(s) or consequences. Tuberculosis was previously thought to be due to close unsanitary living conditions because it occurred so frequently in association with such circumstances. Subsequently, Koch demonstrated that the tubercle bacillus was the *causa vera*, since the disease could not occur in its absence. Some diseases, like hypertension, do not have a *causa vera*. "Essential" hypertension is not a diagnosis but simply a description of some disturbance that causes a persistently elevated blood pressure measurement. Hypertension can have many causes, which explains why we have over 80 different drugs to treat it with no guarantee of which will work best in any individual patient.

Similarly, several factors can contribute to coronary heart disease as well as other complications of atherosclerosis. The notion that these result from a slow, steady deposition of cholesterol on the inner lining of vessel walls, like rust and sediment that builds up to restrict flow in a plumbing system is easier for the public to understand. This appealing analogy has been perpetuated for decades by powerful vested interests despite overwhelming evidence to the contrary. **Hyperlipidemia is only an associated risk marker**, like hypertension, diabetes, smoking, stress, high homocysteine, CRP, fibrinogen, genetic and other indicators of increased thrombotic tendencies.

Recent studies suggest that instability of plaque is what causes most problems and that this results from inflammation rather than elevated lipids. Even individuals with minimal atherosclerosis but elevated CRP levels are at much greater risk for vascular complications due to plaque rupture. Like homocysteine, inflammation is an important piece of the puzzle, but there may be others and their significance will depend on the ability to prove that correcting them dramatically reduces atherosclerosis. While hypertension is a "risk factor", it is more likely a result rather than cause of atherosclerosis since its successful treatment does not reduce coronary disease. **Association never proves causation.**

The Subtle But Sinister Side Effects Of Statins And The Importance of Q10

The reason all of this is important is the widespread and indiscriminate use of statins to lower cholesterol. **These drugs are now advocated not only for patients with elevated cholesterol, but also anyone with a risk factor for coronary disease, including hypertension, smoking, family history and all diabetics, including those with normal lipids.** It is impossible to watch TV or read a magazine without being told about the benefits of Lipitor, Zocor, Pravachol and other statins and it is estimated that close to 40 million Americans will soon be taking them. Unlike most of their predecessors, statins have been shown to significantly reduce coronary events without increasing mortality from other causes. Although they are constantly referred to in peer reviewed medical articles as "having a very high safety profile" this is not true. Baycol was withdrawn because of at least 52 deaths and numerous cases of rhabdomyolysis (a deterioration in muscle tissue that can cause severe pain and kidney failure) but all statins have this potential. Manufacturers do warn of this but state that such problems can be avoided by monitoring creatine phosphokinase (CPK), an enzyme believed to be a sensitive indicator of muscle damage. However, a recent paper showed that significant muscle damage could occur with normal CPK levels. It is also stated that statins are contraindicated in patients with liver disease or during pregnancy and lactation. What the public is not aware of is that statins can also cause severe fatigue, memory loss, amnesia, neuropathies and congestive heart failure. **All statins have**

been shown to be carcinogenic in laboratory animals at doses equivalent to those being prescribed for humans. There are increasing concerns about this based on clinical trials and the lag period between onset and clinical detection.

Many problems are ameliorated when statins are stopped and improvement is usually more rapid with Coenzyme Q10 supplementation. The reason for this is that **statins deplete the body of Q10**, an essential nutrient found in all cells that is responsible for converting calories from food into energy. Drug companies are well aware of this and Merck has had two patents on statin-Q10 combination pills since 1990, one of which states it is "a pharmaceutical composition comprising a pharmaceutical carrier and an effective anti-hypercholesterolemic amount of an HMG-CoA reductase inhibitor and an amount of Coenzyme Q10 effective to counteract HMG-CoA reductase inhibitor-associated skeletal muscle myopathy." These have never been advertised or implemented for obvious reasons.

Statins reduce coronary events but they do so not by lowering lipids but inhibiting inflammation. Since side effects are related to dosage, the current treatment goal of lowering LDL to an arbitrary level that is often difficult to achieve could prove very harmful. This will only lead to higher and higher doses and more adverse complications. Like aspirin, the amount required to reduce inflammation may be much lower than previously suspected and entirely unrelated to its use for other indications. Many believe that the current epidemic of heart failure stems from statins and that everyone taking statins should be on Q10 - **Stay tuned for more on Q10!**

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