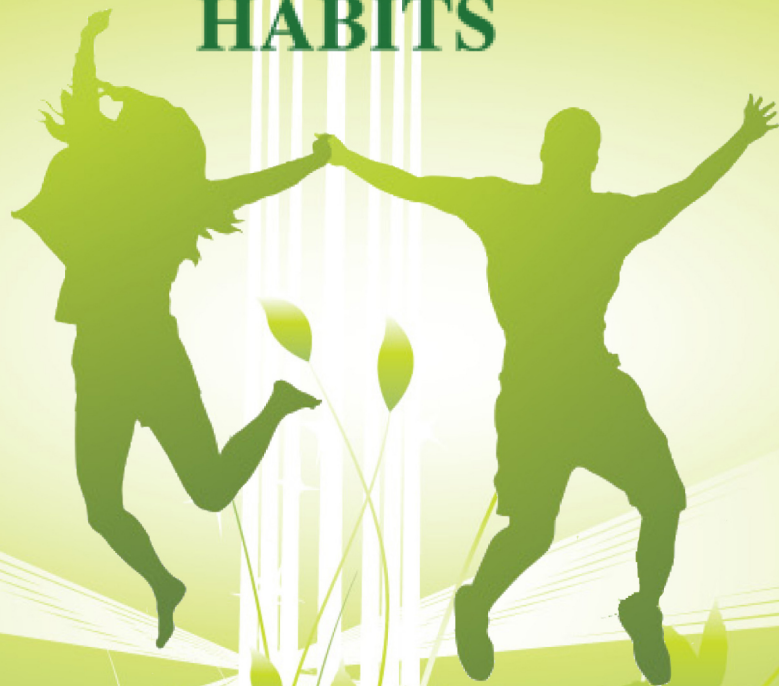




## CHANGING HABITS



### CHOLESTEROL REPORT

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## **The Cholesterol Scam**

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### **The Cholesterol Scam – Could This Be The Biggest Mistake in Medical History?**

“The cholesterol hypothesis can be likened to a cathedral built on a bog. Rather than admit they made a horrible mistake and let it sink, the builders decided to try and keep the cathedral afloat at all costs. Each time a crack appeared, a new buttress was built. Then further buttresses were built to support the original buttresses.” Dr Malcolm Kendrick (Medical Doctor and Author of Panic Nation)

The words ‘cholesterol’ and ‘saturated fat’ bring about a sense of fear for many people. So much so that they avoid manufactured foods containing them at all costs, checking the labels to make sure they are not on the nutritional information or are in minor amounts. People are also well versed in foods that naturally contain them, like butter, eggs, avocado, coconut and so on. This fear has been growing over the past two generations but it is time to stop this irrational trend. This report aims to take that fear away and help you understand, firstly, how this fear started and, secondly, how these nutrients are essential for health, energy, vitality and life.

I’ve always believed that, in essence, all discoveries and ideas about our diet came from a place of integrity in the beginning, but as discoveries were made and money was



invested and time showed another truth, then greed, public brainwashing, fear and momentum have turned many noble ideas into disasters for public health.

The saturated fat/cholesterol fear is one such disaster. I can tell you now that most doctors will not know this information and continue to see high blood cholesterol levels as an enemy and prescribe statin drugs and margarine. Many dietitians and nutritionists will also be naive and continue to say that margarine and low-fat is better than butter. And although an astounding amount of money has been spent on research to conclusively prove the link between saturated fat, cholesterol and heart disease, there exists an even greater amount of research published in peer-reviewed journals that completely liberates dietary saturated fat, cholesterol and elevated blood cholesterol from any role in heart disease.

In order to understand this irrational fear of cholesterol and saturated fat, it is important to go back to where it really started.

## The History of Heart Disease

Heart disease is a relatively new disease in that it has only been known since around 1912, when the physician James Herrick described the first heart attack. By the 1940s, an epidemic of heart disease had hit the USA which spawned interest in this area of research and medicine.

Many researchers implicated a high consumption of saturated fat and cholesterol in the diet as the cause of heart disease. In 1954, Dr Ancel Keys' influential Seven Countries Study was published demonstrating clear links between saturated fat intake and heart disease. He failed to add countries into his study that did not support his hypothesis of saturated fat intake and heart disease. By 1961, a growing momentum regarding fat intake and heart disease had started; after only 13 years of the Framingham study there seemed to be some confirmation of the link between raised cholesterol levels and heart disease, and not long after the first cholesterol-lowering drugs were developed. It's interesting that the Framingham study (which is still underway) can no longer prove this link. In the 1970s, Brown and Goldstein discovered the gene that leads to extremely high cholesterol levels (familial hypercholesterolaemia) and premature heart disease, and in 1985 they received a Nobel Prize for their work. The 1980s also brought about the discovery of statin drugs and the real fear about cholesterol levels started amongst the general population and medical fraternity. There has been a growing trend for all people with cholesterol levels deemed to be high to be on statin drugs. The number of people who are now on statin drugs is growing and the industry is worth billions and billions of pounds, euros and dollars per annum. There is a prediction that not too far into the future, 50% of the population will be on these cholesterol lowering drugs. In Australia alone in 1995 4.5% of the population aged over 45 were prescribed statin drugs, by 2010 the figure had reached 30%. The reason for this rise was that the "figure" for cholesterol levels being dangerous was lowered. A very convenient and lucrative measure to have changed.



Here we are in 2012 and the cholesterol/saturated fat fear is almost beyond control. This brief history of heart disease implicating dietary saturated fat and cholesterol has been like a freight train out of control, but, in reality, this hypothesis is far from the truth. In 1997, Professor Emeritus, Ancel Keys of the University of Minnesota said, "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." Ancel Keys must be turning in his grave seeing the health disaster that has come about due to his Seven Countries Study. He was never a fanatic about diet. He was prudent in what he did and this was exemplified by his long and active 100 years of life. Ancel Keys is known as the father of low-fat but he never would have predicted the stampede of fanaticism against fat that has ensued.

As a result of Keys' study, and further confirmation from other studies, a plethora of food manufacturers set about creating food that was low in saturated fat but high in sugar and additives to make you think you're eating fat. Now we have on the supermarket shelf thousands of so-called foods that are not food, but rather manipulated chemicals disguised as something edible that in reality are making us sick and fat. One such food is margarine and the hydrogenated and partially hydrogenated fats that adorn many food-like substances. It was because of the Seven Country Study that margarine got a foothold in the 'health' market.

Functional foods have become common place. These are foods with added nutrients in order to change a health outcome. For example, manufacturers of margarine, cream



cheese and sliced cheese to name a few, have added plant sterols and as a result the claim on the packaging states; “Clinically Proven to Lower Cholesterol” or “Lowers Cholesterol Re-Absorption”. These products remain on the supermarket shelf despite a meta-analysis (study of many studies on the subject) in 2012, published in the European Heart Journal concluding; “Our systematic review and meta-analysis did not reveal any evidence of an association between serum concentrations of plant sterols and risk of CVD”. In other words the claim is false and bogus. The unsuspecting buyer will be tricked into purchasing this product believing that it is helping their cholesterol levels.

A brief history of margarine is required to help understand the fear of cholesterol and saturated fat and the turn away from butter and stampede towards margarine. Procter and Gamble (a global company that supplies consumer products including pharmaceuticals, cleaning and personal care products) were once candle and soap manufacturers. In the 1920s their candles were made from tallow and had a very short shelf life, so the company commissioned a scientist to find a wax that never went off. The result was the adding of hydrogen to vegetable oil (hydrogenated vegetable oil) which produced a very hard fat/wax. When electricity became the main light source and candles were no longer needed, Procter and Gamble used the fat/wax as the consumable - Crisco Oil, eventually dyeing it yellow and adding flavour to make margarine. It was sold as the ‘cheap alternative to butter’.

When Keys' study was released, margarine was upgraded to the 'healthy alternative to butter' rather than the cheap version of butter. When the cholesterol fear mounted in the 1980s, margarine was sold as the food that would lower cholesterol and advertising was directly linked to this claim with 3-week challenges issued to the public to try it. But of course this was false and now when you look at a tub of margarine that claims to lower cholesterol you will see the word 'absorption' in very small print after 'lower cholesterol'. What a claim! Firstly, cholesterol consumption has nothing to do with blood cholesterol levels and, secondly, the addition of plant sterols to the margarine is what enables the manufacturers to have the claim that it reduces absorption of cholesterol (which is as you now know an unfounded claim). And, if you have cottoned on to what I'm saying, yes, vegetables and grains (like oats, for instance) have these plant sterols which naturally lower cholesterol absorption. (The price of margarine is also a worry as once it was sold as the cheap version of butter and now a 375gm tub can cost up to \$8.50.)

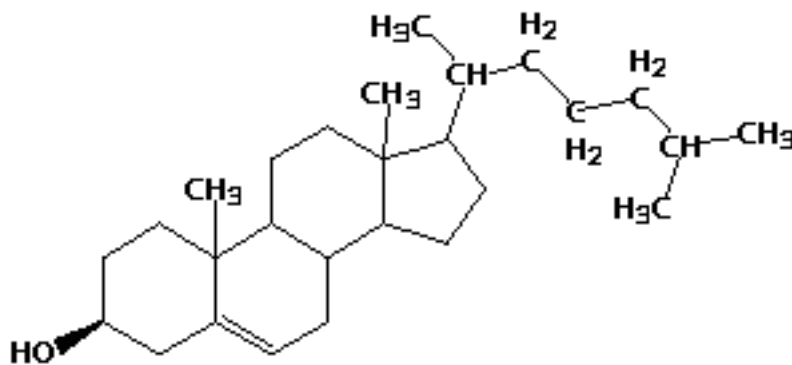
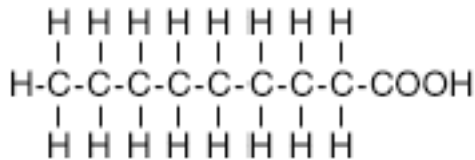
What is disturbing is that in 2007 on many front pages of national newspapers the heading bellowed that 'Trans Fats' were the new fat danger and were more dangerous than saturated fat. Trans fats are found in nature but in very small amounts. The partial hydrogenation of a vegetable oil (which gives a less hard fat than the full hydrogenation of vegetable oil, but uses the same process) is what creates trans fat. All margarine, vegetable fats, hydrogenated and partially-hydrogenated vegetable oils plus any food made up with these foods – including baked goods, donuts, pastries, breads, tinned soups, crackers and biscuits, to name a few – contained trans fats up until 2007. But

with the public now knowing that trans fats were dangerous, the formula for margarine had to be changed. For more information on what they did, see the Trans Fat/Hydrogenation Report (available from [www.changinghabits.com.au](http://www.changinghabits.com.au)). What is so infuriating to me is that Dr Mary Enig and other prominent scientists had been warning people about the dangers of trans fat since 1978. In many countries around the world and in some states in the USA trans fats are totally banned, yet in Australia and New Zealand there is complacency on the part of our food authority FSANZ – they are yet to do anything about it.

Margarine has become a chameleon, beginning its career as candle wax and soap, moving to Crisco shortening, then the cheap version of butter. Later becoming the healthy version of butter, to cholesterol-lowering, and now trans fat free and expensive. Many doctors with very little knowledge of nutrition and food, prescribe margarine and statin drugs for their patients. Let's now look at the reality of saturated fat, cholesterol and heart disease.

I've never quite understood why cholesterol and saturated fat have gone hand-in-hand as the culprits for heart disease. They are two very different structures with very different jobs in the body. Take a look for yourself at the two diagrams below: saturated fat is the molecule that looks straight and the other molecule is cholesterol.





## Natural Saturated Fats in the Body

Saturated fats play a very important role in total body health. They constitute at least 50% of the cell membrane, which gives the cell its necessary stiffness and integrity, and when you realise that we are made up of trillions of cells, this is a very important function. Saturated fat also plays a vital role in the health of bones; for calcium and other minerals to be effectively incorporated into the bones, at least 50% of the dietary fat should be saturated. They also protect the liver from alcohol and other toxins like painkillers. These wonderful fats help enhance the immune system, they have



antimicrobial properties and are protective against harmful microorganisms in the digestive tract. Saturated fats are required for proper use of essential fatty acids. Elongated omega 3 fatty acids are better retained in the tissues when the diet is rich in saturated fats. What is amazing is that the saturated fats; stearic acid and palmitic acid are the preferred foods for the heart, which might be why the fat around the heart muscle is high in saturated fat. The heart will draw on this reserve of fat in times of stress. It is important to note here that I am talking about natural saturated fats found in butter, coconut and grass fed animal meat, rather than saturated fats made as a result of the hydrogenation of a vegetable oil as is found in margarine, vegetable shortenings and partially hydrogenated vegetable oils.

It is also interesting to note that breast milk is high in saturated fat, and the body makes saturated fat in order to store fat in fat cells for future energy use.

## **Cholesterol in the Body**

Cholesterol, to me, is a super food. Your liver and small intestines produce and regulate cholesterol levels needed as building blocks for cells and hormones. Making up more than 50% of the cell membrane, cholesterol maintains membrane permeability and protects the core of the cell while still allowing the cell to be fluid. Cholesterol is the precursor for Vitamin D; when converting sunlight into vitamin D, cholesterol in the tissues acts as the catalyst for this important process. Vitamin D, in turn, is crucial for mineral metabolism and targets over 2000 human genes. Vitamin D is also the gate keeper between the digestive tract and the immune system, warding off leaky gut



syndrome and helping the immune system do its job. Vitamin D deficiency is linked to some cancers, heart disease and autoimmune diseases. Cholesterol maintains proper gender-specific hormonal levels. It is also important for the production of the hormone, cortisol (the stress hormone) and works to digest and absorb fats, nutrients and vitamins. Studies on cholesterol are beginning to unveil its role in metabolising calcium, boosting the immune system, cancer prevention and mental health. So you can see that cholesterol is a vital nutrient that is important for physical and mental health.

## **Cholesterol Readings**

When a cholesterol reading is taken the total cholesterol is worked out as well as triglycerides and high density lipo proteins (HDL) and low density lipo proteins (LDL). LDLs are called 'bad' and HDLs are called 'good'. The first time I heard this I was puzzled as I couldn't figure out why anything the body makes for regular function could possibly be bad. Let me explain. The job of LDLs is to take cholesterol from the liver, where it is made, into the tissues where it manufactures the products we need, like hormones and vitamin D. The job of HDLs is to take cholesterol from the tissues to the liver for storage, so when the body has no immediate need for the cholesterol to make hormones etc. it sends it into storage. In my way of thinking, HDLs and LDLs are both necessary and wonderful products that create a cholesterol homeostasis. Neither is bad and neither is good; they are just natural functions working on a regular basis for normal healthy body function. I hope that is now cleared up. While we are on cholesterol readings, if you have what is deemed a high triglyceride level then a change in diet away from refined carbohydrates and alcohol will correct the situation.

When you ask someone what their cholesterol reading is, they will tell you a figure, but total cholesterol (LDL, HDL) is not a static figure but something that is very fluid. From day to day, week to week, cholesterol readings will change significantly. In fact one day you could have a blood test and your reading may be something that your doctor tells you is dangerous and you need to go onto drugs immediately to lower your cholesterol because the chances of you having a heart attack the next day is eminent. But if you go back a week later for another blood test and the demand by the body for cholesterol is less then you may find your reading is at the optimal level. In fact a total cholesterol reading can change 1.8mmol/l up or down without changing diet or taking medication just because of the body's needs. LDL and HDL levels will also change up or down depending on the bodies need for storage or use. Following are three tables outlining the current thinking of desirable and non-desirable levels of cholesterol, HDL and LDL and the variance that can occur from one day to the next.

Total Cholesterol can vary day to day week to week up or down by 70mg/dl or 1.8mmol/l

Total cholesterol = LDL, HDL and VLDL

mg/dl	mmol/l	interpretation
Below 200	Below 5.18	Desirable
200 - 239	5.18 - 6.19	Borderline High
240 and over	6.19 and over	High

HDL Cholesterol can vary day to day, week to week up or down by 10mg/dl or .25 mmol

mg/dl	mmol/l	Interpretation
Below 40	Below 1.04	Low
40-59	1.04 - 1.53	Moderate
60 and over	1.53 and over	High

LDL Cholesterol can vary day to day, week to week up or down by 30mg/dl or .8 mmol/l

mg/dl	mmol/l	Interpretation
Below 100	below 2.59	Optimal
100-129	2.59-3.34	Near or above optimal
130-159	3.35-4.12	Borderline high
160-189	4.13-4.90	High
190 and over	4.90 and over	Very high

The body does not need you to eat cholesterol; it is quite capable of making cholesterol from other products that you consume. Dietary cholesterol has nothing or very little to do with blood cholesterol – it is a nice benefit when you eat it, but it is not essential.

The reason the establishment suggest the LDL is bad is because atherosclerosis plaque (narrowing of the arteries and the precursor to heart disease and stroke) has cholesterol within its fold, so the assumption was if we keep cholesterol out of the

tissues then we will stop atherosclerosis. The old paradigm goes back to Anitschkov, who is the scientist who did the experiments with cholesterol in rabbits and is responsible for developing the theory that cholesterol clogs the arteries and causes heart disease.

But the body is not that simple. By reducing LDL and raising HDL, we then take the precursors for products required for the body away from the tissues into storage. What I find quite disturbing at the moment is that we now have an epidemic of Vitamin D deficiencies. After I explain this whole process many people tell me they are on cholesterol-lowering drugs and have developed a Vitamin D deficiency since taking them. Hello? Can no one see the correlation? Oh, and by the way, Vitamin D will not be the only substance being affected. Cortisol (the stress hormone) will also be reduced in production along with gender-specific hormones and other important substances for the body.

A number or a ratio of blood readings does not indicate disease but rather tells us that perhaps something is wrong and that we need to investigate further.

High Cholesterol Readings may be indicators of the following;

1. Leptin Resistance
2. Thyroid Imbalances - T3 is required to activate LDL receptors therefore, hashimotos, graves, iodine deficiency and hypothyroidism can be the reason for increase LDL cholesterol.



3. Infection - LDL levels will rise with infection (both HDL and LDL have a big part to play in immunity)
4. Inflammation - after surgery, dental work, sprains, foods we eat LDL levels will increase.
5. Iron and copper deficiency related to thyroid conditions
6. Familial hypercholesterolemia where one of the genes that codes for the LDL receptor doesn't function well. Or it doesn't function at all.

## **The Importance of Cholesterol**

There is a genetic disorder called Smith-Lemli-Opitz syndrome, or SLOS. This is where the body cannot manufacture cholesterol. Most conceptions where the baby has SLOS are spontaneously aborted. But in rare cases when someone is actually born with it, they have facial and skeletal abnormalities, mental retardation, autism, hyperactivity disorders, ADHD, visual dysfunction, endocrine dysfunction, and then serious self-aggression and violent behaviour.

I just love the human body – in most situations it is born perfect and given the right resources and left alone to do what it does best, it can be healthy. So many people do not understand this and believe that their magnificent body is flawed and that they need drugs to control all processes from blood pressure, blood sugar, blood cholesterol, brain chemistry and so on in order to be healthy. The scary thing is that many doctors tell their patients that they will need to be on these drugs for the rest of their lives. If any doctor has said this to you, I suggest you need to find another doctor whose aim is



to get you healthy so you don't need to be on body and mind altering drugs. Drugs have a black and white job, but the body is not black and white. When you affect one process you affect 1000s of others, not just the one the medication is prescribed for. The human body is not a machine. It possesses an innate intelligence that has lived for 1000s of years on this planet eating foods we evolved to eat without heart disease and without microscopic observation. It is important that I add here that medicine is a wonderful art and we have advanced so much in what we have learnt from research and science. Without a doubt there are situations where medicine has me in awe, but like all healing arts it has limitations and it is time that medicine and science realise they are not the panacea for everything.

## **The Chicken or The Egg**

Let's go back to cholesterol being part of the atherosclerotic plaques. Yes, it is true that it is, but is the cholesterol the chicken or the egg? Does cholesterol cause the plaque or does the cholesterol come after the plaque has started? More and more research is showing that it is inflammation (or damage) that causes the beginnings of a plaque, and cholesterol goes to the point of inflammation as a policeman would go to a robbery. Imagine if you always showed up after the robbery had occurred, and the police had arrived; after sometime you might assume that the policeman is the cause of the robbery, but of course you would be wrong. So when cholesterol was being researched, the scientist always arrived after the police (cholesterol) came and therefore declared them to be the culprits in the robbery (plaque). It is an easy assumption to make, but so very wrong.



Evidence is mounting as to the cause of atherosclerosis and it does not include blood cholesterol in a healthy state although damaged cholesterol without antioxidants is another matter. More and more professionals and heart specialists realise that inflammation of the blood vessels is what begins and sustains the plaque formation. Causes of this inflammation can be parasites, rogue foreign bodies including fungi, bacteria and viruses, chemicals that we consume, breathe in or put on our skin, as well as foreign food particles, including the 1000s of flavours, colours, additives, man-made fats, processed foods and technology-driven foods we eat that the body has no idea what to do with. I also believe the mechanism of vitamin supplementation (single dose vitamins or minerals made in a laboratory) may have some effect on the inflammation of blood vessels.

The problem is that the acute inflammation, which is good, turns into chronic inflammation from the continuous bombardment to the body from the minute we get up till we go to bed. If, on occasion, some foreign food substance or virus enters our system then the body will mount a defense and clean up the foreign substance with acute inflammation and that's the end of that. But when we constantly bombard the body, eventually it protects itself causing chronic inflammation which, in turn, leads to plaque on arterial walls. A good analogy for this is to imagine that if you kept scratching your skin in exactly the same place day in and day out. Firstly you will bleed, then a scab will form, but if the irritation keeps going the scab will never fully heal and the skin will manufacture a scar that will get thicker and thicker until it has protected itself from the offending scratcher. Of course this is an oversimplification of the amazing process of the

body but it will give you an idea. Prominent New York pathologist and doctor Meyer Texon says, “Accusing fat and cholesterol of causing the injury that led to atherosclerosis is akin to accusing the white blood cells of causing infection; they are both there to help repair.”

## **The Good, the Good, and the Good of Cholesterol**

Historically (1936, 1961, 1962), before epidemiology research, it was found, by studying bodies post mortem, that cholesterol levels whether high or low, had no impact on the development of atherosclerosis, the major cause of heart disease. This fact has been confirmed in subsequent studies. In the last decade the studies concerning atherosclerotic plaque have been centered on inflammation, as previously discussed. For many years, scientists have suspected that viruses and bacteria, in particular cytomegalovirus and chlamydia pneumonia, participate in the development of atherosclerosis. There is a growing belief that rather than checking blood cholesterol, a better blood indicator is to test for levels of C-reactive, a protein secreted by the liver during infection and inflammation.

There is a plethora of research about levels of cholesterol and how higher levels of cholesterol are advantageous to health. The risk of colon cancer increases with low blood cholesterol. Two decades of research in Japan showed that low cholesterol increased the risk of stroke; further studies in the US agreed with this, showing that death by brain hemorrhage in middle-aged men was six times greater if blood cholesterol levels were low. A study at Yale University revealed that persons aged older



than 70 with low blood cholesterol levels, died twice as often from a heart attack as did old people with high blood cholesterol. Interesting!

Brain function relies on cholesterol; in fact much of the brain is cholesterol. There has been some observation that when men in particular are undertaking cholesterol-lowering regimes there has been an increase in the incidence of suicide. There is also an association between aggressive behaviour and low cholesterol, as observed in institutions, and current research has revealed an increase in Alzheimer's and diabetes associated with cholesterol lowering medications.

The whole idea of reducing cholesterol levels is to prolong life and to be healthy but evidence seems to be pointing to the exact opposite: those with low cholesterol levels have higher morbidity than those with high cholesterol. The evidence in the scientific literature is mounting and more cardiologists are beginning to agree.

Immunity is also helped by a healthy higher cholesterol – it protects against infection. LDL cholesterol binds and inactivates dangerous bacterial toxins. Research has shown that with cholesterol levels below 4.5mmol/L, the number of various types of white blood cells were significantly lower. Men with high cholesterol levels have stronger immune systems as they have more lymphocytes, total T-cells, helper T-cells and CD8 cells. And lastly, as far as we know, cholesterol works as an antioxidant protecting cell membranes from free-radical damage.



Wow! Whoever thought with the propaganda that circulates about the dangers of cholesterol that it could be so very, very important for healthy normal function in the human body?

I was speaking with a medical doctor and writer and we were discussing the whole issue of cholesterol and how the medical fraternity has it so wrong. The doctor said that it was very similar to what we first thought caused asthma and how the medications that were used to stop asthma attacks actually did work. But the people who were taking the medication lived shorter lives. Now that they understand what actually does cause asthma, lifestyle choices can make all the difference so asthmatics have a long and healthy life. Yes, the drugs work to lower cholesterol (as did the drugs to help people breathe easier) but it doesn't necessarily mean that the patient will live a longer and more healthy life.

## **What about the Medications, like Statins?**

Statin Drugs, which are a group of medications that lower cholesterol, have been known as the miracle medicine, but in a recent study published in the British Medical Journal a different story is told. For every heart attack prevented by the statin drugs, two or more people suffered cataracts, liver damage, kidney failure and/or extreme muscle weakness as a result of taking the drug. Other side effects that result from taking this drug are nausea, diarrhea, abdominal pain, headaches, skin rashes, dementia, diabetes and, of course, Vitamin D deficiency.





Cholesterol-lowering drugs do what they say – they lower cholesterol levels in the blood, and they do this by inhibiting the enzyme needed to manufacture cholesterol in the liver. But as we have seen this is not necessarily a good thing.

The following drugs are statin drugs: Lipitor (atorvastatin), Mevacor (lovastatin), Lescol (fluvastatin), Pravachol (pravastatin), Zocor (simvastatin) and Crestor (rosuvastatin). Often a doctor will start you on one and if that doesn't agree with you they will keep trying a different statin drug until the side effects do not affect your quality of life. Each medicine has its own particular characteristics but, in essence, they inhibit the enzyme in the liver that creates cholesterol.

For many of you, this report has probably presented a bunch of new concepts and ideas. I recommend that you read this report a couple of times to fully understand the ramifications of what we thought was right but is now such a terrible train wreck that destroys health.

## **What To Do Now**

1. Find yourself a doctor who is more concerned about nutrition and lifestyle than dispensing medications and is prepared to look at leptin resistance, thyroid function, inflammation and immunity issues before medications. [www.acnem.org](http://www.acnem.org)
2. Read this report a couple of times so you really understand that what we thought was correct now causes major problems – then you can make the right choice for your health and your life.



3. Start the 21 Day Program and make sure you begin your day with the protein breakfast shake.
4. Look at the Four Phase Fat Elimination HCG Protocol on Changing Habits which helps create Leptin Sensitivity.
5. After you have finished the 21 Day program and 4PFEHCG Protocol begin changing habits by starting at habit one in Changing Habits Changing Lives. This book will help you to understand which foods do not cause inflammation in the blood vessels.
5. Atherosclerotic plaques can be reversed, but you will need to stop doing the things that cause the inflammation. Following, with the most important ones listed first, is a list of things you should consider:
  1. What you put in your mouth (go to [www.changinghabits.com.au](http://www.changinghabits.com.au))
  2. What you put on your skin (visit [www.likechocolateforwomen.com](http://www.likechocolateforwomen.com))
  3. The water you drink – find yourself a good water filter
  4. Having yourself checked for parasites, and try to use a natural remedy if any are found
  5. The chemicals you use in your home (go to [www.enjo.com](http://www.enjo.com))
  6. Driving your car with the windows down for the first 5 minutes
  7. Not using any automatic or electrical devices for room sprays or fresheners
  8. Not using any car fresheners
  9. Any bug killing sprays you use – be aware of chemicals



10. The washing detergents you use – again, be wary of chemicals; there are many natural products available.



## **Changing Habits References**

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The Great Cholesterol Con by Anthony Colpo

Overdose by Jay S Cohen

The Great Cholesterol Lie by Dr Dwight Lundell MD [www.thecholesterolie.com](http://www.thecholesterolie.com)

The Great Cholesterol Myth by Dr Malcolm Kendrick

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<http://eurheartj.oxfordjournals.org/content/33/4/444.abstract> 2012

Controversial Role of Plant Sterol Esters in the Management of Hypercholesterolemia

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The Healthy Skeptic <http://chriskresser.com/heartdisease>

Dr Jack Kruse Blog <http://jackkruse.com/rewiring-the-leptin-rx-reset/>

Cholesterol Clarity - Jimmy Moore and Dr Eric Westman

**Additional information about Statin Drugs.** Stephanie Seneff has given me permission to add her work at the end of my cholesterol report. Her knowledge of how the body works and how Statins work will help you make the decision as to whether you want to continue taking the medications. She is detailed in her description of how the medication effects the body. It is well worth the read.

[http://people.csail.mit.edu/seneff/why\\_statins\\_dont\\_really\\_work.html](http://people.csail.mit.edu/seneff/why_statins_dont_really_work.html)



## How Statins Really Work Explains Why They Don't Really Work.

by Stephanie Seneff

March 11, 2011

### **1. Introduction**

The statin industry has enjoyed a thirty year run of steadily increasing profits, as they find ever more ways to justify expanding the definition of the segment of the population that qualify for statin therapy. Large, placebo-controlled studies have provided evidence that statins can substantially reduce the incidence of heart attack. High serum cholesterol is indeed correlated with heart disease, and statins, by interfering with the body's ability to synthesize cholesterol, are extremely effective in lowering the numbers. Heart disease is the number one cause of death in the U.S. and, increasingly, worldwide. What's not to like about statin drugs?

I predict that the statin drug run is about to end, and it will be a hard landing. The thalidomide disaster of the 1950's and the hormone replacement therapy fiasco of the 1990's will pale by comparison to the dramatic rise and fall of the statin industry. I can see the tide slowly turning, and I believe it will eventually crescendo into a tidal wave, but misinformation is remarkably persistent, so it may take years.

I have spent much of my time in the last few years combing the research literature on metabolism, diabetes, heart disease, Alzheimer's, and statin drugs. Thus far, in addition to posting essays on the web, I have, together with collaborators, published two journal



articles related to metabolism, diabetes, and heart disease (Seneff1 et al., 2011), and Alzheimer's disease (Seneff2 et al., 2011). Two more articles, concerning a crucial role for cholesterol sulfate in metabolism, are currently under review (Seneff3 et al., Seneff4 et al.). I have been driven by the need to understand how a drug that interferes with the synthesis of cholesterol, a nutrient that is essential to human life, could possibly have a positive impact on health. I have finally been rewarded with an explanation for an apparent positive benefit of statins that I can believe, but one that soundly refutes the idea that statins are protective. I will, in fact, make the bold claim that nobody qualifies for statin therapy, and that statin drugs can best be described as toxins.

## **2. Cholesterol and Statins**

I would like to start by reexamining the claim that statins cut heart attack incidence by a third. What exactly does this mean? A meta study reviewing seven drug trials, involving in total 42,848 patients, ranging over a three to five year period, showed a 29% decreased risk of a major cardiac event (Thavendiranathan et al., 2006). But because heart attacks were rare among this group, what this translates to in absolute terms is that 60 patients would need to be treated for an average of 4.3 years to protect one of them from a single heart attack. However, essentially all of them will experience increased frailty and mental decline, a subject to which I will return in depth later on in this essay.

The impact of the damage due to the statin anti-cholesterol mythology extends far beyond those who actually consume the statin pills. Cholesterol has been demonized by





the statin industry, and as a consequence Americans have become conditioned to avoid all foods containing cholesterol. This is a grave mistake, as it places a much bigger burden on the body to synthesize sufficient cholesterol to support the body's needs, and it deprives us of several essential nutrients. I am pained to watch someone crack open an egg and toss out the yolk because it contains "too much" cholesterol. Eggs are a very healthy food, but the yolk contains all the important nutrients. After all, the yolk is what allows the chick embryo to mature into a chicken. Americans are currently experiencing widespread deficiencies in several crucial nutrients that are abundant in foods that contain cholesterol, such as choline, zinc, niacin, vitamin A and vitamin D.

Cholesterol is a remarkable substance, without which all of us would die. There are three distinguishing factors which give animals an advantage over plants: a nervous system, mobility, and cholesterol. Cholesterol, absent from plants, is the key molecule that allows animals to have mobility and a nervous system. Cholesterol has unique chemical properties that are exploited in the lipid bilayers that surround all animal cells: as cholesterol concentrations are increased, membrane fluidity is decreased, up to a certain critical concentration, after which cholesterol starts to increase fluidity (Haines, 2001). Animal cells exploit this property to great advantage in orchestrating ion transport, which is essential for both mobility and nerve signal transport. Animal cell membranes are populated with a large number of specialized island regions appropriately called lipid rafts. Cholesterol gathers in high concentrations in lipid rafts, allowing ions to flow freely through these confined regions. Cholesterol serves a crucial role in the non-lipid raft regions as well, by preventing small charged ions,

predominantly sodium ( $\text{Na}^+$ ) and potassium ( $\text{K}^+$ ), from leaking across cell membranes. In the absence of cholesterol, cells would have to expend a great deal more energy pulling these leaked ions back across the membrane against a concentration gradient.

In addition to this essential role in ion transport, cholesterol is the precursor to vitamin D3, the sex hormones, estrogen, progesterone, and testosterone, and the steroid hormones such as cortisol. Cholesterol is absolutely essential to the cell membranes of all of our cells, where it protects the cell not only from ion leaks but also from oxidation damage to membrane fats. While the brain contains only 2% of the body's weight, it houses 25% of the body's cholesterol. Cholesterol is vital to the brain for nerve signal transport at synapses and through the long axons that communicate from one side of the brain to the other. Cholesterol sulfate plays an important role in the metabolism of fats via bile acids, as well as in immune defenses against invasion by pathogenic organisms.

Statin drugs inhibit the action of an enzyme, HMG coenzyme A reductase, that catalyses an early step in the 25-step process that produces cholesterol. This step is also an early step in the synthesis of a number of other powerful biological substances that are involved in cellular regulation processes and antioxidant effects. One of these is coenzyme Q10, present in the greatest concentration in the heart, which plays an important role in mitochondrial energy production and acts as a potent antioxidant (Gottlieb et al., 2000). Statins also interfere with cell-signaling mechanisms mediated by so-called G-proteins, which orchestrate complex metabolic responses to stressed conditions. Another crucial substance whose synthesis is blocked is dolichol, which



plays a crucial role in the endoplasmic reticulum. We can't begin to imagine what diverse effects all of this disruption, due to interference with HMG coenzyme A reductase, might have on the cell's ability to function.

### **3. LDL, HDL, and Fructose**

We have been trained by our physicians to worry about elevated serum levels of low density lipoprotein (LDL), with respect to heart disease. LDL is not a type of cholesterol, but rather can be viewed as a container that transports fats, cholesterol, vitamin D, and fat-soluble anti-oxidants to all the tissues of the body. Because they are not water-soluble, these nutrients must be packaged up and transported inside LDL particles in the blood stream. If you interfere with the production of LDL, you will reduce the bioavailability of all these nutrients to your body's cells.

The outer shell of an LDL particle is made up mainly of lipoproteins and cholesterol. The lipoproteins contain proteins on the outside of the shell and lipids (fats) in the interior layer. If the outer shell is deficient in cholesterol, the fats in the lipoproteins become more vulnerable to attack by oxygen, ever-present in the blood stream. LDL particles also contain a special protein called "apoB" which enables LDL to deliver its goods to cells in need. ApoB is vulnerable to attack by glucose and other blood sugars, especially fructose. Diabetes results in an increased concentration of sugar in the blood, which further compromises the LDL particles, by gumming up apoB. Oxidized and glycated LDL particles become less efficient in delivering their contents to the cells.



Thus, they stick around longer in the bloodstream, and the measured serum LDL level goes up.

Worse than that, once LDL particles have finally delivered their contents, they become "small dense LDL particles," remnants that would ordinarily be returned to the liver to be broken down and recycled. But the attached sugars interfere with this process as well, so the task of breaking them down is assumed instead by macrophages in the artery wall and elsewhere in the body, through a unique scavenger operation. The macrophages are especially skilled to extract cholesterol from damaged LDL particles and insert it into HDL particles. Small dense LDL particles become trapped in the artery wall so that the macrophages can salvage and recycle their contents, and this is the basic source of atherosclerosis. HDL particles are the so-called "good cholesterol," and the amount of cholesterol in HDL particles is the lipid metric with the strongest correlation with heart disease, where less cholesterol is associated with increased risk. So the macrophages in the plaque are actually performing a very useful role in increasing the amount of HDL cholesterol and reducing the amount of small dense LDL.

The LDL particles are produced by the liver, which synthesizes cholesterol to insert into their shells, as well as into their contents. The liver is also responsible for breaking down fructose and converting it into fat (Collison et al., 2009). Fructose is ten times more active than glucose at glycation proteins, and is therefore very dangerous in the blood serum (Seneff1 et al., 2011). When you eat a lot of fructose (such as the high fructose corn syrup present in lots of processed foods and carbonated beverages), the liver is burdened with getting the fructose out of the blood and converting it to fat, and



it therefore can not keep up with cholesterol supply. As I said before, the fats can not be safely transported if there is not enough cholesterol. The liver has to ship out all that fat produced from the fructose, so it produces low quality LDL particles, containing insufficient protective cholesterol. So you end up with a really bad situation where the LDL particles are especially vulnerable to attack, and attacking sugars are readily available to do their damage.

#### **4. How Statins Destroy Muscles**

Europe, especially the U.K., has become much enamored of statins in recent years. The U.K. now has the dubious distinction of being the only country where statins can be purchased over-the-counter, and the amount of statin consumption there has increased more than 120% in recent years (Walley et al, 2005). Increasingly, orthopedic clinics are seeing patients whose problems turn out to be solvable by simply terminating statin therapy, as evidenced by a recent report of three cases within a single year in one clinic, all of whom had normal creatine kinase levels, the usual indicator of muscle damage monitored with statin usage, and all of whom were "cured" by simply stopping statin therapy (Shyam Kumar et al., 2008). In fact, creatine kinase monitoring is not sufficient to assure that statins are not damaging your muscles (Phillips et al., 2002).

Since the liver synthesizes much of the cholesterol supply to the cells, statin therapy greatly impacts the liver, resulting in a sharp reduction in the amount of cholesterol it can synthesize. A direct consequence is that the liver is severely impaired in its ability to convert fructose to fat, because it has no way to safely package up the fat for

transport without cholesterol (Vila et al., 2011). Fructose builds up in the blood stream, causing lots of damage to serum proteins.

The skeletal muscle cells are severely affected by statin therapy. Four complications they now face are: (1) their mitochondria are inefficient due to insufficient coenzyme Q10, (2) their cell walls are more vulnerable to oxidation and glycation damage due to increased fructose concentrations in the blood, reduced cholesterol in their membranes, and reduced antioxidant supply, (3) there's a reduced supply of fats as fuel because of the reduction in LDL particles, and (4) crucial ions like sodium and potassium are leaking across their membranes, reducing their charge gradient. Furthermore, glucose entry, mediated by insulin, is constrained to take place at those lipid rafts that are concentrated in cholesterol. Because of the depleted cholesterol supply, there are fewer lipid rafts, and this interferes with glucose uptake. Glucose and fats are the main sources of energy for muscles, and both are compromised.

As I mentioned earlier, statins interfere with the synthesis of coenzyme Q10 (Langsjoen and Langsjoen, 2003), which is highly concentrated in the heart as well as the skeletal muscles, and, in fact, in all cells that have a high metabolic rate. It plays an essential role in the citric acid cycle in mitochondria, responsible for the supply of much of the cell's energy needs. Carbohydrates and fats are broken down in the presence of oxygen to produce water and carbon dioxide as by-products. The energy currency produced is adenosine triphosphate (ATP), and it becomes severely depleted in the muscle cells as a consequence of the reduced supply of coenzyme Q10.





The muscle cells have a potential way out, using an alternative fuel source, which doesn't involve the mitochondria, doesn't require oxygen, and doesn't require insulin. What it requires is an abundance of fructose in the blood, and fortunately (or unfortunately, depending on your point of view) the liver's statin-induced impairment results in an abundance of serum fructose. Through an anaerobic process taking place in the cytoplasm, specialized muscle fibers skim off just a bit of the energy available from fructose, and produce lactate as a product, releasing it back into the blood stream. They have to process a huge amount of fructose to produce enough energy for their own use. Indeed, statin therapy has been shown to increase the production of lactate by skeletal muscles (Pinieux et al, 1996).

Converting one fructose molecule to lactate yields only two ATP's, whereas processing a sugar molecule all the way to carbon dioxide and water in the mitochondria yields 38 ATP's. In other words, you need 19 times as much substrate to obtain an equivalent amount of energy. The lactate that builds up in the blood stream is a boon to both the heart and the liver, because they can use it as a substitute fuel source, a much safer option than glucose or fructose. Lactate is actually an extremely healthy fuel, water-soluble like a sugar but not a glyating agent.

So the burden of processing excess fructose is shifted from the liver to the muscle cells, and the heart is supplied with plenty of lactate, a high-quality fuel that does not lead to destructive glycation damage. LDL levels fall, because the liver can't keep up with fructose removal, but the supply of lactate, a fuel that can travel freely in the blood (does not have to be packaged up inside LDL particles) saves the day for the heart,



which would otherwise feast off of the fats provided by the LDL particles. I think this is the crucial effect of statin therapy that leads to a reduction in heart attack risk: the heart is well supplied with a healthy alternative fuel.

This is all well and good, except that the muscle cells get wrecked in the process. Their cell walls are depleted in cholesterol because cholesterol is in such short supply, and their delicate fats are therefore vulnerable to oxidation damage. This problem is further compounded by the reduction in coenzyme Q10, a potent antioxidant. The muscle cells are energy starved, due to dysfunctional mitochondria, and they try to compensate by processing an excessive amount of both fructose and glucose anaerobically, which causes extensive glycation damage to their crucial proteins. Their membranes are leaking ions, which interferes with their ability to contract, hindering movement. They are essentially heroic sacrificial lambs, willing to die in order to safeguard the heart.

Muscle pain and weakness are widely acknowledged, even by the statin industry, as potential side effects of statin drugs. Together with a couple of MIT students, I have been conducting a study which shows just how devastating statins can be to muscles and the nerves that supply them (Liu et al, 2011). We gathered over 8400 on-line drug reviews prepared by patients on statin therapy, and compared them to an equivalent number of reviews for a broad spectrum of other drugs. The reviews for comparison were selected such that the age distribution of the reviewers was matched against that for the statin reviews. We used a measure which computes how likely it would be for the words/phrases that show up in the two sets of reviews to be distributed in the way they are observed to be distributed, if both sets came from the same probability model.

For example, if a given side effect showed up a hundred times in one data set and only once in the other, this would be compelling evidence that this side effect was representative of that data set. Table 1 shows several conditions associated with muscle problems that were highly skewed towards the statin reviews.

Side Effect	# Statin Reviews		# Non-Statin Reviews	Associated P-value
Muscle Cramps	678	193	0.00005	
General Weakness	687	210	0.00006	
Muscle Weakness	302	45	0.00023	
Difficulty Walking	419	128	0.00044	
Loss of Muscle Mass	54	5	0.01323	
Numbness	293	166	0.01552	
Muscle Spasms	136	57	0.01849	

Table 1: Counts of the number of reviews where phrases associated with various symptoms related to muscles appeared, for 8400 statin and 8400 non-statin drug reviews, along with the associated p-value, indicating the likelihood that this distribution could have occurred by chance.

I believe that the real reason why statins protect the heart from a heart attack is that muscle cells are willing to make an incredible sacrifice for the sake of the larger good. It

is well acknowledged that exercise is good for the heart, although people with a heart condition have to watch out for overdoing it, walking a careful line between working out the muscles and overtaxing their weakened heart. I believe, in fact, that the reason exercise is good is exactly the same as the reason statins are good: it supplies the heart with lactate, a very healthy fuel that does not glycate cell proteins.

## **5. Membrane Cholesterol Depletion and Ion Transport**

As I alluded to earlier, statin drugs interfere with the ability of muscles to contract through the depletion of membrane cholesterol. (Haines, 2001) has argued that the most important role of cholesterol in cell membranes is the inhibition of leaks of small ions, most notably sodium ( $\text{Na}^+$ ) and potassium ( $\text{K}^+$ ). These two ions are essential for movements, and indeed, cholesterol, which is absent in plants, is the key molecule that permits mobility in animals, through its strong control over ion leakage of these molecules across cell walls. By protecting the cell from ion leaks, cholesterol greatly reduces the amount of energy the cell needs to invest in keeping the ions on the right side of the membrane.

There is a widespread misconception that "lactic acidosis," a condition that can arise when muscles are worked to exhaustion, is due to lactic acid synthesis. The actual story is the exact opposite: the acid build-up is due to excess breakdown of ATP to ADP to produce energy to support muscle contraction. When the mitochondria can't keep up with energy consumption by renewing the ATP, the production of lactate becomes absolutely necessary to prevent acidosis (Robergs et al., 2004). In the case of statin



therapy, excessive leaks due to insufficient membrane cholesterol require more energy to correct, and all the while the mitochondria are producing less energy.

In in vitro studies of phospholipid membranes, it has been shown that the removal of cholesterol from the membrane leads to a nineteen fold increase in the rate of potassium leaks through the membrane (Haines, 2001). Sodium is affected to a lesser degree, but still by a factor of three. Through ATP-gated potassium and sodium channels, cells maintain a strong disequilibrium across their cell wall for these two ions, with sodium being kept out and potassium being held inside. This ion gradient is what energizes muscle movement. When the membrane is depleted in cholesterol, the cell has to burn up substantially more ATP to fight against the steady leakage of both ions. With cholesterol depletion due to statins, this is energy it doesn't have, because the mitochondria are impaired in energy generation due to coenzyme-Q10 depletion.

Muscle contraction itself causes potassium loss, which further compounds the leak problem introduced by the statins, and the potassium loss due to contraction contributes significantly to muscle fatigue. Of course, muscles with insufficient cholesterol in their membranes lose potassium even faster. Statins make the muscles much more vulnerable to acidosis, both because their mitochondria are dysfunctional and because of an increase in ion leaks across their membranes. This is likely why athletes are more susceptible to muscle damage from statins (Meador and Huey, 2010, Sinzinger and O'Grady, 2004): their muscles are doubly challenged by both the statin drug and the exercise.

An experiment with rat soleus muscles in vitro showed that lactate added to the medium was able to almost fully recover the force lost due to potassium loss (Nielsen et al, 2001). Thus, production and release of lactate becomes essential when potassium is lost to the medium. The loss of strength in muscles supporting joints can lead to sudden uncoordinated movements, overstressing the joints and causing arthritis (Brandt et al., 2009). In fact, our studies on statin side effects revealed a very strong correlation with arthritis, as shown in the table.

While I am unaware of a study involving muscle cell ion leaks and statins, a study on red blood cells and platelets has shown that there is a substantial increase in the Na<sup>+</sup>-K<sup>+</sup> pump activity after just a month on a modest 10 mg/dl statin dosage, with a concurrent decrease in the amount of cholesterol in the membranes of these cells (Lohn et al., 2000). This increased pump activity (necessitated by membrane leaks) would require additional ATP and thus consume extra energy.

Muscle fibers are characterized along a spectrum by the degree to which they utilize aerobic vs anaerobic metabolism. The muscle fibers that are most strongly damaged by statins are the ones that specialize in anaerobic metabolism (Westwood et al., 2005). These fibers (Type IIb) have very few mitochondria, as contrasted with the abundant supply of mitochondria in the fully aerobic Type 1A fibers. I suspect their vulnerability is due to the fact that they carry a much larger burden of generating ATP to fuel the muscle contraction and to produce an abundance of lactate, a product of anaerobic metabolism. They are tasked with both energizing not only themselves but also the



defective aerobic fibers (due to mitochondrial dysfunction) and producing enough lactate to offset the acidosis developing as a consequence of widespread ATP shortages.

## **6. Long-term Statin Therapy Leads to Damage Everywhere**

Statins, then, slowly erode the muscle cells over time. After several years have passed, the muscles reach a point where they can no longer keep up with essentially running a marathon day in and day out. The muscles start literally falling apart, and the debris ends up in the kidney, where it can lead to the rare disorder, rhabdomyolysis, which is often fatal. In fact, 31 of our statin reviews contained references to "rhabdomyolysis" as opposed to none in the comparison set. Kidney failure, a frequent consequence of rhabdomyolysis, showed up 26 times among the statin reviews, as opposed to only four times in the control set.

The dying muscles ultimately expose the nerves that innervate them to toxic substances, which then leads to nerve damage such as neuropathy, and, ultimately Amyloid Lateral Sclerosis (ALS), also known as Lou Gehrig's disease, a very rare, debilitating, and ultimately fatal disease which is now on the rise due (I believe) to statin drugs. People diagnosed with ALS rarely live beyond five years. Seventy-seven of our statin reviews contained references to ALS, as against only 7 in the comparison set.

As ion leaks become untenable, cells will begin to replace the potassium/sodium system with a calcium/magnesium based system. These two ions are in the same rows of the periodic table as sodium/potassium, but advanced by one column, which means that they are substantially larger, and therefore it's much harder for them to accidentally



leak out. But this results in extensive calcification of artery walls, heart valves, and the heart muscle itself. Calcified heart valves can no longer function properly to prevent backflow, and diastolic heart failure results from increased left ventricular stiffness. Research has shown that statin therapy leads to increased risk to diastolic heart failure (Silver et al., 2004, Weant and Smith, 2005). Heart failure shows up 36 times in our statin drug data as against only 8 times in the comparison group.

Once the muscles can no longer keep up with lactate supply, the liver and heart will be further imperilled. They're now worse off than they were before statins, because the lactate is no longer available, and the LDL, which would have provided fats as a fuel source, is greatly reduced. So they're stuck processing sugar as fuel, something that is now much more perilous than it used to be, because they are depleted in membrane cholesterol. Glucose entry into muscle cells, including the heart muscle, mediated by insulin, is orchestrated to occur at lipid rafts, where cholesterol is highly concentrated. Less membrane cholesterol results in fewer lipid rafts, and this leads to impaired glucose uptake. Indeed, it has been proposed that statins increase the risk to diabetes (Goldstein and Mascitelli, 2010, Hagedorn and Arora, 2010). Our data bear out this notion, with the probability of the observed distributions of diabetes references happening by chance being only 0.006.

Side Effect	# Statin Reviews	# Non-Statin Reviews	Associated P-value
Rhabdomyolysis	31	0	0.02177
Liver Damage	326	133	0.00285

Diabetes	185	62	0.00565
ALS	71	7	0.00819
Heart Failure	36	8	0.04473
Kidney Failure	26	4	0.05145
Arthritis	245	120	0.01117
Memory Problems	545	353	0.01118
Parkinson's Disease	53	3	0.01135
Neuropathy	133	73	0.04333
Dementia	41	13	0.05598

Table 2: Counts of the number of reviews where phrases associated with various symptoms related to major health issues appeared, besides muscle problems, for 8400 statin and 8400 non-statin drug reviews, along with the associated p-value, indicating the likelihood that this distribution could have occurred by chance.

## 7. Statins, Caveolin, and Muscular Dystrophy

Lipid rafts are crucial centers for transport of substances (both nutrients and ions) across cell membranes and as a cell signaling domain in essentially all mammalian cells. Caveolae ("little caves") are microdomains within lipid rafts, which are enriched in a



substance called caveolin (Gratton et al., 2004). Caveolin has received increasing attention of late due to the widespread role it plays in cell signaling mechanisms and the transport of materials between the cell and the environment (Smart et al., 1999).

Statins are known to interfere with caveolin production, both in endothelial cells (Feron et al., 2001) and in heart muscle cells, where they've been shown to reduce the density of caveolae by 30% (Calaghan, 2010). People who have a defective form of caveolin-3, the version of caveolin that is present in heart and skeletal muscle cells, develop muscular dystrophy as a consequence (Minetti et al., 1998). Mice engineered to have defective caveolin-3 that stayed in the cytoplasm instead of binding to the cell wall at lipid rafts exhibited stunted growth and paralysis of their legs (Sunada et al., 2001). Caveolin is crucial to cardiac ion channel function, which, in turn, is essential in regulating the heart beat and protecting the heart from arrhythmias and cardiac arrest (Maguy et al., 2006). In arterial smooth muscle cells, caveolin is essential to the generation of calcium sparks and waves, which, in turn, are essential for arterial contraction and expansion, to pump blood through the body (Taggart et al., 2010).

In experiments involving constricting the arterial blood supply to rats' hearts, researchers demonstrated a 34% increase in the amount of caveolin-3 produced by the rat's hearts, along with a 27% increase in the weight of the left ventricle, indicating ventricular hypertrophy. What this implies is that the heart needs additional caveolin to cope with blocked vessels, whereas statins interfere with the ability to produce extra caveolin (Kikuchi et al., 2005).

## **8. Statins and the Brain**

While the brain is not the focus of this essay, I cannot resist mentioning the importance of cholesterol to the brain and the evidence of mental impairment available from our data sets. Statins would be expected to have a negative impact on the brain, because, while the brain makes up only 2% of the body's weight, it houses 25% of the body's cholesterol. Cholesterol is highly concentrated in the myelin sheath, which encloses axons which transport messages long distances (Saher et al., 2005). Cholesterol also plays a crucial role in the transmission of neurotransmitters across the synapse (Tong et al, 2009). We found highly skewed distribution of word frequencies for dementia, Parkinson's disease, and short term memory loss, with all of these occurring much more frequently in the statin reviews than in the comparison reviews.

A recent evidence-based article (Cable, 2009) found that statin drug users had a high incidence of neurological disorders, especially neuropathy, parasthesia and neuralgia, and appeared to be at higher risk to the debilitating neurological diseases, ALS and Parkinson's disease. The evidence was based on careful manual labeling of a set of self-reported accounts from 351 patients. A mechanism for such damage could involve interference with the ability of oligodendrocytes, specialized glial cells in the nervous system, to supply sufficient cholesterol to the myelin sheath surrounding nerve axons. Genetically-engineered mice with defective oligodendrocytes exhibit visible pathologies in the myelin sheath which manifest as muscle twitches and tremors (Saher et al, 2005). Cognitive impairment, memory loss, mental confusion, and depression were also

significantly present in Cableâ€™s patient population. Thus, his analysis of 351 adverse drug reports was largely consistent with our analysis of 8400 reports.

## **9. Cholesterol's Benefits to Longevity**

The broad spectrum of severe disabilities with increased prevalence in statin side effect reviews all point toward a general trend of increased frailty and mental decline with long-term statin therapy, things that are usually associated with old age. I would in fact best characterize statin therapy as a mechanism to allow you to grow old faster. A highly enlightening study involved a population of elderly people who were monitored over a 17 year period, beginning in 1990 (Tilvis et al., 2011). The investigators looked at an association between three different measures of cholesterol and manifestations of decline. They measured indicators associated with physical frailty and mental decline, and also looked at overall longevity. In addition to serum cholesterol, a biometric associated with the ability to synthesize cholesterol (lathosterol) and a biometric associated with the ability to absorb cholesterol through the gut (sitosterol) were measured.

Low values of all three measures of cholesterol were associated with a poorer prognosis for frailty, mental decline and early death. A reduced ability to synthesize cholesterol showed the strongest correlation with poor outcome. Individuals with high measures of all three biometrics enjoyed a 4.3 year extension in life span, compared to those for whom all measures were low. Since statins specifically interfere with the



ability to synthesize cholesterol, it is logical that they would also lead to increased frailty, accelerated mental decline, and early death.

For both ALS and heart failure, survival benefit is associated with elevated cholesterol levels. A statistically significant inverse correlation was found in a study on mortality in heart failure. For 181 patients with heart disease and heart failure, half of those whose serum cholesterol was below 200 mg/dl were dead three years after diagnosis, whereas only 28% of the patients whose serum cholesterol was above 200 mg/dl had died. In another study on a group of 488 patients diagnosed with ALS, serum levels of triglycerides and fasting cholesterol were measured at the time of diagnosis (Dorstand et al., 2010). High values for both lipids were associated with improved survival, with a p-value < 0.05.

## **10. What to do Instead to Avoid Heart Disease**

If statins don't work in the long run, then what can you do to protect your heart from atherosclerosis? My personal opinion is that you need to focus on natural ways to reduce the number of small dense LDL particles, which feed the plaque, and alternative ways to supply the product that the plaque produces (more about that in a moment). Obviously, you need to cut way back on fructose intake, and this means mainly eating whole foods instead of processed foods. With less fructose, the liver won't have to produce as many LDL particles from the supply side. From the demand side, you can reduce your body's dependency on both glucose and fat as fuel by simply eating foods that are good sources of lactate. Sour cream and yogurt contain lots of lactate, and milk



products in general contain the precursor lactose, which gut bacteria will convert to lactate, assuming you don't have lactose intolerance. Strenuous physical exercise, such as a tread machine workout, will help to get rid of any excess fructose and glucose in the blood, with the skeletal muscles converting them to the much coveted lactate.

Finally, I have a set of perhaps surprising recommendations that are based on research I have done leading to the two papers that are currently under review (Seneff<sup>3</sup> et al, Seneff<sup>4</sup> et al.). My research has uncovered compelling evidence that the nutrient that is most crucially needed to protect the heart from atherosclerosis is cholesterol sulfate. The extensive literature review my colleagues and I have conducted to produce these two papers shows compellingly that the fatty deposits that build-up in the artery walls leading to the heart exist mainly for the purpose of extracting cholesterol from glycated small dense LDL particles and synthesizing cholesterol sulfate from it, providing the cholesterol sulfate directly to the heart muscle. The reason the plaque build-up occurs preferentially in the arteries leading to the heart is so that the heart muscle can be assured an adequate supply of cholesterol sulfate. In our papers, we develop the argument that the cholesterol sulfate plays an essential role in the caveolae in the lipid rafts, in mediating oxygen and glucose transport.

The skin produces cholesterol sulfate in large quantities when it is exposed to sunlight. Our theory suggests that the skin actually synthesizes sulfate from sulfide, capturing energy from sunlight in the form of the sulfate molecule, thus acting as a solar-powered battery. The sulfate is then shipped to all the cells of the body, carried on the back of the cholesterol molecule.





Evidence of the benefits of sun exposure to the heart is compelling, as evidenced by a study conducted to investigate the relationship between geography and cardiovascular disease (Grimes et al., 1996). Through population statistics, the study showed a consistent and striking inverse linear relationship between cardiovascular deaths and estimated sunlight exposure, taking into account percentage of sunny days as well as latitude and altitude effects. For instance, the cardiovascular-related death rate for men between the ages of 55 and 64 was 761 in Belfast, Ireland but only 175 in Toulouse, France.

Cholesterol sulfate is very versatile. It is water soluble so it can travel freely in the blood stream, and it enters cell membranes ten times as readily as cholesterol, so it can easily resupply cholesterol to cells. The skeletal and heart muscle cells make good use of the sulfate as well, converting it back to sulfide, and synthesizing ATP in the process, thus recovering the energy from sunlight. This decreases the burden on the mitochondria to produce energy. The oxygen released from the sulfate molecule is a safe source of oxygen for the citric oxide cycle in the mitochondria.

So, in my view, the best way to avoid heart disease is to assure an abundance of an alternative supply of cholesterol sulfate. First of all, this means eating foods that are rich in both cholesterol and sulfur. Eggs are an optimal food, as they are well supplied with both of these nutrients. But secondly, this means making sure you get plenty of sun exposure to the skin. This idea flies in the face of the advice from medical experts in the United States to avoid the sun for fear of skin cancer. I believe that the excessive use of sunscreen has contributed significantly, along with excess fructose consumption, to the



current epidemic in heart disease. And the natural tan that develops upon sun exposure offers far better protection from skin cancer than the chemicals in sunscreens.

## **11. Concluding Remarks**

Every individual gets at most only one chance to grow old. When you experience your body falling apart, it is easy to imagine that this is just due to the fact that you are advancing in age. I think the best way to characterize statin therapy is that it makes you grow older faster. Mobility is a great miracle that cholesterol has enabled in all animals. By suppressing cholesterol synthesis, statin drugs can destroy that mobility. No study has shown that statins improve all-cause mortality statistics. But there can be no doubt that statins will make your remaining days on earth a lot less pleasant than they would otherwise be.

To optimize the quality of your life, increase your life expectancy, and avoid heart disease, my advice is simple: spend significant time outdoors; eat healthy, cholesterol-enriched, animal-based foods like eggs, liver, and oysters; eat fermented foods like yogurt and sour cream; eat foods rich in sulfur like onions and garlic. And finally, say "no, thank-you" to your doctor when he recommends statin therapy.

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