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Statins: Did Your Doctor Tell You . . . ? **(the longer version)**

NOTE: Nothing in this article should be construed as medical advice. It is informational in purpose only and taken from numerous readily available articles written by physicians and researchers. For medical advice consult with an ***informed*** physician.

This is the information I would want any friend or family member on statins to be told by their physician. Extensive endnotes included. Don't just take my word for this stuff: your life and health may depend on it.

What are statins?

Statins, also called HMG-CoA reductase inhibitors, are a class of drugs that are designed to interfere with the biosynthesis of cholesterol. They operate by interfering with one of the first steps in cholesterol synthesis: unfortunately they also inhibit the production of other intermediary substances needed by our body.¹

A study of 10 major trials of statins concludes . . .

“Do Statins Have a Role in Primary Prevention” is a review of 10 major statin trials conducted by the Therapeutics Initiative of the Department of Pharmacology & Therapeutics of the University of British Columbia. Here are their conclusions²:

- “If cardiovascular serious adverse events are viewed in isolation, 71 primary prevention patients with cardiovascular risk factors have to be treated with a statin for 3 to 5 years to prevent one myocardial infarction or stroke.”
- “This cardiovascular benefit is not reflected in 2 measures of overall health impact, total mortality and total serious adverse events. Therefore, statins have not been shown to provide an overall health benefit in primary prevention trials.”

In plain English, the study says that if you are taking statins to prevent myocardial infarction (“heart attack”) or stroke:

- Only 1 of 71 people (1.4%) will have a heart attack or stroke prevented every 3-5 years. [So, yes, statins do provide some protection against heart attacks.]
- Despite protecting 1 person in 71, the death rate of those taking statins was just as high as those not taking statins: as a group, there was no increase in longevity.

By taking statins you are betting that you will be that 1 person in 71 who benefits, that the statins won't cause you to die by some other means and that any adverse effects caused by the drug (see below) will be not be too severe.

Joel M. Kaufman tells us this result of studies before 2000³:

Long-term use of statins for primary prevention of heart disease produced a 1% greater risk of death over 10 years vs. placebo when the results of all the big controlled trials reported before 2000 were combined (Jackson PR et al. Statins for primary prevention: at what coronary risk is safety assured? Br J Clin Pharmacol 2001;52:439-46).

A note on relative vs. absolute risk

The statistics your doctor tells you about statins may sound different than the “1 in 71” figure above. Dr. Paul Rosch gives three ways to report the same result⁴:

1. “Over five years, patients taking this drug had 34% fewer heart attacks compared to controls who took a placebo. (Sounds pretty convincing)”

2. “Over five years only 2.7% of patients taking this drug had a heart attack compared to 4.1% taking a placebo. (Also not too bad)”
3. “If seventy-one people take this drug every day for five years it will prevent one of them from having a heart attack. However, there is no guarantee that you will be that person. (These odds are not very attractive)”

Item 1 uses “relative risk,” item 2 uses “absolute risk” and item 3 tells you what the statistics really mean. (“Numbers never lie but you can lie with numbers.”)

If you are a woman, elderly, or a man at lower cardiac risk . . .

According to Beatrice A. Golomb, MD, PhD, the leading researcher for the National Institutes of Health’s study of the “side effects” of statins and a firm believer that statins can save lives:

However benefit to survival with statins or other cholesterol-lowering agents *has never been demonstrated* in women (even those at high cardiac risk), in the older elderly, or in men at lower cardiac risk . . .⁵ [emphasis added]

Let me repeat that: statins have *never* been shown to provide survival benefit for women, the older elderly, or men at lower cardiac risk. *Never!* No benefit ever shown for women at high risk for a heart attack. *Never!*

The next Hormone Replacement Therapy (HRT)?

There are no long-term studies of statins: they have not been around long enough. This is reminiscent of Hormone Replacement Therapy (HRT), which was widely prescribed in the absence of any long-term studies on safety. In 2001 there were 100 million women worldwide taking HRT. HRT was widely promoted not only as treatment for menopausal symptoms but also as long-term preventive medicine for osteoporosis, heart disease and dementia; it was also said to increase overall vitality and enhance sexual function. This was before 2003, when evidence began to show that long term HRT use increases women’s risk of heart attacks, strokes, blood clots, breast cancer and dementia.⁶

Many of the same people who were pushing HRTs are now pushing statins, apparently unconcerned about the ignorance of the long-term consequences. And, just as with HRT, statins are being touted as preventative of other conditions than heart disease without any good evidence. At what point in the future will there be front-page articles revealing adverse effects of statins that the doctors never considered?

If you are taking statins, you are volunteering to test the long-term effects of an unknown drug that disrupts the mechanism of one of the most critical components of the body (cholesterol: see below).

Coenzyme Q10 (Ubiquinone, or CoQ10) depletion.

Why is CoQ10 important?

Ubiquinone, or Coenzyme Q10, commonly referred to as CoQ10 is a “vitaminlike” substance – it has the properties of a vitamin, can be obtained from foods (mostly animal foods), but is also produced in our bodies by biosynthesis.⁷ Its name “ubiquinone” indicates its “ubiquitous nature” (meaning everywhere present) – it is found in every living cell.

CoQ10 has a number of critical functions in our body:

- It is essential for all cellular ATP production (occurring in the mitochondria, the power plants of the cell, indeed, of the body). ATP is what our cells use for energy. “Without coenzyme Q-10, mitochondrial respiration would be unable to function, and energy production would be minimal.”⁸

- CoQ10 is particularly important in heart muscle function – the heart uses a lot of it.⁹
- It is a potent anti-oxidant (free radical scavenger) especially important in diminishing the oxidation of LDL cholesterol.¹⁰
- It is found in all cell membranes and is vital for maintaining membrane integrity.¹¹
- It is critical to the formation of elastin and collagen (found in connective tissues such as tendons and ligaments.)¹²

Statins deplete CoQ10 in the body.

That statins deplete CoQ10 in the body is widely known; information about this was first published in 1990.¹³ Statins work by interrupting the process of the biosynthesis of cholesterol and “. . . the biochemical pathway for CoQ10 synthesis is a branch of the same pathway where cholesterol is made.”¹⁴ One pharmaceutical company (Merck) has a patent on a drug combining statins and CoQ10 in one dose.¹⁵ In Canada a precaution is included in the prescribing information for statins.¹⁶

A review of studies on the depletion of coenzyme Q10 by Peter H. Langsjoen, M.D., F.A.C.C. says:

Statin-induced decreases in CoQ10 are more than just hypothetical drug-nutrient interactions. Good evidence exists of significant CoQ10 depletion in humans and animals during statin therapy.¹⁷

Langsjoen observes that all statins deplete “both the blood levels and the cellular concentrations of Q10.” A higher dose will produce greater depletion of CoQ10. One problem is that the depletion can be gradual over years making it hard to tie an adverse effect three years (for instance) after starting statins back to the drug. This depletion will be most dangerous in the elderly, for as we age our levels of CoQ10 decrease.¹⁸

Perhaps most important, supplemental CoQ10 can completely reverse statin-induced CoQ10 depletion.¹⁹

NOTE: Of the 9 people I know taking statin drugs, only one was initially informed by their physician that statins deplete CoQ10. A second friend was later told by a different physician, whom he was seeing to deal with adverse effects from taking statins.

Possible effects of depleted CoQ10 in the body.

- Congestive heart failure.²⁰ (Ironic, no? Statins, prescribed to prevent heart attacks, may precipitate congestive heart failure by depleting the body of CoQ10.)
- Fatigue, muscle weakness and soreness.²¹ (See below under polyneuropathy.)
- Muscle and cell breakdown and nerve conduction defects.²² (See below under polyneuropathy.)
- Cancer (See below, under Cancer.)

Known “side effects” of statins.

Note: “Side effect” is a term used by physicians and drug manufacturers to describe an undesirable effect of the drug that they wish did not exist. In truth, *any* effect of a drug results from its designed mechanism. The human body is extremely complex: interfering with one of its critical mechanisms is almost certain to have unforeseen consequences, be they trivial or major.

Theoretically the Food and Drug Administration (FDA) is protecting you from drugs with adverse reactions. Baycol was a statin drug that was recalled after 31 deaths in the United States, 50 worldwide: each of those deaths was reported over a period of 2 years before the drug was recalled.²³

According to the petition filed by Dr. Julian M. Whitaker, M.D. with the FDA to mandate a warning about CoQ10 depletion with all statins, the Physicians' Desk Reference (PDR) estimates .5% to 2.3% of patients using statins experience adverse events.²⁴ We can compare this to the number of heart attacks and strokes prevented (1 in 71), which is 1.4%.²⁵

Note: "The adverse effects of statin drugs increase as the dosage increases."²⁶

Most common problems

There are many possible undesirable effects of statin drugs:

The most common problems we hear reported pertain to muscle pain or weakness, fatigue, memory and cognitive problems, sleep problems, and neuropathy. Erectile dysfunction, problems with temperature regulation (feeling hot or cold, or having sweats), are among the other problems reported.²⁷

Other effects include personality changes and irritability.²⁸

Cognitive problems

These include:

- memory loss, from
 - poor memory, to
 - mild memory impairment, to
 - global amnesia;
- confusion and disorientation.

According to Dr. Duane Graveline, author of a forthcoming book entitled *Lipitor, Thief of Memory*:

Total Global Amnesia, once so rare that most physicians have never seen a case in their entire careers, is now quite common in our emergency rooms associated with simply being on a statin drug. But amnesia is only the tip of the iceberg. For every amnesia case report there are hundreds if not thousands of cases of confusion and severe memory disturbance associated with being on statin drugs.²⁹

Graveline says that, over a period of two years, hundreds of reports of cognitive "side effects" have been passed onto FDA's Medwatch program, as well as the pharmaceutical companies' reporting system for adverse reactions to drugs: no action has been taken. He says:

Personally, I think statin drugs should be withheld from military flight personnel until further study demonstrates their complete safety with regard to brain function.³⁰

Polyneuropathy³¹

The Colorado Health Site defines polyneuropathy thus (these are all possible effects from taking statins):

Polyneuropathy A disorder that involves the slow progressive (or recurrent) inflammation of multiple nerves. Loss of movement and sensation are common findings.

Some symptoms that may be associated with this disease include:

- facial weakness
- difficulty walking
- difficulty using the arms and hands or legs and feet
- sensation changes (usually of the arms and hands or legs and feet), such as pain, burning, tingling, numbness, or decreased sensation

- swallowing difficulty
- speech impairment
- loss of muscle function or feeling in the muscles
- joint pain
- hoarseness or changing voice
- fatigue

They report the following result of a study on statins and the risk of polyneuropathy:

The authors note that their study showed that long-term exposure to statins may substantially increase the risk of polyneuropathy. These findings suggest that statins may have a toxic effect on peripheral nerves. One possible mechanism may be that by interfering with cholesterol synthesis, statins may alter nerve membrane function.³²

Other statin-associated muscle problems³³

According to the Colorado Health Site, statin drugs are associated with:

- *myopathy* (any disease of the muscles; symptoms include weakness of limbs) – can be progressive, severely disabling, may result in severe renal failure and can be fatal. A standard blood test (CK Blood test) may be inadequate in diagnosing it.³⁴
- *myositis* – which involves chronic or persistent muscle inflammation at the hips, shoulders, one arm, one leg, or even muscles that move the eye. It may be associated with inflammation in organs such as the joints, intestines, skin, heart or lungs. There is no cure. This condition can occasionally progress to:
- *Rhabdomyolysis* – where the kidney becomes injured due to toxic effect of muscle cell contents. Symptoms include muscle weakness up to acute kidney failure. (It is this condition that caused the deaths attributed to Baycol, a statin drug made by Bayer that was pulled off the market after over 50 deaths worldwide, 31 in the U.S.³⁵)
- *Myalgia* – muscle pain or discomfort.

It is noted that “Patients should be instructed on the importance of discontinuing the medication and promptly reporting unexpected muscle pain or weakness or dark discoloration of urine” and that prevention, the best approach, may involve using the lowest possible statin dose.

Insulin rise

According to Beatrice Golomb, some people have reported “dramatic” increases in blood sugar levels on statins, that stop when the statins are discontinued. She mentions this is not among the more common adverse effects reported and speculates that its cause might be the reduction in coenzyme Q10.³⁶

Cancer

One review found statin drugs stimulate cancer growth in rodents.³⁷ One statin trial (CARE) found breast cancer in 12 women in the treatment group and only one in the control group while another trial (PROSPER) saw a “significant increase” in cancer for people ages 70 to 82.³⁸ Here is a reference to another study:

[S.] Sinatra also pointed out that an increase in cancer rates has been observed in those taking cholesterol-lowering drugs. The relationship is all too clear: When the function of the mitochondria is disrupted, cancerous cells are more likely to emerge.³⁹

So what is this “cholesterol” that we are trying to lower?

Cholesterol is a substance that is absolutely critical to the correct functioning and health of the body. It is an alcohol rather than a fat or lipid, although it does not behave like an alcohol. Insoluble in water (as are lipids or fats), cholesterol is transported in the body in lipoproteins – these lipoproteins also are carrying fatty acids. The best known are HDL (High Density Lipoprotein) – which mostly carry cholesterol from the peripheral tissues to the liver – and LDL (Low Density Lipoprotein) – which mostly carry cholesterol in the opposite direction. About 15 to 20% of our cholesterol is transported by the HDL.⁴⁰

About 80% of the cholesterol our body needs is made in the liver. If we do not get enough from our diet, the liver will make more cholesterol to accommodate.

The many functions of cholesterol⁴¹

Cholesterol is one of the most vital and necessary substances in the body. It is the basis of several hormones essential to life. Without cholesterol, we would not exist. In fact, it is so important that every cell of the body (except the brain cells) has the ability to make it.⁴² Here are some of its essential functions. Cholesterol is:

- found in the membrane of *every* cell in our body where it adjusts fluid level and rigidity: it is necessary for the proper stability and functioning of every cell in our body. Essentially, it makes cells waterproof and allows them to function.
- the precursor to the sex hormones; it is our sole source for estrogen, progesterone, and androgen. No cholesterol, no sex hormones, no humans.
- the precursor to the two steroid hormones created in the adrenal glands: aldosterone (which protects from loss of water and sodium) and cortisol (important for glucose metabolism and in responding to stress).
- the precursor to calcitriol, which maintains the proper level of calcium in our body (necessary to prevent osteoporosis, to name only one function).
- the precursor to vitamin D (vital for bone health and hormone production) and to the bile salts (necessary for assimilation of fats from the diet).
- needed for proper function of serotonin receptors in the brain; serotonin is necessary for our well-being and many anti-depressants seek to increase the serotonin available to us.
- necessary for developing the synapses (contact sites of adjacent neurons) of the brain; the largest concentration of cholesterol in the body is in the brain and other parts of the nervous system.
- necessary for maintaining the health of the intestinal wall.
- used by the body to repair tears in tissue; found in high levels in scar tissue and tears in arterial wall.
- an antioxidant protecting against free radical damage (which may explain why cholesterol levels go up with age).

Given this incredible number of critically important functions dependant on cholesterol, does it really make any sense to go in and attempt to interfere with its production? ***Cholesterol is needed for the proper functioning of every cell of our body.*** Aren't adverse effects from meddling with its production inevitable? When we mess with something so complex and integral to life there have to be unintended consequences.

One interesting fact is the level of cholesterol in our blood goes up when we are under stress. This seems to indicate that cholesterol is part of our body's mechanism for dealing with and mitigating

the effects of stress. Its role as an antioxidant seems to indicate another protective function. If cholesterol is used in protecting the body, wouldn't it naturally occur at the site of tears in the artery as the cholesterol attempts to repair the tear? As Sally Fallon & Mary Enig write: "Blaming heart disease on high serum cholesterol levels is like blaming firemen who have come to put out a fire for starting the blaze."⁴³

And if cholesterol *is* protective, a raised level would indicate that the body is in need of protecting. Do we really want to go in and lower something that may be the very thing helping our body to heal?

Dangers of low blood cholesterol

The following conditions have been linked with low blood cholesterol⁴⁴:

- depression and suicide, as well as aggressive and violent behavior⁴⁵
- cognitive impairment
- suppression of the immune system
- colon cancer
- possible relationship to Alzheimer's Disease
- increase in strokes
- One study showed that mortality is higher for women with low cholesterol than for women with high cholesterol."⁴⁶
- Under certain conditions – LDL levels below 130 may *increase* the chance of heart disease.⁴⁷ In at least one study, the Framingham study, as reported in 1987, coronary heart disease rates went up as cholesterol levels went down.⁴⁸

As Dr. Duane Graveline reminds us:

We can be certain only that we do not yet know the long-term consequences of artificially lowered serum cholesterol through the use of statin drugs.⁴⁹

Cholesterol and heart disease

Cholesterol is not a cause of heart disease?

The standard explanation for heart disease is that dietary saturated fat and cholesterol lead to raised cholesterol levels in the blood and that these raised levels cause plaques (atheromas) that block blood vessels leading directly to heart attacks.⁵⁰

A growing number of physicians and researchers question this notion. Under certain circumstances and for certain age groups raised cholesterol *is* a risk factor (often a small one) for heart disease – but so are roughly 300 other things (including being male and high selenium toenail levels).⁵¹ One writer facetiously suggests that since a deep ear lobe crease is a risk factor for coronary artery disease, we should probably cut off our ear lobes.⁵² It makes as much sense as lowering cholesterol.

If raised cholesterol is merely a risk factor or marker for heart disease, removing the risk factor will do nothing about the underlying disease – in order to successfully treat the disease you must reduce or remove its cause.⁵³ Reducing cholesterol merely alleviates a symptom and, at the same time, affects numerous processes in the body that are dependent on cholesterol.

Part of the conventional strategy to prevent heart attacks is to reduce the intake of foods containing saturated fats and/or cholesterol in order to lower blood cholesterol. One writer points out the physiological impossibility of saturated fat (or any fat) turning into cholesterol because chemically they are unrelated. (For one thing, cholesterol contains nitrogen atoms, which come from protein.)⁵⁴

The same author points out the absurdity of saying that eating cholesterol causes cholesterol to rise: it is like saying that if you eat too much protein your blood level protein will rise. Many well-known studies absolutely do not support the hypothesis.⁵⁵ Another problem is that the majority of the 1,000 mg or so per day of cholesterol needed by the body is made in the liver and does not come from diet: if there is not enough in the diet, the liver simply makes more cholesterol.

One of the biggest problems with the whole standard theory is that people with normal and even low levels of cholesterol die from coronary heart disease. I think this needs repeating. High cholesterol is said to cause heart disease but people without high cholesterol die from heart disease. Do you think there is a flaw somewhere here?⁵⁶

A wealth of physicians and researchers review all of the literature and point out that there is no real evidence for the theory at the same time there is evidence to the contrary.⁵⁷ Two (of many) examples⁵⁸:

- Multiple Risk Factor Intervention (MRFIT) did find that annual heart disease deaths were 1 per 1,000 for cholesterol levels of 180 but rose to a bit less than two deaths per 1,000 for cholesterol levels of 300. This was an actual increase in rate of less than .1% (although proponents of the cholesterol-heart disease theory will tell you that it is a 100% increase in risk: they prefer relative risk here to absolute risk because it strengthens their case). Unfortunately, there was an increase in total deaths for cholesterol levels below 160. This study involved 362,000 men.
- In 1992, After 44 years of research for the Framingham Study, there was virtually no difference in heart disease between people with cholesterol levels of 182 and 244. (A 240% increase in “risk” for coronary heart disease was cited – this amounted to an absolute risk of .13%). Between cholesterol levels of 244 and 294 the rate of CHD actually declined. According to Dr. William Castelli, the director of the project:

In Framingham, Massachusetts, the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower people's serum cholesterol...we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories weighed the least and were the most physically active.

But statins reduce deaths from heart attack.

Many doctors and researchers believe that the reduction in mortality from coronary heart disease (recall the 1 in 71 figure) due to statins does not come from lowering cholesterol – it possibly comes from an anti-inflammatory effect.⁵⁹

This is important because it means the drug is doing something it's designers did not intend. When we interfere with one small mechanism of the incredibly complex human body, especially for a substance needed by every cell of the body, there almost certainly *will* be unintended consequences and effects elsewhere; they are just as likely to be adverse as benign.

Just as with Hormone Replacement Therapy, drug companies have been touting these unanticipated effects as bonuses in preventing other diseases and as a reason to more widely prescribe statins. Prudence would seem to dictate that we be extremely careful about taking any drug until *all* of its effects are known, accounted for and thoroughly tested. I, personally, am very uncomfortable with this indication that drug companies are interfering with something that they don't thoroughly understand and expecting patients to pay them for the privilege of long-term testing.

What DOES cause heart disease?

Some current thinking & theories

In considering coronary heart disease, one researcher points out that the real problem is not “atherosclerosis” – the thickening of the arteries. For the most part, arteries can safely thicken unless there is a rupture in the artery wall which causes “plaque” (an obstruction) to develop, causing conditions such as angina. When these plaques break loose, a clot can develop. A plaque is not enough – you also need the blood clot. It would appear that the usual process leading to death is damage to the artery wall, development of a plaque causing a blockage and development of a clot.⁶⁰ However, it is also possible for a blood clot to cause death in the absence of any plaque.

So what causes the plaques to develop and what causes an increase in the possibility of a blood clot? Some physicians are pointing to “metabolic syndrome” as the culprit. Metabolic syndrome develops because of abnormal cortisol levels, which leads to insulin resistance and a number of metabolic abnormalities such as raised sugar levels, low HDL levels and raised triglycerides. Raised cortisol levels can come about from depression, use of certain drugs (steroids, for one), and stress. According to one doctor, “It is likely the most common cause of metabolic syndrome is chronic stress. . .”⁶¹

Stress causes cortisol to rise. No wonder cholesterol rises under stress: it is a precursor to the cortisol, its rise an effect, not a cause. Note the irony: how much stress are all of us subjected to by worrying about our cholesterol levels?

One study found that metabolic syndrome predicted heart disease independently of the usual risk factors (including high LDL cholesterol levels). Men with metabolic syndrome had a 76% greater risk of heart attack than those without the syndrome.⁶²

One theory points to the fact that excess levels of the amino acid homocysteine in the blood can help cause LDL (low-density lipoproteins) to adhere to arterial tissue, building up plaques. Deficiencies in vitamins B6, B12 and folic acid can cause this condition.⁶³

Inflammation can also cause blockages and can cause plaques to rupture, thus provoking a heart attack-causing clot. The vitamins that may help to reduce heart disease (conclusive proof is said to be lacking) include vitamins A, D and E (all fat-soluble vitamins) and vitamins C, folic acid, B6 and B12. Deficiencies of certain minerals may cause heart disease, namely magnesium, selenium, possibly copper and zinc. Other nutrients that might help are coenzyme Q10 and the Omega-3 essential fatty acids. Note that the best sources for many of these nutritional substances are meat and saturated fat.

Overconsumption of certain foods might also help cause heart disease: for instance, too much polyunsaturated oil.⁶⁴ Sugar might also be a culprit, both by increasing adhesiveness of blood platelets (making them more prone to clotting) and by increasing blood corticosteroid (a stress hormone) levels.⁶⁵ Sugar may further be implicated because consuming it in quantity can help cause insulin-resistance; the “carbohydrate theory of arteriosclerosis” notes that an increase in catabolic hormones (that cause substances to break down into simpler ones, as opposed to anabolic, which build molecules and tissue) in the bloodstream due to insulin resistance can damage arteries. There is some evidence that low-carbohydrate diets are preventative of heart disease.⁶⁶

Possibilities on how to prevent heart disease

Although these are not currently part of mainstream medical thinking, here are a number of suggestions from two different articles, one by Malcolm Kendrick and the other by Sally Fallon and Mary G. Enig, about how to protect yourself from heart disease.⁶⁷ Both articles agree on the following:

- Don't smoke.
- Exercise regularly.

- Avoid being overweight.

Both agree that you should reduce stress, although they word it differently:

- Don't work too hard and do something you love daily (Fallon/Enig).
- "If you feel 'trapped' in your life, change it" (Kendricks).
- Avoid exposure to chemicals, pollutants and pesticides, as much as possible. (Fallon/Enig).

Kendrick also stresses:

- You need a good social network.

Enig and Fallon, focus on nutrition:

- Avoid processed foods, especially those with sugar, white flour, hydrogenated fats, polyunsaturated vegetable oils or additives.
- Eat high-quality animal products, including seafood, organ meats.
- Eat a variety of fresh fruits and vegetables, organically grown if possible.
- Eat nutrient dense foods such as whole dairy products, bone broths, and whole grains, properly prepared to reduce factors that block mineral absorption.
- "Supplement the diet with foods rich in protective factors including small amounts of cod liver oil (vitamins A and D); wheat germ oil (vitamin E); flax oil (omega-3 fatty acids); kelp (iodine); brewers yeast (B vitamins); desiccated liver (vitamin B12); rose hip or acerola powder (vitamin C); and coconut oil (antimicrobial fatty acids)." (Fallon & Enig)

A suggestion

Please don't take my word on all this. Go to the sources in the endnotes and read for yourself.

Conclusions

I think that the current enthusiasm of the medical world for statin drugs should give all of us pause. The parallels with Hormone Replacement Therapy are quite clear: in the absence of proof of long-term safety, a drug is widely prescribed to tens of millions of patients.

Particularly disturbing is the fact that by depleting CoQ10 these drugs interfere with at least one other absolutely critical component of the body's functioning. Who knows how many others? Yet most doctors appear to be unaware of this potentially life-threatening adverse effect, as well as the possibility of, for instance, statin-induced global amnesia or severe polyneuropathy.

Apparently what happened with HRT has not inspired the institution of fail-safe mechanisms to prevent inadequately tested drugs to be widely prescribed.

Is there something wrong with our medical system? Pharmaceutical companies know about the CoQ10 depletion – Merck has a patent on a pill combining statins with CoQ10. In Canada the precaution statement given with the drug includes a warning about this depletion.⁶⁸ How is it possible that our medical system is not set up so that every doctor in the United States who potentially could prescribe statin drugs knows not only about the CoQ10 depletion but is fully informed about all other adverse effects? Why don't they know about the University of British Columbia review that concludes statins have not demonstrated a health benefit in primary prevention trials? I, personally, am very nervous when I have critical information about a health issue that physicians lack: they are the professionals and *should* be informed. The system apparently lacks mechanisms to insure that physicians are fully informed about life and death issues such as this.

Physicians are given an incredibly high level of trust in our culture. One friend taking statins (and experiencing severe adverse effects) read all of the information on CoQ10 – at which point he knew more about the CoQ10 issue than his original prescribing physician. Yet it was only weeks afterwards, when a second physician, a neurologist, told him to take CoQ10, that he actually began taking supplements. Even after what happened with HRT, people don't seem to question that the same thing might be going on with other drugs, such as statins.

I believe that in a situation if physicians are not being adequately informed, we *must* educate ourselves about our medical treatment and challenge doctors to educate *themselves*.

An increasing number of physicians and researchers are questioning the whole cholesterol theory of heart disease. (See endnote 57 for a list of reading on this subject.) Surely discovering the incredible role of cholesterol in the body ought to make anyone question how such a critically important substance could be responsible for the disease that is our biggest killer.

But the minds of most people are closed on this subject: they won't even consider a look at anything, no matter how well documented, that contradicts the commonly held view. I guess they figure their physician would tell them if it was wrong; but, physicians don't always have all the answers, as we have seen with HRT and the overall silence on CoQ10 depletion. Unfortunately, most doctors, already certain that they do have the answers, don't like to receive information from outside of a medical system that, as we have seen, is not always right.

Unfortunately, physicians, historically, have been slow to change and quick to ridicule those who would change. When Dr. Kilmer McCully, a Harvard pathologist, began presenting proof about the homocysteine theory of heart disease in the early 1970s, he was ridiculed. In 1978 he was dismissed from the Harvard faculty and released from the hospital where he worked. It took him two years to find a job – no one would hire such a pariah, who was questioning orthodox medical treatment. How dare he! When he was finally able to continue his work, he proved his theory.⁶⁹ How many lives might have been saved had his work been supported rather than denigrated?

So what do we do if we think the medical system is wrong? My answer is to educate myself so my decisions will be informed: ultimately, I have to decide what is best for my body. I know many people who have begun such a process of education and questioning only after contracting a serious illness that could not be treated by Western medicine or after suffering an adverse effect that was unknown to, ignored or belittled by their physicians.

As far as statin drugs go, I offer no medical advice to anyone else. If my doctor ever suggests that I take statins, I will make very certain that she has access to all of the information I have and is able to answer all my questions to my satisfaction.

A note in November 2010.

Although this was originally written in 2003 I would not change any of the information. Actually, since 2003 there are a large number of new resources and information about the topic. As a starting point, I suggest you go to the website for The International Network of Cholesterol Skeptics (at <http://thincs.org/>). I also recommend the book *Good Calories. Bad Calories: Challenging the Conventional Wisdom on Diet, Weight Control, and Disease* by Gary Taubes (Alfred A. Knopf, 2007) and *Fat and Cholesterol are Good for You* by Uffe Ravnskov (GB Publishing, 2009).

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(03-09-03)

(Note: URLs in Endnotes updated 10-11-14)

Endnotes

Statins: Did Your Doctor Tell You . . . ?

Note: With a few exceptions the material referenced is found on the internet. I have not followed standard bibliographical convention in these notes: my goal is to make them as easy to track down as possible. I have placed all footnotes and links on my website at:

<http://home.earthlink.net/~mbabc/statinlink.html>

as well as the links in the form of a bibliography at:

<http://home.earthlink.net/~mbabc/statinbib.html>

¹ Duane Graveline, M.D., M.P.H., “Statin Damage to the Mevalonate Pathway” – http://www.spacedoc.net/statin_damage_mevalonate.html

Also Uffe Ravnskov, “6. The Effect of the statins is not due to cholesterol-lowering” – <http://www.ravnskov.nu/myth6.htm>

The statins inhibit the body’s production of a substance called mevalonate, which is a precursor of cholesterol. When the production of mevalonate goes down, less cholesterol is produced by the cells and thus blood cholesterol goes down as well. But mevalonate is a precursor of other substances also, substances with important biologic functions.

² “Do Statins Have a Role in Primary Prevention,” a review by the Therapeutics Initiative of the Department of Pharmacology & Therapeutics of the University of British Columbia, Therapeutics Letters, April – May – June 2003. – <http://www.ti.ubc.ca/pages/letter48.htm> or <http://www.ti.ubc.ca/PDF/48.pdf> (Note: the pdf version is more suitable for printing.)

³ Joel M. Kauffman (Professor of Chemistry Emeritus), “Statin Sales Slow” – <http://www.thincs.org/unpublic.Joel3.htm>

⁴ Paul Rosch, “Converting Millions of Healthy People into Perpetual Patients” in Redflagsdaily.com, July 21, 2003 – <http://www.mombu.com/medicine/medicine/t-converting-millions-of-healthy-people-into-perpetual-patients-diabetes-cough-aspirin-asthma-psychiatry-2349959-last.html>

⁵ “Beatrice A. Golomb, MD, PhD on Statin Drugs” – <http://web.archive.org/web/20041024074619/http://www.coloradohealthsite.org/topics/interviews/golomb.html>

⁶ Jocalyn Clark, “A hot flush for Big Pharma,” BMJ (British Medical Journal) 2003;327;400 (16 August) – <http://bmj.com/cgi/content/full/327/7411/400>

⁷ According to Dr. Peter Langsjoen, a specialist in “congestive heart failure, primary and statin-induced diastolic dysfunction and other diseases of the heart muscle.” Maryann Napoli, “Cholesterol skeptics and the Bad News about Statin Drugs” – http://www.medicalconsumers.org/pages/cholesterol_skeptics.html

⁸ Christian B. Allan, Ph.D. and Wolfgang Lutz, M.D., *Life Without Bread* (Keats Publishing, 2000), p. 67. Chapter five, “Less is More,” found from pages 35-72, is an explanation of the body’s energy production.

⁹ Peter H. Langsjoen, M.D., F.A.C.C., “The Clinical Use of HMG CoA-reductase inhibitors (statins) and the associated depletion of the essential co-factor coenzyme Q10; a review of pertinent human and animal data.” – http://www.fda.gov/ohrms/dockets/dailys/02/May02/052902/02p-0244-cp00001-02-Exhibit_A-vol1.pdf

¹⁰ See footnote 9 (above).

¹¹ “Ubiquinone in a slightly altered form known as ubiquinol is found in all membranes where it has a vital function in maintaining membrane integrity. Liver inflammation, with breakdown of liver cells releasing their enzymes into the blood stream and thereby serving as a marker of statin damage, is likely due, at least in part, to loss of cell wall integrity.” Duane Graveline, M.D., M.P.H., “Aging and CoQ10” – http://www.spacedoc.net/aging_CoQ10

¹² “Ubiquinone is also vital to the formation of elastin and collagen formation. Tendon and ligament inflammation and rupture have frequently are frequently reported in the elderly it is likely that the mechanism of this predisposition to damage is related to some yet unknown compromise of ubiquinone’s role in connective tissue formation.” Duane Graveline, M.D., M.P.H., “Aging and CoQ10” – http://www.spacedoc.net/aging_CoQ10

¹³ “Statins block the endogenous biosynthesis of both cholesterol and CoQ10 by inhibiting the enzyme HMG CoA reductase, thus decreasing mevalonate, the precursor of both cholesterol and CoQ10.” Peter H. Langsjoen, M.D., F.A.C.C., “The Clinical Use of HMG CoA-reductase inhibitors (statins) and the associated depletion of the essential co-factor coenzyme Q10; a review of pertinent human and animal data.” Click – http://www.fda.gov/ohrms/dockets/dailys/02/May02/052902/02p-0244-cp00001-02-Exhibit_A-vol1.pdf

¹⁴ Christian B. Allan, Ph.D. and Wolfgang Lutz, M.D., *Life Without Bread* (Keats Publishing, 2000), p. 160.

¹⁵ Duane Graveline, M.D., M.P.H., “The Dark Side of Statins” – http://www.spacedoc.net/dark_side_statins.html

¹⁶ Nicholas Regush, Red Flag Daily’s “Health Trend Forecast,” May 16, 2003:

Anyone on the statin drug, Mevacor (as merely one example), might like to know that Canadian prescribing information includes a notice in the Precautions section stating the following:

Effect on CoQ10 Levels (Ubiquinone)

A Significant decrease in plasma CoQ10 levels in patients treated with Mevacor and other statins has been observed in short-term clinical trials. The clinical significance of a potential long-term statin-induced deficiency of CoQ10 has not yet been established....”

¹⁷ Peter H. Langsjoen, M.D., F.A.C.C., “The Clinical Use of HMG CoA-reductase inhibitors (statins) and the associated depletion of the essential co-factor coenzyme Q10; a review of pertinent human and animal data.” Click on “Cholesterol Drugs And The Depletion of Coenzyme Q10: A Review of Human And Animal Data” – http://www.fda.gov/ohrms/dockets/dailys/02/May02/052902/02p-0244-cp00001-02-Exhibit_A-vol1.pdf

¹⁸ Maryann Napoli, “Cholesterol skeptics and the Bad News about Statin Drugs” – http://www.medicalconsumers.org/pages/cholesterol_skeptics.html

¹⁹ Peter H. Langsjoen, M.D., F.A.C.C., “The Clinical Use of HMG CoA-reductase inhibitors (statins) and the associated depletion of the essential co-factor coenzyme Q10; a review of pertinent human and animal data.” – http://www.fda.gov/ohrms/dockets/dailys/02/May02/052902/02p-0244-cp00001-02-Exhibit_A-vol1.pdf

²⁰ “It has been pretty well documented from biopsies that the severity of heart failure correlates with the people who have the lowest levels of Q10.” (Attributed to Peter H. Langsjoen.) Maryann Napoli, “Cholesterol skeptics and the Bad News about Statin Drugs” – http://www.medicalconsumers.org/pages/cholesterol_skeptics.html

“Statins kill people - lots of people - and they wound many, many more.” Peter H. Langsjoen, M.D., “Statin-induced Cardiomyopathy” – http://www.redflagsweekly.com/features/2002_july08P.html

²¹ Peter H. Langsjoen, M.D., “Introduction to the Citizen’s Petition on Statins” – <http://forum.lowcarber.org/archive/index.php/t-49880.html>

²² “Ubiquinone in its slightly altered form known as ubiquinol is found in all cellular membranes where it has a vital function in maintaining membrane integrity. Myopathy and rhabdomyolysis represent breakdown of weakened muscle cell walls due to lack of sufficient ubiquinol for muscle cell wall integrity. This same mechanism also is responsible for nerve cell breakdown and neuropathies.” Duane Graveline, M.D., M.P.H., “Muscle Pain and Statins” – http://www.spacedoc.net/muscle_pain_statins.htm

²³ “Watchover [a program of the FDA] was informed of each Baycol death as it occurred during these past two years but bureaucratic inertia allowed over fifty fatalities to occur before action was taken placing the credibility and effectiveness of FDA in serious question.” Duane Graveline, M.D., M.P.H., “The Statin Dialogues – A Fable” – http://www.spacedoc.net/statin_dialogues.htm

²⁴ Dr. Julian M. Whitaker, M.D., “Citizen Petition to Change the Labeling for All Statin Drugs . . .” – <http://www.fda.gov/ohrms/dockets/dailys/02/May02/052902/02p-0244-cp00001-01-vol1.pdf>

²⁵ “Do Statins Have a Role in Primary Prevention,” a review by the Therapeutics Initiative of the Department of Pharmacology & Therapeutics of the University of British Columbia, *Therapeutics Letters*, April – May – June 2003. – <http://www.ti.ubc.ca/pages/letter48.htm> or <http://www.ti.ubc.ca/PDF/48.pdf> (Note: the pdf version is more suitable for printing.)

²⁶ “Editorial Comment on Cholesterol and Statin Drugs” – http://web.archive.org/web/20041113151702/www.coloradohealthsite.org/CHNReports/editorial_cholesterol.html

²⁷ “Beatrice A. Golomb, MD, PhD on Statin Drugs” – <http://web.archive.org/web/20041024074619/http://www.coloradohealthsite.org/topics/interviews/golomb.html>

²⁸ “Zocor® (Simvastatin) – UK Side Effect Reports” – http://www.spacedoc.net/zocor_UK_side_effect_reports

²⁹ Duane Graveline, M.D., M.P.H., “The Statin Dialogues – A Fable” – http://www.spacedoc.net/statin_dialogues.htm

³⁰ Duane Graveline, M.D., M.P.H., “Transient Global Amnesia Associated With The Statin Drugs” (no longer available online)

³¹ “Statins and Risk of Polyneuropathy: A Case-control Study,” by D. Gaist, MD, PhD; U. Jeppesen, MD, PhD; M. Andersen, MD, PhD; L. A. Garcia Rodriguez, MD, MSc; J. Hallas, MD, PhD; and S. H. Sindrup MD, PhD; *Neurology*, May 2002 – http://web.archive.org/web/20041205141430/www.coloradohealthsite.org/CHNReports/statins_polyneuropathy.html

³² See footnote 31 (above).

³³ Except where noted, information from this section is from “Identifying and Preventing Statin-Associated Muscle Problems” based on the study “Statin-Associated Myopathy,” by Paul D. Thompson, MD; Priscilla Clarkson, PhD; Richard H. Karas, MD, PhD ; JAMA, April 2, 2003. – http://web.archive.org/web/20041205141430/http://www.coloradohealthsite.org/CHNReports/statins_muscleproblems.html – See also “Beatrice A. Golomb, MD, PhD on Statin Drugs,” – <http://web.archive.org/web/20041024074619/http://www.coloradohealthsite.org/topics/interviews/golomb.html>

³⁴ “CK Blood Test Inadequate to Diagnose Statin-Associated Myopathy,” a review of “Statin-Associated Myopathy with Normal Creatine Kinase Levels,” by Paul S. Phillips, MD; Richard H. Haas, MD; Sergei Bannykh, MD, PhD; Stephanie Hathaway, RN; Nancy L. Gray, RN; Bruce J. Kimura, MD; Georgirene D. Vladutiu, PhD; John D.F. England, MD, the Scripps Mercy Clinical Research Center; *Annals of Internal Medicine*, October 1, 2002 – http://web.archive.org/web/20041205141430/http://www.coloradohealthsite.org/CHNReports/statins_CKtest.html

³⁵ Joseph Mercola, M.D., “The Baycol Recall: How Safe is Your Statin?” – http://www.mercola.com/2001/aug/18/baycol_recall.htm

³⁶ “Beatrice A. Golomb, MD, PhD on Statin Drugs” – <http://web.archive.org/web/20041024074619/http://www.coloradohealthsite.org/topics/interviews/golomb.html>

³⁷ “Recently, Drs. Thomas Newman and Stephen Hulley published the results from a meticulous review of what we know about cancer and lipid-lowering drugs. They found that clofibrate, gemfibrozil and all the statins stimulate cancer growth in rodents (90).” (90 = Newman TB, Hulley SB. Carcinogenicity of lipid-lowering drugs. *JAMA* 1996;275:55-60.) – Uffe Ravnskov, “6. The Effect of the statins is not due to cholesterol-lowering” – <http://www.ravnskov.nu/myth6.htm>

³⁸ Uffe Ravnskov, “Evidence that statin treatment causes cancer,” Letter to the editor of *Lancet*, sent 10. December 2002 – <http://www.thincs.org/unpublic.UR3.htm>

³⁹ Christian B. Allan, Ph.D. and Wolfgang Lutz, M.D., *Life Without Bread* (Keats Publishing, 2000), p. 160. This quote has a footnote as follows: “Sinatra, S. “Care, cancer and coenzyme Q10.” *J. Am. Coll. Cardiol.* 33 (1999): 897-899.” Also Paul Rosch, M.D., F.A.C.P., letter to the editor of the *Washington Post* – <http://www.thincs.org/unpublic.Paul3.htm> – “All statins have been shown to be carcinogenic in animals in doses equivalent to those currently being prescribed. Although the lag time between exposure to a carcinogen and clinical detection is often a decade or more, a disturbing twelve-fold increase in breast cancer has already been reported in one study and more skin malignancies were noted in another.”

⁴⁰ Uffe Ravnskov, M.D., Ph.D., “1. Your cholesterol tells very little about your future health” – <http://www.ravnskov.nu/myth1.htm>

⁴¹ The information on cholesterol is taken from three articles:

Sally Fallon and Mary G. Enig, Ph.D., “What Causes Heart Disease” – <http://www.westonaprice.org/modern-diseases/cardiovascular-disease/585-what-causes-heart-disease.html>

Duane Graveline, M.D., M.P.H., “Cholesterol – Friend or Foe?” – http://www.spacedoc.net/cholesterol_friend_or_foe.html

Uffe Ravnskov, M.D., Ph.D., “1. Your cholesterol tells very little about your future health” – <http://www.ravnskov.nu/myth1.htm>

⁴² Barry Groves, “The Cholesterol Myth: Part 1: Introduction,” – http://www.second-opinions.co.uk/cholesterol_myth_1.html

⁴³ Sally Fallon and Mary G. Enig, Ph.D., “The Oiling of America” – <http://www.westonaprice.org/know-your-fats/525-the-oiling-of-america.html>

⁴⁴ Except where noted, information in the following list is taken from:

Uffe Ravnskov, “Statins as the new aspirin” – <http://bmj.com/cgi/content/full/324/7340/789>

Barry Groves, “The Dangers of Low Cholesterol” – http://www.second-opinions.co.uk/cholesterol_myth_4.html

⁴⁵ Mary G. Enig, Ph.D. and Sally Fallon, “What Causes Heart Disease” – <http://www.westonaprice.org/modern-diseases/cardiovascular-disease/585-what-causes-heart-disease.html> – Also in “The Skinny on Fats” – http://www.westonaprice.org/know_your_fats/skinny.html – “The few studies that indicate a correlation between fat reduction and a decrease in coronary heart disease mortality also document a concurrent increase in deaths from cancer, brain hemorrhage, suicide and violent death. (6 – “Multiple Risk Factor Intervention Trial; Risk Factor Changes and Mortality Results,” *JAMA*, September 24, 1982, 248:12:1465.)”

⁴⁶ “A study of elderly French women living in a nursing home showed that those with the highest cholesterol levels lived the longest (*The Lancet*, 4/22/89). The death rate was more than five times higher for women with very low cholesterol. Several other studies have shown similar results.” Maryann Napoli, “Cholesterol Skeptics And The Bad News About Statin Drugs” – http://www.medicalconsumers.org/pages/cholesterol_skeptics.html

⁴⁷ The following quote is from “Editorial Comment on Cholesterol and Statin Drugs” – http://web.archive.org/web/20041113151702/www.coloradohealthsite.org/CHNReports/editorial_cholesterol.html

Media messages and advertisements often urge patients to use drugs to reduce their LDL cholesterol below 130 and even below 100 in order to lower their risk of heart attack. The messages typically *fail to warn patients that LDL cholesterol levels below 130 may actually increase the risk of coronary heart disease*. For example, the FDA has recently approved a new laboratory blood test that can increase the ability of doctors to predict the risk of coronary heart disease (CHD). The test, called PLAC, works by measuring an enzyme called lipoprotein-associated phospholipase A2. FDA cleared the test based on results of a 9 year study of more than 1,348 patients. The study was a part of a large multi-center epidemiologic study sponsored by the National Heart, Lung, and Blood Institute. Patients were free from CHD at the start of the study and were followed for the development of disease for nine years. **The greatest increased risk for CHD was found in subjects with the highest PLAC test results, and LDL cholesterol levels lower than 130mg/dL.** [Emphasis in original.]

Giving a statin drug to a person with LDL cholesterol of 140 or below, as is frequently done today, would result in a 30 to 60% reduction in his or her LDL cholesterol, potentially placing him or her at increased risk for heart attack if his or her lipoprotein-associated phospholipase A2 is high.

⁴⁸ Malcolm Kendricks MbChB, MRCGP, “Why The Cholesterol-Heart Disease Theory Is Wrong (Part Three: A Raised LDL Level Has No Impact on Heart Disease)” – you’ll need to scroll down the page: <http://www.thincs.org/Malcolm.choltheory.htm>

Kendricks says, about the Framingham Study:

There is a direct association between falling cholesterol levels over the first 14 years and mortality over the following 18 years (11% overall and 14% CVD death rate increase per 1 mg/dL per year drop in cholesterol levels). Anderson KM JAMA 1987

⁴⁹ Duane Graveline, M.D., M.P.H., “Cholesterol – Friend or Foe?” – http://www.spacedoc.net/cholesterol_friend_or_foe.html

⁵⁰ Sally Fallon and Mary G. Enig, Ph.D., “Diet and Disease: Not What you Think” – http://www.coconut-info.com/diet_and_disease.htm

⁵¹ “Most risk factors for heart disease are merely “risk markers” that simply have some statistical association with an increased incidence of coronary events. There are over 300 risk factors for heart attacks, including a deep earlobe crease, premature vertex baldness, high selenium toenail levels, having a pot belly, not having a nap or one or two glasses of wine a day.” Paul J. Rosch, M.D., F.A.C.P., “Do You Have Good Blood Pressure?” – <http://articles.mercola.com/sites/articles/archive/2003/06/21/good-blood-pressure.aspx> (originally in the July Health and Stress monthly newsletter of the American Institute of Stress)

⁵² Dr. Peter H. Langsjoen, “To Reduce the Risk of Heart Disease, Why Don’t We All Cut Off Our Ear Lobes” – <http://consumercide.com/health/earlobeoff.html>

⁵³ “In order to successfully treat a disease it is necessary to remove or reduce its cause rather than its manifestations or markers.” “Attempting to treat or remove such markers will accomplish nothing since they do not cause coronary disease.” Paul J. Rosch, M.D., F.A.C.P., “Do You Have Good Blood Pressure?” – <http://articles.mercola.com/sites/articles/archive/2003/06/21/good-blood-pressure.aspx> (Originally Published in the July Health and Stress monthly newsletter of the American Institute of Stress.)

The following quote is from Malcolm Kendrick MbChB, MRCGP, “Idiotic Thinking In Medicine C-Reactive Protein: How The Medical Profession Will Turn A Symptom Into A Disease” – <http://www.thincs.org/Malcolm.htm#CRP>

When you find an abnormality of some sort that is associated with a disease, you can make a number of different conjectures:

1. The abnormality is caused by the disease
2. An underlying problem causes both the abnormality and the ‘disease’
3. The disease is caused by the abnormality
4. It’s a coincidence (one in twenty chance)
5. You haven’t measured things properly

⁵⁴ Malcolm Kendricks MbChB, MRCGP, “Why The Cholesterol-Heart Disease Theory Is Wrong (Part Two)” – you’ll need to scroll down the page – <http://www.thincs.org/Malcolm.choltheory.htm>

“So, at what point does saturated fat get turned into cholesterol?”

“Answer, it doesn’t. You don’t make cholesterol out of saturated fat. Cholesterol, when it is made in the liver, starts out as a substance called Acteyl-co A. This is not a fat; it is nothing like a fat. It has several nitrogen atoms in it, and nitrogen comes from protein.”

⁵⁵ Sally Fallon and Mary G. Enig, Ph.D., “Diet and Disease: Not What you Think” – http://www.coconut-info.com/diet_and_disease.htm

⁵⁶ Sally Fallon and Mary G. Enig, Ph.D in “The Oiling of America” – <http://www.westonaprice.org/know-your-fats/525-the-oiling-of-america.html> – mention a 1964 study of 1700 people by the famous heart surgeon De Bakey that showed that blocked arteries were found in people with low cholesterol as often as those with high cholesterol. (They refer to the following study: M De Bakey, et al, "Serum Cholesterol Values in Patients Treated Surgically for Atherosclerosis," JAMA, 1964, 189:9:655-59.) This is just one example.

⁵⁷ There is a wealth of material available online and off about this topic, fully supported by articles from peer-reviewed medical and scientific journals. Significantly, none of the authors have any financial stake in the notion that cholesterol causes heart disease.

- Visit the website of The International Network of Cholesterol Skeptics (THINCS). “The aim with this website is to inform our colleagues and the public that this idea is not supported by scientific evidence; in fact, for many years a huge number of scientific studies have directly contradicted it.” – <http://www.thincs.org/>
- Barry Groves, Ph. D (a doctor of nutritional science) has an excellent article “The Cholesterol Myth” – http://www.second-opinions.co.uk/cholesterol_myth_1.html
- Uffe Ravnskov, M.D., Ph.D. has written a book *The Cholesterol Myths: Exposing the Fallacy that Saturated Fat and Cholesterol Cause Heart Disease*. (New Trends Publishing, 2000, now out of print). This book could be a textbook on how to manipulate scientific data to produce a desired result regardless of facts. A large amount of the information found in the book can be found on his website – <http://www.ravnskov.nu/cholesterol.htm> (scroll down to “Here are the Facts” and click on the blue numbers).
- “The Oiling of America” by Sally Fallon and Mary G. Enig, Ph.D., reviews how this theory came to be promulgated – <http://www.westonaprice.org/know-your-fats/525-the-oiling-of-america.html>
- Another article, “The Soft Science of Dietary Fat” by Gary Taubes, also points out how the cholesterol theory of heart disease was able to become accepted as fact – <http://www.second-opinions.co.uk/taubes.html>
- Thomas J, Moore, wrote a book *Heart Failure: A Critical Inquiry into American Medicine and the Revolution in Heart Care*, (Touchstone Books, 1990) Chapter 5 from the book is found online – <http://www.oralchelation.net/heartdisease/ChapterFive/page5g.htm>

⁵⁸ Both examples taken from Sally Fallon and Mary G. Enig, Ph.D., “Diet and Disease: Not What you Think” – http://www.coconut-info.com/diet_and_disease.htm

⁵⁹ “Several speakers expressed the opinion that the statin drugs’ ability to reduce cardiovascular mortality has nothing to do with cholesterol reduction, but instead can be attributed to their anti-inflammatory effects. (A viewpoint that has been appearing in medical journals over the last few years.)” Maryann Napoli, “Cholesterol Skeptics And The Bad News About Statin Drugs” – http://www.medicalconsumers.org/pages/cholesterol_skeptics.html

“That statin treatment works in patient and age groups in whom a high cholesterol concentration is not a risk factor for cardiovascular disease shows that the benefit is not the result of cholesterol lowering. High or low cholesterol concentrations are markers for other, more important disease factors; they are not causal factors themselves.” Uffe Ravnskov, letter to the editor, *BMJ* 2002;324:789 (30 March) – <http://bmj.com/cgi/content/full/324/7340/789>

Uffe Ravnskov, both in his book and online at “6. The Effect of the statins is not due to cholesterol-lowering” – <http://www.ravnskov.nu/myth6.htm> – points out a half dozen reasons. “And finally, the statins protected against coronary heart disease whether the cholesterol was high or low although most studies have shown that a normal or low cholesterol is no risk factor for coronary disease.” They also protected against strokes, although high cholesterol is not considered a risk factor for stroke. Clearly something else is going on.

“Statins have strong anti-coagulant effects, they stabilise plaques and increase NO synthesis.” [NO is nitric oxide, a powerful anti-coagulant.] Malcolm Kendrick, MbChB, MRCGP, “Is Heart Disease All Due to Blood Clots?” – <http://www.thincs.org/Malcolm.htm#clots>

See also Malcolm Kendrick, MbChB, MRCGP, “Statins Do Not Prevent Heart Disease – At Least Not By Lowering LDL/Cholesterol Levels” – you’ll need to scroll down the page – <http://www.thincs.org/Malcolm.choltheory.htm>

⁶⁰ Malcolm Kendrick MbChB, MRCGP, “So, What Does Cause Heart Disease” – <http://www.thincs.org/Malcolm.htm#heart1>

⁶¹ Same as 60 (above).

⁶² “Metabolic Syndrome and its Effects on Heart Disease and Diabetes” – A Colorado Health Site review of the study “Metabolic Syndrome With and Without C-Reactive Protein as a Predictor of Coronary Heart Disease and Diabetes in the West of Scotland Coronary Prevention Study, by Naveed Sattar, Allan Gaw, Olga Scherbakova, Ian Ford, Denis St.J. O’Reilly, Steven M. Haffner, Chris Isles, Peter W. Macfarlane, Chris J. Packard, Stuart M. Cobbe, and James Shepherd; *Circulation*, July 29, 2003” – http://web.archive.org/web/20040807235133/www.coloradohealthsite.org/CHNReports/metabolicsyndrome_heartdisease.html

⁶³ Christian B. Allan, Ph.D. and Wolfgang Lutz, M.D., *Life Without Bread*, (Keats Publishing, 2000), p. 89.

⁶⁴ Sally Fallon and Mary G. Enig, Ph.D., “What Causes Heart Disease” – <http://www.westonaprice.org/modern-diseases/cardiovascular-disease/585-what-causes-heart-disease.html>

The situation is further complicated by the fact that commercial vegetable oils contain mostly omega-6 fatty acids. The body uses these types of fatty acids to make localized hormones, called prostaglandins, that initiate the process of blood clotting and of inflammation. This is an important mechanism. Without it, we would bleed to death when we cut ourselves and wounds would not heal. The problem occurs when these clot- and inflammation-promoting prostaglandins are not balanced by prostaglandins that inhibit clotting.

Many of the anti-inflammatory and clot-inhibiting prostaglandins are made from omega-3 fatty acids, of which there are very few in commercial vegetable oils, or indeed in fruits, vegetables, fish and eggs raised by modern farming methods. Thus, when the diet contains too much of omega-6 fatty acids and not enough of omega-3 fatty acids, there may be a tendency to form blood clots leading to heart attacks. (25)

Here is the footnote (25) referred to by Fallon and Enig in the above quote:

Kinsella, JE. *Food Technology*, October 1988, page 134; Lasserre M and others. *Lipids* 20 (4), 227, 1985; Horrobin, DF. *Reviews in Pure and Applied Pharmacological Sciences*, Vol 4, Freund Publishing House, 1983, pages 339-383; Devlin, TM, ed. *Textbook of Biochemistry*, 2nd Ed, Wiley Medical, 1982, 429-430; Fallon S and Enig MG. Tripping Lightly Down the Prostaglandin Pathways, *The Price-Pottenger Nutrition Foundation Health Journal* 20(3), 5-8, 1996. (Also posted at http://westonaprice.org/know_your_fats/tripping.html)

⁶⁵ Sally Fallon and Mary G. Enig, Ph.D., “What Causes Heart Disease” – <http://www.westonaprice.org/modern-diseases/cardiovascular-disease/585-what-causes-heart-disease.html>

⁶⁶ Christian B. Allan, Ph.D. and Wolfgang Lutz, M.D., *Life Without Bread*, (Keats Publishing, 2000), pp. 104-109. This book is based, in part, on the experience of Dr. Lutz with thousands of patients over several decades.

⁶⁷ Malcolm Kendrick MbChB, MRCP, “So, What Does Cause Heart Disease” – <http://www.thincs.org/Malcolm.htm#heart1> and Mary G. Enig, Ph.D. and Sally Fallon, “What Causes Heart Disease” – <http://www.westonaprice.org/modern-diseases/cardiovascular-disease/585-what-causes-heart-disease.html> – The Enig/Fallon article stresses the importance of saturated fats in the diet – they are rich in the protective factors Vitamins A, D and E.

⁶⁸ Nicholas Regush, Red Flag Daily’s “Health Trend Forecast,” May 16, 2003:

Anyone on the statin drug, Mevacor (as merely one example), might like to know that Canadian prescribing information includes a notice in the Precautions section stating the following:

Effect on CoQ10 Levels (Ubiquinone)

A Significant decrease in plasma CoQ10 levels in patients treated with Mevacor and other statins has been observed in short-term clinical trials. The clinical significance of a potential long-term statin-induced deficiency of CoQ10 has not yet been established....”

⁶⁹ Bruce Fife, N.D., *Saturated Fat May Save Your Life* (HealthWise, 1999), pp. 156 to 161.

Doctors have been conservative for centuries (possibly millennia). In the mid-19th century, prior to the discovery of bacteria, Ignaz Semmelweiss (1818-1865) discovered that when physicians washed their hands in a chlorine solution prior to assisting with a woman's labor, the death rate was dramatically reduced. When he published a book about his findings in 1861, he was ridiculed. He was lost at least one job for his discovery and went insane as he tried to fight for his discovery. See Dr. Mark Taylor, "Ignaz Semmelweiss: 'Please wash your hands'"