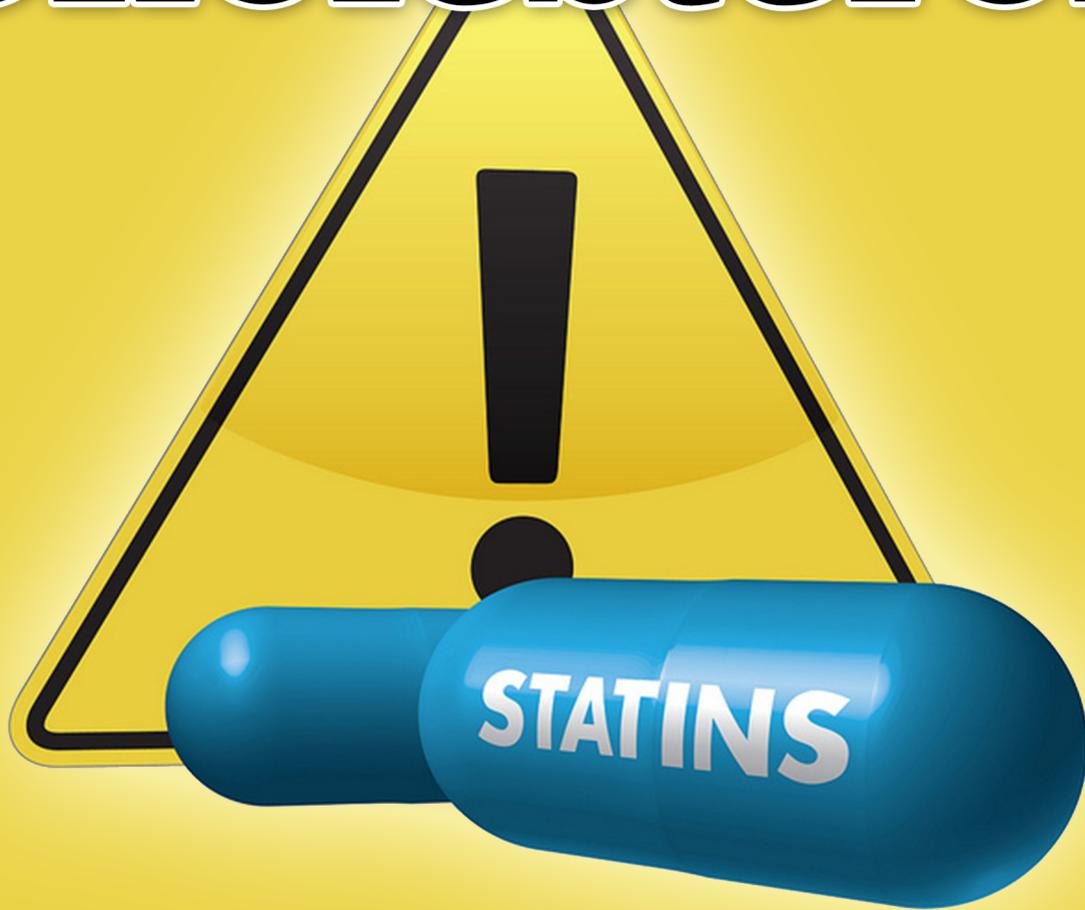


The Truth About Statins & Cholesterol



**A true science exclusive report from
the cutting edge of medicine**

by

Keith Scott-Mumby MD, MB ChB, PhD

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The Hoax

Statins and their supposed beneficial effects is probably the greatest medical scam of all time. It is a travesty of lies, massaging facts, bendy statistics and *relentless marketing hype* that has created a world-view, almost, that statins are effective and work so well they are practically essential for life.

The use of these drugs is now so ingrained in medical "science" that a doctor who does not prescribe them to anybody and everybody over the age of 50 is considered negligent and incompetent.

Yet there is abundant science that statins reduce longevity (death from all causes), ruin the quality of life and do NOT do what is claimed. In fact their very mode of action itself is proof positive that they cannot and do not work.

Statins kill.

How did this incredible fairy story, this triumph of lies over reason, get started?

The answer is contained in one word: money (OK, profits, that's two words!)

Lipitor (atorvastatin) by 2003 had become the best-selling pharmaceutical in history, with manufacturers Pfizer reporting sales of US\$12.4 billion in 2008 ["Doing Things Differently", Pfizer 2008 Annual Review, April 23, 2009, p. 15].

Through the clever and extensive manipulation of the media, including medical science journals, Big Pharma has convinced even doctors that statins work and that we need them. Once again it is an insane story of "Nature screwed up and we clever (pharma) scientists know better than Mother Nature."

I don't buy it.

It's gone beyond criminal and has entered obscene. Yes, I mean obscene, which is defined in law as: "having a tendency to deprave or corrupt" (<http://www.thefreedictionary.com/obscene>)

It's not just medical folly. The media have joined in the chorus of "*Halleluja, we are saved!*" [<http://www.dailymail.co.uk/health/article-2194892/All-50s-statins-regardless-health-history-says-Oxford-professor.html>]

The Earlier Cholesterol Folly

Little wonder that statins shorten life. Their supposed mode of action (and I use the word supposed advisedly) is that they reduce cholesterol. Too much cholesterol is bad for you, therefore statins are, almost by definition, a good thing.

There are two utterly irrational stupidities to this hypothesis:

1. It only makes sense if statins don't do some other damage, while reducing cholesterol. In fact they do a lot of concomitant damage.
2. It only makes sense if cholesterol is bad, as claimed. Cholesterol is GOOD and we need lots of it to be healthy.

Point 1. Is important. Whilst some statin clinical trials have shown a very slight reduction in cardiac events with statins, this has always been counter-acted by deaths from other causes. The net result being that people did not live any longer after taking statins.

Moreover raised cholesterol levels have never been shown to be the real cause of heart disease. Raised cholesterol (that needs defining) may just be the oil warning light on the car dashboard and not doing any direct damage to the engine.

So neither of the two elements of the hypothesis are true or tenable. Yet irresponsible physicians, like Professor Sir Rory Collins, claim that we should all take statins for life: apparently he's smart and God is an idiot, or something...

Doctors are claiming healthy people need this class of drugs? What? It seems they don't even care what the patient's lab numbers are; in the rush to get out their prescription pads and help out their pals in the manufacturing side of the healthcare bonanza, doctors don't even care whether the patient is sick or healthy!

We are led to believe that the benefits associated with statins far outweigh any risks. However, when it comes to primary prevention (accounting for around 75 percent of all the people who take a statin), no clinical trial has been able to conclusively show any net benefit from statins.

[<http://www.sott.net/article/251393-New-Documentary-Exposes-the-Over-Prescription-of-Statins>]

Think I'm Exaggerating About The Money Angle?

Look at this list of declared conflict of interest from just ONE paper on blood lipids [Lancet. 2010 May 1; 375(9725): 1536–1544. doi: 10.1016/S0140-6736(10)60319-4]

Conflicts of interest:

Alexander Thompson's institution has received research funding from the British Heart Foundation, GlaxoSmithKline, and UK Medical Research Council. He has received honoraria and reimbursement of costs for speaking at scientific meetings from GlaxoSmithKline.

Pei Gao, Lia Orfei, Sarah Watson, Emanuele Di Angelantonio, and Stephen Kaptoge's institution has received research funding from the British Heart Foundation, GlaxoSmithKline, and UK Medical Research Council.

Christie Ballantyne's institution has received research funding from diaDexus and GlaxoSmithKline, and he has received consultancy fees from GlaxoSmithKline.

Christopher P Cannon has received research funding from Bristol-Myers Squibb, Sanofi-Aventis, Intekrin, Novartis, Takeda, and GlaxoSmithKline. His institution has received research funding from Accumetrics, AstraZeneca, and Merck. He has received honoraria from Bristol-Myers Squibb and Sanofi-Aventis, and reimbursement of costs for attending scientific meetings from AstraZeneca, GlaxoSmithKline, and Merck. He is a member of Data Safety Monitoring Boards for GlaxoSmithKline and Merck. He has received consultancy payments from, and holds an equity interest in, Automedics Medical Systems. He has received payment for expert testimony from the University of Michigan.

Michael Criqui has received honoraria and reimbursement of costs for attending scientific meetings from GlaxoSmithKline.

Mary Cushman's institution has received research funding from GlaxoSmithKline. She has received honoraria from GlaxoSmithKline and the US National Institutes of Health, and reimbursement of costs for attending scientific meetings from GlaxoSmithKline.

Albert Hofman declares that he has no conflicts of interest.

Chris Packard's institution has received research funding from GlaxoSmithKline and he has received honoraria and reimbursement of costs for attending scientific meetings from GlaxoSmithKline.

Simon G Thompson has received honoraria and reimbursement of costs for attending scientific meetings from GlaxoSmithKline.

Rory Collins is paid by the British Heart Foundation, National Health Service, and UK Biobank, and has received research funding and reimbursement of costs for attending scientific meetings (but no honoraria or consultancy payments) from AstraZeneca, Bayer, Bristol-Myers Squibb, British Heart Foundation, Cancer Research UK, European

Union, GlaxoSmithKline, Kadoorie Trust, Medical Research Council, Merck, Roche, Sanofi, Schering, Solvay, and UK Biobank.

John Danesh has received research funding from the British Heart Foundation, BUPA Foundation, Denka, diaDexus, European Union, Evelyn Trust, Fogarty International Centre, GlaxoSmithKline, Medical Research Council, Merck, National Heart, Lung, and Blood Institute, National Institute of Neurological Disorders and Stroke, Novartis, Pfizer, Roche, the Wellcome Trust, and UK Biobank. He has also received honoraria and reimbursement of costs for speaking at scientific meetings from GlaxoSmithKline and Novartis. He is a member of the Merck Sharp and Dohme UK Atherosclerosis Advisory Board and Novartis Cardiovascular and Metabolic Advisory Board.

How can you believe in the objectivity of a bunch whose very careers are in the pocket of Big Pharma? It's not about bribes; it's about the fact they can't even pay the mortgage, unless they dance to Big Pharma's tune.

Think I am Exaggerating About The Crooked Science?

Think again.

Pharmaceutical companies and much of the world's media have been touting the results of the JUPITER trial. However, if we take a closer look at the data for this trial, we can see that the statin and the placebo group had exactly the same number of cardiovascular related deaths.

In addition, an article published in the Archives of Internal Medicine in 2010 questioned the validity of the data from the JUPITER trial and raised concerns about the role of the company sponsoring the trial (AstraZeneca, manufacturers of Crestor, a statin). Another article published in the journal Cardiology in 2011 raised similar concerns.

In 2010, a meta-analysis of 11 statin trials was published in the Archives of Internal Medicine. Professor Kausik Ray and colleagues concluded that statins provided no benefit in terms of deaths from all causes.

In 2011, the highly respected Cochrane Collaborative conducted a review of statin clinical trials. Based on this review, lead authors Dr Shah Ebrahim and Dr Fiona Taylor said that they could not recommend the use of statins for primary prevention. The absolute benefit was so small that it could have been down to chance, and even if it was a real benefit, 1000 people would have to be treated for one year to prevent one death.

The Blizzard Of Statins

So, despite all the hooey and bendy science, we are awash with statins, yet only a tiny proportion of the population have any benefit whatsoever from them. In fact the only human beings who benefit just a little from this junk are middle-aged men who have had a heart attack. That's not a big group.

Statins have never been documented to benefit any woman of any age with any condition.

They have not been documented to help people who have not had a previous heart attack of any age or gender.

[Dr Dwight Lundell, interview: "The True Cause Of Heart Disease" <http://www.totalhealthbreakthroughs.com/2010/07>]

What's more, demographics don't back up the ridiculous false claims of the pharmaceutical industry and jackanapes like Rory Collins. The US Centers for Disease Control and Prevention recently reported that the prevalence of heart disease in the US population remained stable over the past decade, while the use of statin drugs increased 10-fold! [Voelker, JAMA, 305:16, p.1641, 2011]

If ten-times the drug usage doesn't alter the prevalence of heart disease one tiny bit, what use are those drugs?

But it's worse even: the incidence of heart failure doubled in the ten years following the introduction of statin therapy and heart failure is now the number one cause of death!

[R. Foelker, JAMA. 2011;305(16):1641 <http://www.cdc.gov/nchs/hus.htm>]

How is this possible if statins are as great as being claimed? And why would we follow Sir Rory Collins' advice and take statins when we reach the age of 50, if that is going to DOUBLE our risk of death by heart failure?

Duh!

Let's look at why cholesterol is good and statins are BAD...

Why Cholesterol Is Good

Despite all of the negative reports regarding cholesterol, this life-sustaining lipid is essential for several important functions required for optimal health. Cholesterol is a vitally important component of all cell membranes; serves as a precursor molecule for the synthesis of steroid hormones, vitamin D, and bile acids; and plays a role in nerve conduction, brain function, and mood.

Without cholesterol, we wouldn't be able to exist.

The majority of cholesterol in the body is synthesized in the liver and the intestines. Cholesterol is transported through the blood as lipoproteins, which are lipid-protein complexes that differ in their content of lipid (fat) and protein.

Dietary intake of saturated fats is much less important than what we make on board. Diets low in cholesterol and saturated fats modulate low-density lipoprotein (LDL) cholesterol by only approximately 10 percent [National Institutes of Health. Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III Final Report). Available at: <http://www.nhlbi.nih.gov/guidelines/cholesterol/atp3full.pdf>. Accessed on: 12-27-11].

Of particular concern is the amount of small, dense LDL particles (the “bad” cholesterol). Research indicates that these particles are more likely to be related to suboptimal cardiovascular health and blood flow than larger, “fluffy” LDL particles [Lamarche B, Lemieux I, Despres JP. The small, dense LDL phenotype and the risk of coronary heart disease: epidemiology, patho-physiology and therapeutic aspects. *Diabetes Metab.* 1999 Sep;25(3):199-211].

High-density lipoprotein (HDL or the “good” cholesterol) is also involved in transport. However, these lipoproteins acquire cholesterol from peripheral tissues and transport this cholesterol back to the liver for incorporation into bile acids, which is known as reverse cholesterol transport.

Current standard medical guidelines suggest that total cholesterol should be below 200 mg/dL, LDL below 100 mg/dL, triglycerides below 150 mg/dL, and HDL above 40 mg/dL for men and 50 mg/dL for women.

Yet, there remains a degree of controversy on the topic, as heart disease is not merely determined by these indices, and without a more comprehensive approach looking at LDL size (small dense) or (large buoyant), lipoprotein A, fibrinogen, homocysteine and cardio-CRP, one misses the proverbial boat when it comes to protecting the heart.

In my day, 260 mgm/dL was considered about right. I think anything under 200 mgm/dL will eventually emerge as sub-optimum.

Cholesterol is not and never has been “bad”. We need plenty, as I said. The only hazard with cholesterol is when it becomes oxidized (rancid). Just take your antioxidants; eat lots of fresh colored food; no problem.

The Lipid Hypothesis

The so-called “lipid hypothesis” assumes that fats are the cause of heart disease. But is that true? Everyone parrots it. But it’s a lie.

A group of scientists, calling themselves The International Network of Cholesterol Skeptics, question the lipid hypothesis and argue that elevated cholesterol has not been adequately shown to cause heart disease. These organizations maintain that statins are not as beneficial

or safe as suggested [Ravnskov U, Rosch P, Sutter M, Houston M (2006). "Should we lower cholesterol as much as possible?". *BMJ* 332 (7553): 1330–2. doi:10.1136/bmj.332.7553.1330. PMC 1473073. PMID 16740566]

The US market for statins nearly tripled when the National Cholesterol Education Program (NCEP) revised its guidelines to recommend statins as primary prevention. Although the panel cited randomized trials to support statin therapy for primary prevention of occlusive cardiovascular disease, British journal the *Lancet* notes "not one of the studies provides such evidence." [Abramson J, Wright J (2007). "Are lipid-lowering guidelines evidence-based?". *Lancet* 369 (9557): 168–9. doi:10.1016/S0140-6736(07)60084-1. PMID 17240267]

Journalists have questioned the interests of the doctors who made such recommendations, as eight out of the nine doctors on the panel were discovered to have been paid by statin manufacturers. [Associated Press (16 October 2004). "Cholesterol guidelines become a morality play". *USA Today*]

All-cause mortality is highest when total serum cholesterol is lowest (forget about "good" and "bad" cholesterol).

Cholesterol is essential for mobility and the nervous system.

The brain is 2% of our body weight; but holds a massive 25% of our total cholesterol. Why would the brain need so much cholesterol? It's essential for neurotransmitter transport.

In the brain:

Cholesterol works at the synapses to promote cell-to-cell communication

Cholesterol is integral with the myelin sheath which coats and insulates our nerve axons, preserving the channel from signal loss

In the cells:

It protects from oxidative damage and ion leaks

LDL the so-called "bad" cholesterol:

Needed for protecting fats during transport in the blood and preventing oxidative damage

LDL is an antibacterial agent

Cholesterol is a precursor of many important hormones, such as testosterone, estrogen, cortisol, etc.

Cholesterol aids in the digestion of fats (via bile)

I aim to get my cholesterol levels high and keep them that way. Anything under 200 mgm is probably risky (when I was a medical student 260 mgm or thereabouts was "normal"). The poor guys boasting their cholesterol is 160 mgm or so will have trouble getting erections and surviving stress in years to come.

It's too early to say if low cholesterol increases the risk of cancer—nobody is looking at that! But we need cortisol to cope with stress, cancer is a disease of stress and statins lower cortisol production. Low cholesterol inhibits vitamin D production; vitamin D is one of the most powerful and fully proven anti-cancer substances know: so go figure.

I did find one cancer-related study. It had to do with the wasting away of patients with cancer; a condition called "cachexia". The effects of Simvastatin were studied:

Conclusion: "Simvastatin administration, although capable of negatively modulating the inflammatory response, did not prevent muscle wasting in this experimental model of cancer cachexia. Moreover, the further muscle loss observed in simvastatin-treated tumor-bearing animals suggests that a note of caution should be introduced in treating cancer patients with statins in view of the possible occurrence of harmful side effects."

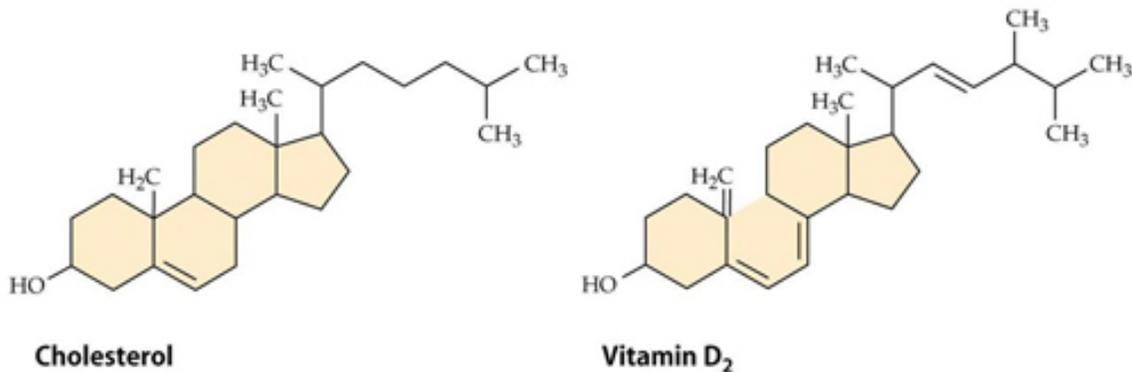
[Muscaritoli et al., Nutrition 19:11, 936-939, 2003]

A clear warning of possible dire consequences therefore.

The Vitamin D Connection

Vitamin D is one of the most vital of all vitamins for the competency of our immune systems.

Cholesterol is virtually identical with vitamin D.



Look closely and see if you can tell the difference!

Vitamin D is synthesized from cholesterol in the skin upon exposure to sunlight.

You mess with this critical biological compound at your peril.

Now the vitamin D paradox! While coenzymeQ10 is significantly decreased in response to statin treatment (see below), vitamin D levels actually increase. We don't know why, except it is likely to be due to this chemical relationship between vitamin D and cholesterol. Maybe

statins work like vitamin D? It's been suggested. My own view is that it is likely that the cholesterol pathway is blocked and so metabolites divert sideways, into vitamin D.

This is probably where the little good the statins do for that small group of middle-aged men with a heart attacks comes from. We do know (from real science, not "statin science") that vitamin D has significant cardioprotective effects.

[Grimes, D. (2006). "Are statins analogues of vitamin D?". The Lancet 368 (9529): 83–82. doi:10.1016/S0140-6736(06)68971-X]

Why Statins Are Bad

Whether or not fooling around with raised cholesterol is a bad idea, *statins still don't add up*.

Fructose, we all know, is bad. High fructose corn syrup is killing Western populations right now. The liver has to detoxify fructose and it does so by turning it into fat (so fructose = obesity). Fat is exported to the tissues, packaged in LDL particles. So LDL isn't really bad cholesterol; it's fructose which is the problem and causes raised LDL and increased risk of death.

Cholesterol is therefore needed to safely keep fats out of circulation, especially synthetic fats.

If there is not enough cholesterol in circulation, the liver struggles and switches to another pathway: lactate (partially anaerobic, Warburg fans, please note).

Lactate actually makes quite good heart food, which is probably why the heart benefits. But the rest of our muscles are wrecked. Rhabdomyolysis, one of the deadly complications of statin therapy, start with this side pathway.

More of that later.

More Bad News From Statins

Statins increase absorption of plant sterols x2. Plant sterols are highly oxidized (140x more than cholesterol). They also promote macrophage apoptosis (programmed cell death); that's the last thing you want! Macrophages are among the chief hunters of the immune system, gobbling up pathogens, including cancer cells.

Plant sterols also cause plaque destabilization; again, not something you really want. Arterial plaque isn't so dangerous, when it is stable. But when it suddenly ruptures, that's when you get the fatal heart attack that fells a person on the street or in the office etc.

Statins especially increase risk of heart attack in sterol "hyperabsorbers"—people who absorb more than usual quantities of sterols. Hyperabsorbers are estimated as 25% of population; that's 1 in 4.

Dietary plant sterol enrichment and statin therapy are additive in effect. So taking statins bars you from good healthy, fresh food. It could be dangerous. Maybe that explains the next section...

Lovastatin treatment in mice caused a profound defect in immune system dendritic cells, leading to an impaired ability to fight infection [Domínguez PM, López-Bravo M, Kalinke U, Ardavín C. Eur J Immunol. 2011 Nov; 41(11):3330-9]

Statin therapy is associate Dominguez with increased risk of developing lupus [de Jong et al., Semin Arthritis Rheum. 2011]

Statins Age You Faster

We all have trouble keeping our body and brain fully functioning as we pass through middle age and beyond.

One of the sure signs that statins are aging us faster is the earlier onset of cataracts. Eye cataracts are a very accurate marker for the aging process. On average, cataracts come on 4 – 6 years sooner, in individuals taking statins. This effect is even worse in diabetics taking statins.

Also, statins lead to more AGEs, that advanced glycation products. AGEs are toxic and what we think builds up plaque in Alzheimer's patients.

You do NOT want AGEs anywhere in your body. Statins manufacture them, through disordered cell death, caused by blockage of the cell signaling pathways.

Plus, cholesterol increases insulin sensitivity, so the use of statins is likely to lead to diabetes [Goldstein and Mascitelli, Q. J. Med, 2010]. I have described elsewhere that diabetes is really a speeded up aging process: tired eyes, tired heart, bad arteries, poor kidneys, functioning mental impairment and blindness. Then there is gangrene of the extremities—much more likely in diabetics.

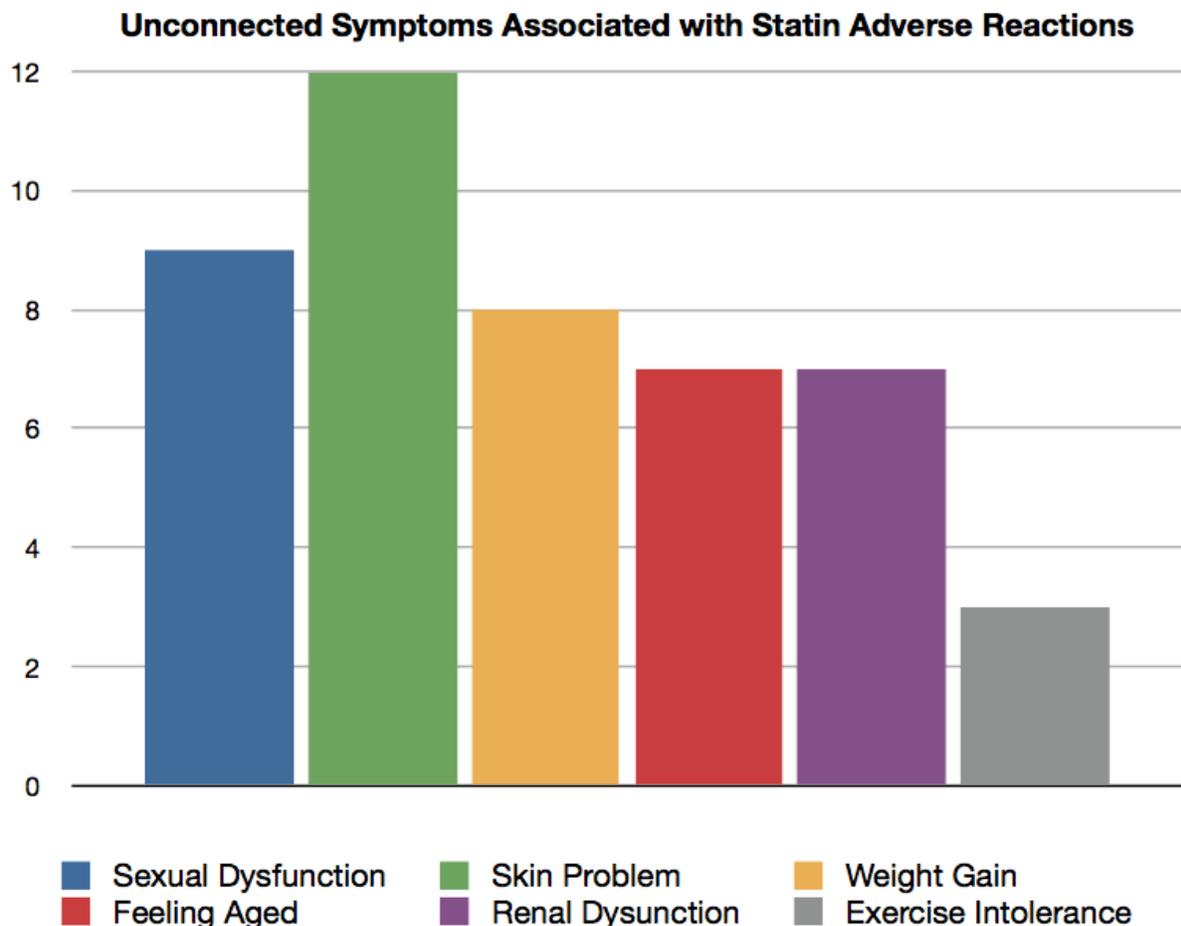
All Kinds Of Other Bad Stuff

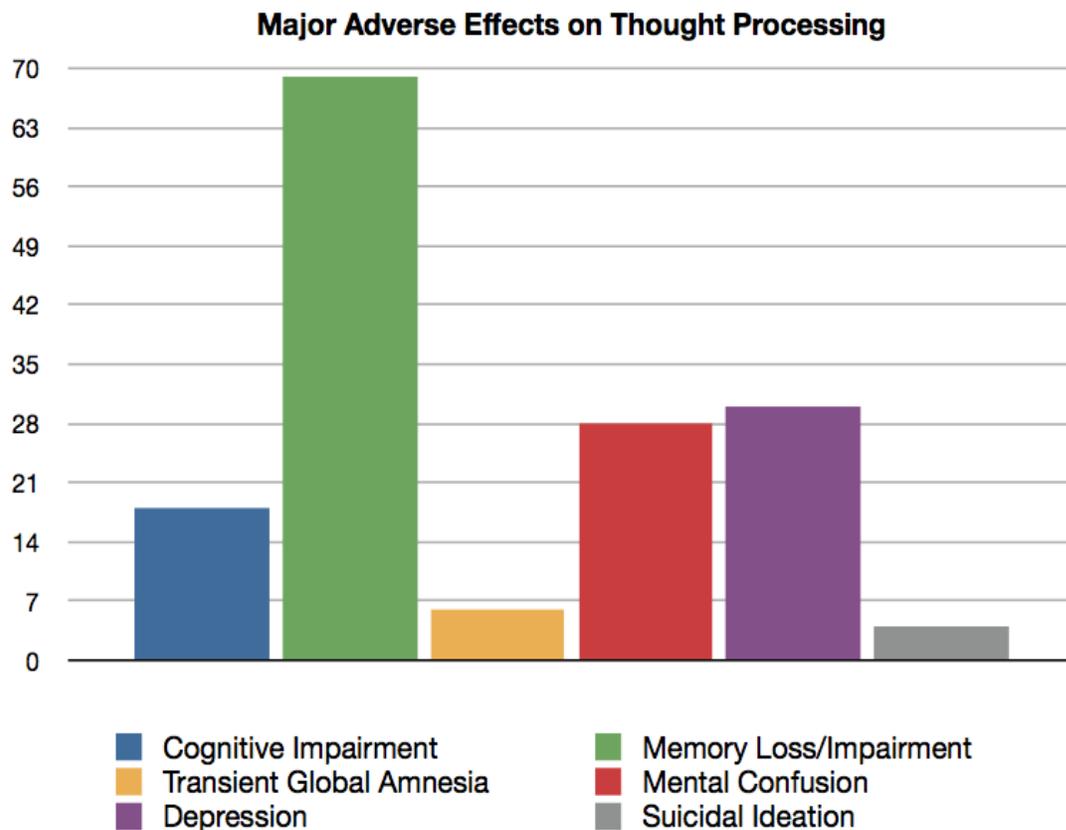
In Summary:

Statins wreck muscles by exposing them to excess fructose that the liver can no longer metabolize

- Statins interfere with exercise
- Low cholesterol is associated with depression
- Statins destroy yeast mitochondria
- Statins impair sodium and chloride ion transport in kidneys
- Statins increase risk to diabetes and lupus
- Statins are associated with a host of side effects that can be best characterized as “getting older faster”

Do I need to go on?





Statistics Tell The Real Story

OK, maybe this is just Prof. rattling on with some bee in his bonnet? Where's the proof? The numbers tell a heavy story.

Decreasing life span; who needs that?

In one 17-year study on elderly people begun in 1990, all subjects were at least 70 years old. Researchers measured three main parameters:

- serum cholesterol
- ability to synthesize cholesterol
- ability to absorb cholesterol through the intestines

Low values of all three parameters were associated with accelerated mental decline and increased physical frailty.

Subjects with low values on all three had 4½ years decreased life span!

[Tilvis et al., Annals of Medicine, Early Online, 2011]

So-Called "Former Use" Of Statins is just as deadly:

"...Former use of statins was associated with an elevated risk of all-cause dementia (HR, 1.88; 95% CI, 1.05-3.36) and AD alone (HR, 2.54; 95% CI, 1.24- 5.20) compared with never users."

Hazard ratio 1.21 for Alzheimer's for "ever used statins" vs. "never used statins." [ea et al., "Statin Use and the Risk of Incident Dementia: The Cardiovascular Health Study," Arch Neurol. 62, 2005]

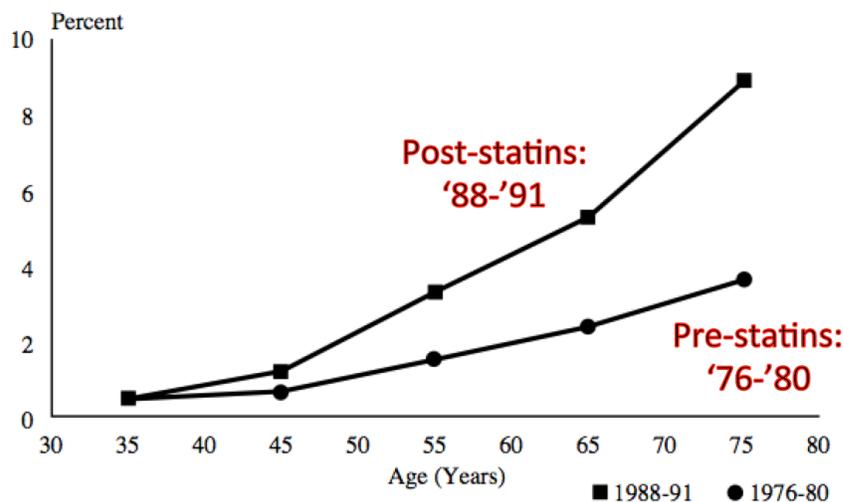
Heart Failure Made Worse:

- An estimated 5.8 million Americans have heart failure
- 670,000 new cases occur each year in the USA alone
- In 2007, America spent 33 billion dollars on heart failure
- Heart failure is our largest single drain on health care funds

Cholesterol has worsened survival due to chronic heart failure (CHF). Again, how can that be, if the drugs work as claimed? Is nobody noticing the glaring anomaly?

See the graph below:

Prevalence of CHF, by Age, 1976-80 and 1988-91



Source: National Health and Nutrition Examination Survey (1976-80 and 1988-91), National Center for Health Statistics.

National Heart, Lung, and Blood Institute National Institutes of Health Data Fact Sheet

Heart failure means that the heart is tired and weak, and can no longer pump blood efficiently

Heart failure patients have low serum Coenzyme Q10 (see next section).

Among heart failure patients, higher Co-Q10 is associated with lower mortality risk. We sure need Co-Q10 [S. Sinatra, <http://www.faim.org/guestwriters/sinatraheartfailureroundup.html>].

But also—look at this—low serum cholesterol is associated with increased mortality [Kalantar-Zadeh et al., *J. American College of Cardiology* 43(8) 1439-1444, 2004].

Statins interfere with synthesis of both Co-Q10.

Bad; very bad.

Statins cause depression:

Low cholesterol can be associated with depression and other mood disorders. This is not surprising, since cholesterol is vital to the integrity and function of nerve tissue (cell walls are all fatty in nature). It plays a role in nerve conduction, brain function, and hence mood.

In one study, researchers demonstrated that increased HDL was negatively associated with mood while higher LDL and triglycerides were positively associated with mood [Lieberman HR, Kellogg MD, Kramer FM, et al. Lipid and other plasma markers are associated with anxiety, depression, and fatigue. *Health Psychol.* 2011 Dec 12].

Another study evaluated 4,115 men and 4,275 women aged 18 or older for serum lipid levels and mood. The results demonstrated a U-shaped curve, meaning that both low and high serum lipids were associated with a negative mood. In fact, the strongest association was found in men with low LDL cholesterol, showing over 5-times the likelihood of having mood changes with LDL levels below 169 mg/dL [Teddens SH, Fokong KD, McKenzie LE, et al. Low cholesterol is associated with depression among US household population. *J Affect Disord.* 2011 Dec;135(1-3):115-21.]

Once again, more is better (with cholesterol)!

The CoenzymeQ10 Connection

One of the most important problems with statins is that they deplete coenzymeQ10, an essential anti-aging nutrient, which occurs in particularly high levels the heart muscle. The heart needs a lot of CoQ10; so statins do not really help the heart at all (which is why heart attacks, death from all causes and deaths from heart failure are rapidly rising, even with statin usage).

Depletion of CoenzymeQ10 interferes with energy generation in mitochondria; that's serious.

Both CoQ10 and cholesterol are synthesized from the same substance, mevalonate. Vitamin D3, estrogen, testosterone, progesterone, cortisol, bile acids, cholesterol and CoQ10 are all downstream from mevalonate. So the blocking of CoQ10 is not a "side effect," of statins, but a direct, inherent function of the drugs.

That's what they do. In fact, the use of statins can decrease the body's synthesis of CoQ10 by as much as 40%!

“The depletion of the essential nutrient CoQ10 by the increasingly popular cholesterol lowering drugs, HMG CoA reductase inhibitors (statins), has grown from a level of concern to one of alarm. ... This drug-induced nutrient deficiency is dose related and more notable in settings of pre-existing CoQ10 deficiency such as in the elderly and in heart failure.”

[Quote from abstract, Langsjoen and Langsjoen, *Biofactors* 18, 104, 101-111, 2003]

Statins also reduce the heart's contracting ability. It's a complex technical point I won't go into here (little cell membrane thingies called caveolae) but effectively statins reduce the “calcium spark” that triggers heart muscle to fire.

Yet almost everyone still believes that statins “help the heart”, just because they lower cholesterol.

The Muscle Connection

The most common side effects of statins was clearly muscle pains. Statins cause myalgia (muscle pain) and myopathy (muscle damage); the latter at its most severe causes muscle death and is called rhabdomyolysis, which means “disintegration of muscles”.

The resulting toxic tissue breakdown releases muscular proteins are disposed into the blood and this, in turn, subsequently causes kidney failure.

In randomized trials, statins increased the risk of an adverse effect by 39% compared to placebo (odds ratios 1.4); two-thirds of these were myalgia or raised liver enzymes with serious adverse effects similar to placebo [Silva MA, Swanson AC, Gandhi PJ, Tataronis GR (January 2006). “Statin-related adverse events: a meta-analysis”. *Clin Ther* 28 (1): 26–35. doi:10.1016/j.clinthera.2006.01.005. PMID 16490577].

However, reliance on clinical trials can be misleading indications of real-world adverse effects – for example, the statin cerivastatin was withdrawn from the market in 2001 due to cases of rhabdomyolysis (muscle breakdown), although rhabdomyolysis did not occur in a meta-analysis of cerivastatin clinical trials. [Golomb BA, Evans MA (2008). “Statin Adverse Effects: A Review of the Literature and Evidence for a Mitochondrial Mechanism”. *Am J Cardiovasc Drugs* 8 (6): 373–418. doi:10.2165/0129784-200808060-00004. PMC 2849981. PMID 19159124].

Other possible adverse effects include cognitive loss, neuropathy, pancreatic and hepatic dysfunction, and sexual dysfunction [ibid.]

Cholesterol Is A Great Antimicrobial

In this day and age of fear of antibiotic-resistance micro-organisms, it should be kept clearly in mind that cholesterol has a well-documented antibiotic effect.

LDL can trap and neutralize bacteria. Moreover, cholesterol in cell walls helps cells fight infection.

Methicillin-resistant *Staphylococcus aureus* (MRSA) has become a major problem worldwide. Researchers have found that Apolipoprotein B (in LDL) limits invasion by MRSA by binding to and neutralizing a key enzyme responsible for promoting invasion through the skin (in other words blocks the “flesh eating” capability).

In experiments, skin penetration and serum bacterial density were significantly greater in ApoB deficient (LDL cholesterol deficient) mice.

[Peterson et al., *Cell Host Microbe* 4(6): 555–566, 2008]

The statin industry likes to claim that statins protect from sepsis (“blood poisoning”). The exact opposite is true, because of their cholesterol-lowering effects.

The industry wants to hype the potential off-label use for the drugs as anti-microbials.

Here’s the reality:

At least 5 negative placebo-controlled studies can be found on the web:

<http://clinicaltrials.gov/ct2/show/NCT00702130>

<http://clinicaltrials.gov/ct2/show/NCT00450840>

<http://clinicaltrials.gov/ct2/show/NCT00452608>

<http://www.controlled-trials.com/ISRCTN92093279>

<http://clinicaltrials.gov/ct2/show/NCT00452608>

All five studies show no results and statins do NOT do what they claim. have no results and no publications

[Stephanie Seneff, *How Statins Really Work Explains Why They Don’t Really Work*, presentation at 40th meeting of ACAM, 2012, Las Vegas]

All this is not surprising, since statins cause pathological changes in immune response.

Mouse white blood cells cultured in vitro (in vitro means “in glass”, in other words in a test tube or dish) with statins resulted in defective immunity against pathogens:

- Reduced ability to kill microbes
- Reduced defense against reactive oxygen species

Similar results were obtained in vivo (in vivo means “in life, in other words in a living organism, not just a test tube).

[Domínguez PM, López-Bravo M, Kalinke U, Ardavin C. *Eur J Immunol.* 2011 Nov; 41(11):3330-9]

Additional Notes

Some types of statins are naturally occurring, and can be found in such foods as oyster mushrooms and red yeast rice. Randomized controlled trials found them to be effective, but the quality of the trials was low.[55]

Most of the block-buster branded statins will be generic by the end of 2012, including atorvastatin, the largest selling branded drug.

Lovastatin (Mevacor, Altacor, Altoprev) is found naturally in the oyster mushroom (*Pleurotus ostreatus*), a fine culinary mushroom, and red yeast rice.



Red yeast rice, or koji in Japanese, means 'grain or bean overgrown with a mold culture', and is a food preparation tradition going back to ca. 300 BC [Shurtleff, W.; Aoyagi, A. 2012. History of Koji - Grains and/or Beans Overgrown with a Mold Culture (300 BCE to 2012)].

Now that you know the truth about statins... I wanted to share with you surprising and effective alternatives to antibiotics in the next section.... This is something that you need to know...

The Golden Age of Antibiotics is Over!

Modern antibiotics are not just failing big time; they are DANGEROUS. Antibiotics can be lethal in their own right.

Did you know that antibiotics are the **most common cause of death** from *acute* liver failure? (acute means sudden and unforeseen; as opposed to "**chronic**", which everyone can see coming).

The truth, is statistics show that antibiotics are the **single largest class of drugs** that cause fatal liver damage.

Yes, antibiotics were the most common cause of death due to liver injury (45%). The next group, nervous system agents, were only 1/3rd as common (15%). That puts antibiotics way out on their own as deadly drugs.

Chief offenders are macrolides (erythromycin type), penicillins, clavulanic acid (Augmentin), tetracycline, and more.

I'm constantly amazed when I hear of colleagues prescribing erythromycin as a first line medicine for a sore throat. *Don't they know how toxic this stuff is?*

It had a bad reputation when I was in med school in the 1960s. Doesn't anybody pay attention? If it's to be used at all, it's in a life-threatening situation where penicillin's and safer drugs have failed.

The bottom line is nobody should be thinking "antibiotics". We should be thinking of "alternatives". And here's the joke: there are 1,000s of viable alternatives. Hundreds of them work as well as antibiotics.

The Golden Age of Antibiotics is over. Bacteria have won the war, hands down, and that's the truth. We're all out of ammunition! The cupboard is bare. *Well, not quite.* There are, as I said, hundreds of healthy, safe and EFFECTIVE alternative modalities of treatment. You need to find out about them and learn NOW, not when somebody is in bed with life-threatening pneumonia (non-hospital pneumonia, which is the 4th most common cause of death in the UK; 6th commonest in the USA).

[Click here to get the knowledge you need](#) to stay safe and take care of your loved ones. [Go here](#) to learn what you need to know to [SURVIVE IN A WORLD WITHOUT ANTIBIOTICS](#).

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