

Statin -- Side Effects Studies

Adverse Side Effects from Long Term Use of Statin Drugs.

What do statins do?

Statins (formally called HMG-CoA reductase inhibitors, because of the enzyme which they inhibit from being produced) work by cutting off a pathway in your body which ultimately results in the production of cholesterol. But, as with most chemicals, it knows no boundaries and along with the blocking of this enzyme there are a cascade of problems in that a whole family of intermediary substances many if not all have very important jobs to do in your body are also blocked.

There have been cases of people born with an inherent defect to make mevalonate (this is the first step to our body's making cholesterol). These children are mentally handicapped, anemic, have frequent fevers, etc. This would represent the extreme in low cholesterol levels you do not want to be there.

One of the major problems is that Coenzyme-Q10 production is also halted. This is essential in the production of ATP our basic energy molecule produced within the cells.

The heart muscle and our cell's membranes for nerve and muscle integrity require a constant, high level of Co-Q10.

Co-Q10 is also vital to the production of elastin and collagen the basis for the structure of skin, muscle and tendons.

This is the reason that one of the most serious effects of statin use is back and muscle pain, weakness, inflammation of tendons and ligaments even to the point of rupture.

Active people such as athletes are more affected by statins than are the sedentary type. People with fibromyalgia, chronic fatigue, etc. *should not go near these drugs.* as they would make their problems worse.

Heart symptoms and problems include *HEART ATTACK!!!* Is not that what we are trying to prevent?

Dolichols also play a role of immense importance. In the cells they direct various proteins manufactured in response to DNA directives to their proper targets, ensuring that the cells respond correctly to genetically programmed instruction. Thus statin drugs can lead to unpredictable chaos on the cellular level, much like a computer virus that wipes out certain pathways or files.

Squalene, the immediate precursor to cholesterol, has anti-cancer effects, according to research.

Very recent research suggests that statins in the short term may help decrease actual heart disease but this probably because it acts as an antioxidant NOT because it blocks cholesterol production.

From Dr. Mary Enig and Sally Fallon in their Statin report...Dr. Enig has summarized some of the more popular and ‘telling’ studies:

Honolulu Heart Program (2001)	This report, part of an ongoing study, looked at cholesterol lowering in the elderly. Researchers compared changes in cholesterol concentrations over 20 years with all-cause mortality. To quote: "Our data accords with previous findings of increased mortality in elderly people with low serum cholesterol, and show that long-term persistence of low cholesterol concentration actually increases risk of death. Thus, the earlier that patients start to have lower cholesterol concentrations, the greater the risk of death. . . The most striking findings were related to changes in cholesterol between examination three (1971-74) and examination four (1991-93). There are few studies that have cholesterol concentrations from the same patients at both middle age and old age. Although our results lend support to previous findings that low serum cholesterol imparts a poor outlook when compared with higher concentrations of cholesterol in elderly people, our data also suggest that <i>those individuals with a low serum cholesterol maintained over a 20-year period will have the worst outlook for all-cause mortality</i> [emphasis ours]."
ALLHAT (2002)	ALLHAT (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial), the largest North American cholesterol-lowering trial ever and the largest trial in the world using Lipitor, showed

	<p>mortality of the treatment group and controls after 3 or 6 years was identical. Researchers used data from more than 10,000 participants and followed them over a period of four years, comparing the use of a statin drug to “usual care,” namely maintaining proper body weight, no smoking, regular exercise, etc., in treating subjects with moderately high levels of LDL cholesterol. Of the 5170 subjects in the group that received statin drugs, 28 percent lowered their LDL cholesterol significantly. And of the 5185 usual-care subjects, about 11 percent had a similar drop in LDL. <i>But both groups showed the same rates of death, heart attack and heart disease.</i></p>
<p>Heart Protection Study (2002)</p>	<p>Carried out at Oxford University, this study received widespread press coverage; researchers claimed “massive benefits” from cholesterol-lowering, leading one commentator to predict that statin drugs were “the new aspirin.” But as Dr. Ravnskov points out, the benefits were far from massive. Those who took simvastatin had an 87.1 percent survival rate after five years compared to an 85.4 percent survival rate for the controls and these results were independent of the amount of cholesterol lowering. The authors of the Heart Protection Study never published cumulative mortality data, even though they received many requests to do so and even though they received funding and carried out a study to look at cumulative data. According to the authors, providing year-by-year mortality data would be an “inappropriate” way of publishing their study results.</p>
<p>PROSPER (2001)</p>	<p>PROSPER (Prospective Study of Pravastatin in the Elderly at Risk) studied the effect of pravastatin compared to placebo in two older populations of patients of which 56 percent were primary prevention cases (no past or symptomatic cardiovascular disease) and 44 percent were secondary prevention cases (past or symptomatic cardiovascular disease). Pravastatin did not reduce total myocardial infarction or total stroke in the primary prevention population but did so in the secondary. However, measures of overall health impact in the combined populations, total mortality and total serious adverse events were unchanged by pravastatin as compared to the placebo and those in the treatment group had increased cancer. In other words: not one life saved.</p>
<p>Statins and Plaque (2003)</p>	<p>A study published in the <i>American Journal of Cardiology</i> casts serious doubts on the commonly held belief that lowering your LDL-cholesterol, the so-called bad cholesterol, is the most effective way to reduced arterial plaque. Researchers at Beth Israel Medical Center in New York City examined the coronary plaque buildup in 182 subjects who took statin drugs to lower cholesterol levels. One group of</p>

	<p>subjects used the drug aggressively (more than 80 mg per day) while the balance of the subjects took less than 80 mg per day. Using electron beam tomography, the researchers measured plaque in all of the subjects before and after a study period of more than one year. The subjects were generally successful in lowering their cholesterol, but in the end there was no statistical difference in the two groups in the progression of arterial calcified plaque. On average, subjects in <i>both</i> groups showed a 9.2 percent increase in plaque <i>buildup</i>.</p>
ASCOT-LLA (2003)	<p>ASCOT-LLA (Anglo-Scandinavian Cardiac Outcomes Trial—Lipid Lowering Arm) was designed to assess the benefits of atorvastatin (Lipitor) versus a placebo in patients who had high blood pressure with average or lower-than-average cholesterol concentrations and at least three other cardiovascular risk factors. The trial was originally planned for five years but was stopped after a median follow-up of 3.3 years because of a significant reduction in cardiac events. Lipitor did reduce total myocardial infarction and total stroke; however, total <i>mortality</i> was not significantly reduced. In fact, women were worse off with treatment. The trial report stated that total serious adverse events “did not differ between patients assigned atorvastatin or placebo,” but did not supply the actual numbers of serious events.</p>
Statins and Women (2003)	<p>No study has shown a significant reduction in mortality in women treated with statins. The University of British Columbia Therapeutics Initiative came to the same conclusion, with the finding that statins offer no benefit to women for prevention of heart disease. Yet in February of 2004, <i>Circulation</i> published an article in which more than 20 organizations endorsed cardiovascular disease prevention guidelines for women with several mentions of “preferably a statin.”</p>

What if someone were to tell you that there was a measurable substance in your body that is theoretically the most important indicator of the health and adaptability of your body’s total biochemistry and your risk of degenerative diseases? Sound intriguing – that’s what is being said about Homocysteine. Recent research is discovering that high levels of homocysteine can damage arteries, the brain and DNA. Elevated homocysteine has been proven to increase the formation of plaques on blood vessel walls leading to clogging and hardening of the arteries. David Wald and colleagues from the Department of Cardiology at Southampton General Hospital (England) published in the British Medical Journal their findings of a study conducted on 20,000 people. They

concluded that there is strong evidence demonstrating a causal relationship between homocysteine and cardiovascular disease.

If you lower your homocysteine level you reduce your risk of developing cardiovascular disease. In addition, with regard to brain degeneration, studies have shown that lowering your Homocysteine levels will significantly lower your risk of getting Alzheimer's disease by at least half. It has also been shown that high levels of homocysteine causes DNA damage. Since DNA damage is a precursor to cancer, lowering your homocysteine level will reduce your risk of getting cancer. Cancers linked to high homocysteine levels include breast, colon and leukaemia. Other diseases linked to high homocysteine levels include diabetes and Rheumatoid arthritis.

There are risk factors that predispose individuals to being vulnerable to high homocysteine levels, they include; genetics (One in ten people carries a genetic mutation that makes them more prone to higher homocysteine levels than others), family history heart disease, stroke, cancer, Alzheimer's disease, schizophrenia or diabetes, foliate intake of less than 900 mcg/day, increasing age, male sex, estrogen deficiency, excessive alcohol, coffee or tea intake, smoking, lack of exercise, hostility and repressed anger, inflammatory bowel diseases, H. pylori-generated ulcers, pregnancy, vegetarian or vegan diet, high fat diet with excessive red meat, high fat dairy intake and high salt intake.

Homocysteine is naturally produced in most of the body's cells. It is derived from Methionine which is an amino acid found in dietary protein. It aids in tissue and cell growth and insulin formation and can act like growth hormone. The body turns homocysteine into glutathione and SAME. Glutathione is the body's most important antioxidant while SAME (S-adenosylmethionine) is a very important "intelligent" nutrient for the brain and body. Your antioxidant IQ is a measure of the glutathione and SAME inside your cells.

If you have suboptimal amounts of B vitamins in your diet, homocysteine cannot be converted and the levels rise dangerously.

The method of lowering Homocysteine involves a molecular process called 'methylation'. Methylation simply means that molecules add or subtract methyl groups. Methylation processes are necessary for the body to maintain optimal chemical balance. High levels of Homocysteine can be reduced by consuming substances that will donate methyl groups. These donated methyl groups will turn toxic homocysteine into SAMe, which, as stated before, is an important nutrient for the brain. Choline and TMG (or betaine) is excellent methyl donors which help reduce high homocysteine. Choline is found in eggs and lecithin while TMG is found in sugar beets and other vegetables. In addition, three vitamins, B6, B12 and Folate are required to metabolize homocysteine.