

Cholesterol Does Not Cause Heart Disease

Analysis by [Dr. Joseph Mercola](#)

✓ Fact Checked

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STORY AT-A-GLANCE

- › A 2018 scientific review presents substantial evidence that high LDL and total cholesterol are not an indication of heart disease risk, and that statin treatment is of doubtful benefit as a form of primary prevention for this reason
- › Three recent that supported the cholesterol hypothesis were found to have misrepresented data and findings of previous studies to support their own conclusions
- › Overall, the analysis found the association between total cholesterol and CVD is weak, absent or inverse in many studies
- › Older people with high LDL do not die prematurely – they actually live the longest, outliving both those with untreated low LDL and those on statin treatment
- › A 2015 meta-analysis of 11 statin drug studies found statin use postponed death by a mere 3.2 days in primary prevention trials and 4.1 days in secondary prevention trials

This article was previously published July 24, 2019, and has been updated with new information.

For the past six decades, the U.S. dietary advice has warned against eating cholesterol-rich foods, claiming dietary cholesterol promotes arterial plaque formation that leads to heart disease. We now have overwhelming evidence to the contrary, yet dogmatic thinking can be persistent, to say the least.

After decades' worth of research failed to demonstrate a correlation between dietary cholesterol and heart disease, the 2015-2020 Dietary Guidelines for Americans^{1,2} finally addressed this scientific shortcoming, announcing "cholesterol is not considered a nutrient of concern for overconsumption." Unfortunately, the 2020-2025 guidelines do not include that statement and, instead, now states:³

"The National Academies recommends that trans fat and dietary cholesterol consumption to be as low as possible without compromising the nutritional adequacy of the diet. The USDA Dietary Patterns are limited in trans fats and low in dietary cholesterol."

While I agree that trans fats should be limited or even eliminated from your diet, it is absurd for the USDA to revert to its old recommendations, as cholesterol is NOT the cause of heart disease.

To this day, the evidence keeps mounting, showing there's no link between cholesterol and heart disease. Similarly, the evidence supporting the use of cholesterol-lowering statin drugs to lower your risk of heart disease is slim to none, and is likely little more than the manufactured work of statin makers – at least that's the implied conclusion of a scientific review⁴ published in the Expert Review of Clinical Pharmacology in 2018.

Cholesterol Myth Kept Alive by Statin Advocates?

The 2018 review⁵ identified significant flaws in three studies "published by statin advocates" attempting "to validate the current dogma." The paper presents substantial evidence that total cholesterol and low-density lipoprotein (LDL) cholesterol levels are not an indication of heart disease risk, and that statin treatment is of "doubtful benefit" as a form of primary prevention for this reason. According to the authors:⁶

"According to the British-Austrian philosopher Karl Popper, a theory in the empirical sciences can never be proven, but it can be shown to be false. If it cannot be falsified, it is not a scientific hypothesis. In the following, we have

followed Popper's principle to see whether it is possible to falsify the cholesterol hypothesis.

We have also assessed whether the conclusions from three recent reviews by its supporters are based on an accurate and comprehensive review of the research on lipids and cardiovascular disease (CVD) ...

Our search for falsifications of the cholesterol hypothesis confirms that it is unable to satisfy any of the Bradford Hill criteria for causality and that the conclusions of the authors of the three reviews are based on misleading statistics, exclusion of unsuccessful trials and by ignoring numerous contradictory observations."

As reported by Reason.com:⁷

"A comprehensive new study on cholesterol, based on results from more than a million patients, could help upend decades of government advice about diet, nutrition, health, prevention, and medication ...

The study ... centers on statins, a class of drugs used to lower levels of LDL-C, the so-called 'bad' cholesterol, in the human body. According to the study, statins are pointless for most people ...

The study also reports that 'heart attack patients were shown to have lower than normal cholesterol levels of LDL-C' and that older people with higher levels of bad cholesterol tend to live longer than those with lower levels."

No Evidence Cholesterol Influences Heart Disease Risk

Indeed, the authors of the Expert Review of Clinical Pharmacology analysis point out that were high total cholesterol in fact a major cause of atherosclerosis, "there should be exposure-response in cholesterol-lowering drug trials."⁸ In other words, patients whose total cholesterol is lowered the most should also see the greatest benefit. Alas, that's not the case.

A review of 16 relevant cholesterol-lowering trials (studies in which exposure-response was actually calculated), showed this kind of exposure-response was not detected in 15 of them. What's more, the researchers point out that the only study⁹ showing a positive exposure-response to lowered cholesterol used exercise-only as the treatment.

Patients with high total cholesterol should also be at increased risk of death from CVD, but the researchers found no evidence of this either, not-so-subtly pointing out that this is "an idea supported by fraudulent reviews of the literature." They provide the following example of how research has been misrepresented:¹⁰

"The hypothesis that high TC [total cholesterol] causes CVD was introduced in the 1960s by the authors of the Framingham Heart Study. However, in their 30-year follow-up study published in 1987, the authors reported that 'For each 1 mg/dl drop in TC per year, there was an eleven percent increase in coronary and total mortality'.

Three years later, the American Heart Association and the U.S. National Heart, Lung and Blood Institute published a joint summary concluding, 'a one percent reduction in an individual's TC results in an approximate two percent reduction in CHD risk'. The authors fraudulently referred to the Framingham publication to support this widely quoted false conclusion."

Contradictory Findings Routinely Ignored or Misrepresented

To determine whether the three reviews under analysis had misrepresented previous findings, they scoured the three papers for quotations from 12 studies reporting results "discordant with the cholesterol hypothesis." Only one of the three reviews had quoted articles correctly, and even then, only two of the dozen studies were quoted correctly.¹¹

"About half of the contradictory articles were ignored. In the rest, statistically nonsignificant findings in favor of the cholesterol hypothesis were inflated, and unsupportive results were quoted as if they were supportive. Only one of the six

randomized cholesterol-lowering trials with a negative outcome was cited and only in one of the reviews."

The researchers also highlight a large meta-analysis that simply ignored "at least a dozen studies" in which no or inverse association was shown. Overall, the Expert Review of Clinical Pharmacology analysis found that "the association between total cholesterol and CVD is weak, absent or inverse in many studies."

No Link Between LDL and Heart Disease Either

The Expert Review of Clinical Pharmacology paper¹² also tears apart claims that high LDL causes atherosclerosis and/or CVD. Just as with total cholesterol, if high LDL was in fact responsible for atherosclerosis, then patients with high LDL would be diagnosed with atherosclerosis more frequently, yet they're not, and those with the highest levels would have the greatest severity of atherosclerosis, yet they don't.

The researchers cite studies showing "no association" between LDL and coronary calcification or degree of atherosclerosis. Ditto for LDL and CVD. In fact, a study looking at nearly 140,000 patients with acute myocardial infarction found them to have lower than normal LDL at the time of admission.

Even more telling, another study, which had originally reported similar findings, still went ahead and lowered the patients' LDL even more. At follow-up three years later, they discovered that patients with an LDL level below 105 mg/dl (2 mmol/L) had double the mortality rate of those with higher LDL.¹³

Interestingly, the authors suggest this inverse relationship may be due to low LDL increasing your risk for infectious diseases and cancer, both of which are common killers.

They also review evidence showing older people with high LDL do not die prematurely — they actually live the longest, outliving both those with untreated low LDL and those on statin treatment. One such study¹⁴ — a meta-analysis of 19 studies — found 92% of individuals with high cholesterol lived longer.

Benefits of Statin Treatment Are Overblown

Lastly, the Expert Review of Clinical Pharmacology paper analyzes statin claims, showing how studies exaggerate benefits through a variety of different tactics. Again, in some cases, by simply excluding unsuccessful trials.

"Furthermore, the most important outcome – an increase of life expectancy – has never been mentioned in any cholesterol-lowering trial, but as calculated recently by Kristensen et al.,¹⁵ statin treatment does not prolong lifespan by more than an average of a few days," the authors state.¹⁶

Indeed, the study they're referring to, published in BMJ Open in 2015, which looked at 11 studies with a follow-up between two and 6.1 years, found "Death was postponed between -5 and 19 days in primary prevention trials and between -10 and 27 days in secondary prevention trials." The median postponement of death in primary prevention trials was 3.2 days, and in secondary prevention trials 4.1 days!

Considering the well-documented health risks associated with statins, this is a mind-bending finding that really should upend the dogma. And yet, the dogma remains, and may even strengthen in coming days.

JAMA Editorial Calls for End to 'Fake News' About Statins

The cholesterol myth has been a boon to the pharmaceutical industry, as cholesterol-lowering statins – often prescribed as a primary prevention against heart attack and stroke – have become one of the most frequently used drugs on the market. In 2012-2013, 27.8% of American adults over the age of 40 reported using a statin, up from 17.9% a decade earlier.^{17,18} But that was six years ago, I suspect over a third of adults over the age of 40 are now using statins.

In addition to the BMJ Open study cited above, an evidence report¹⁹ by the U.S. Preventive Services Task Force, published November 2016 in JAMA, found 250 people need to take a statin for one to six years to prevent a single death from any cause; 233

had to take a statin for two to six years to prevent a single cardiovascular death specifically. To prevent a single cardiovascular event in people younger than 70, 94 individuals would have to take a statin.

As noted in a 2015 report,²⁰ "statistical deception created the appearance that statins are safe and effective in primary and secondary prevention of cardiovascular disease." The paper points out that by using a statistical tool known as relative risk reduction, the trivial benefits of statins appear greatly amplified.

Scientific findings such as these are the core reason why statins are given negative press. However, we may soon see a reversal in the news cycle, with negative statin articles being tagged as "fake news."

According to a June 2019 editorial²¹ in JAMA Cardiology, written by cardiologist Dr. Ann Marie Navar,²² statins are the victim of "fear-based medical information," just like vaccines, and this is what's driving patient nonadherence. Cardiovascular Business reported:²³

"We know that what people read influences their actions, Navar said, and indeed, one 2016 study in the European Heart Journal found that on a population level, statin discontinuation increased after negative news stories about statins surfaced in those communities.

In another study, more than one in three heart patients said they declined a statin prescription solely for fears of adverse effects. 'Measles outbreaks are highly visible: a rash appears, public health agencies respond, headlines are made and the medical community responds vocally,' Navar wrote.

'In contrast, when a patient who has refused a statin because of concerns stoked by false information has an MI, the result is less visible. Nevertheless, cardiologists and primary care physicians observe the smoldering outbreak of statin refusal daily.'

Cardiovascular Business summarizes Navar's suggestions for how doctors can fight back against false information about statins and build adherence, such as handing out yearlong prescriptions with automatic refills.²⁴

When I first wrote about the censorship of anti-vaccine material occurring on every single online platform, I warned that this censorship would not stop at vaccines. And here we're seeing the call for censoring anti-statin information by glibly labeling it all "fake news."

Chances are, the censoring of anti-statin information is already underway. A quick Google search for "statin side effects" garnered pages worth of links talking about minor risks, the benefits of statins, comparison articles, looking at two different brands – in other words, mostly positive news.

The scientific fact is, aside from being a "waste of time" and not doing anything to reduce mortality, statins also come with a long list of potential side effects and clinical challenges, including:

An increased risk for diabetes

Decreased heart function²⁵

Nutrient depletions – Including CoQ10 and vitamin K2, both of which are important for cardiovascular and heart health

Impaired fertility – Importantly, statins are a Category X medication,²⁶ meaning they cause serious birth defects,²⁷ so they should never be used by a pregnant woman or women planning a pregnancy

Increased risk of cancer – Long-term statin use (10 years or longer) more than doubles women's risk of two major types of breast cancer: invasive ductal carcinoma and invasive lobular carcinoma²⁸

Nerve damage – Research has shown statin treatment lasting longer than two years

causes "definite damage to peripheral nerves"²⁹

How to Assess Your Heart Disease Risk



As a general rule, cholesterol-lowering drugs are not required or prudent for the majority of people – especially if both high cholesterol and longevity run in your family. Remember, the evidence overwhelmingly suggests your overall cholesterol level has little to nothing to do with your risk for heart disease.

For more information about cholesterol and what the different levels mean, take a look at the infographic above. As for evaluating your heart disease risk, the following tests will provide you with a more accurate picture of your risk:

HDL/Cholesterol ratio – HDL percentage is a very potent heart disease risk factor. Just divide your HDL level by your total cholesterol. That percentage should ideally be above 24%.

Triglyceride/HDL ratio – You can also do the same thing with your triglycerides and HDL ratio. That percentage should be below 2.

NMR LipoProfile – Large LDL particles do not appear to be harmful. Only small dense LDL particles can potentially be a problem, as they can squeeze through the lining of your arteries. If they oxidize, they can cause damage and inflammation.

Some groups, such as the National Lipid Association, are now starting to shift the focus toward LDL particle number instead of total and LDL cholesterol to better assess your heart disease risk. Once you know your particle size numbers, you and your doctor can develop a more customized program to help manage your risk.

Your fasting insulin level – Heart disease is primarily rooted in insulin resistance,³⁰ which is the result of a high-sugar diet. Sugar, not cholesterol or saturated fat, is the primary driver. Clinical trials have shown **high fructose corn syrup** can trigger risk factors for cardiovascular disease within as little as two weeks.³¹

Any meal or snack high in carbohydrates like fructose and refined grains generates a rapid rise in blood glucose and then insulin to compensate for the rise in blood sugar.

The insulin released from eating too many carbs promotes fat accumulation and makes it more difficult for your body to shed excess weight. Excess fat, particularly around your belly, is one of the major contributors to heart disease.

Your fasting blood sugar level – Research has shown people with a fasting blood sugar level of 100 to 125 mg/dl have a nearly 300% increased higher risk of coronary heart disease than people with a level below 79 mg/dl.^{32,33}

Your iron level – Iron can be a very potent oxidative stress, so if you have excess iron levels you can damage your blood vessels and increase your risk of heart disease. Ideally, you should monitor your ferritin levels and make sure they are not much above 80 ng/ml.

The simplest way to lower them if they are elevated is to donate your blood. If that is not possible you can have a therapeutic phlebotomy and that will effectively eliminate the excess iron from your body.

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