The Diet-Heart Myth

by Chris Kresser
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It’s hard to overstate the impact that cardiovascular disease (CVD) has in the United States. Consider the following:

- Cardiovascular disease affects 65 million Americans.
- Close to one million Americans have a heart attack each year.
- In the U.S., one person dies every 39 seconds of cardiovascular disease.
- 1 of 3 deaths that occurs in the U.S. is caused by cardiovascular disease.
- 1 in 3 Americans have metabolic syndrome, a cluster of major cardiovascular risk factors related to overweight/obesity and insulin resistance.
- The total cost of cardiovascular disease in 2008 was estimated at $300 billion.

To put that last statistic in perspective, the World Health Organization has estimated that ending world hunger would cost approximately $195 billion. One might argue that the $300 billion we spend on treating cardiovascular disease in the U.S. is a necessary expenditure; however, a recent study which looked at the relationship between heart disease and lifestyle suggested that 90% of CVD is caused by modifiable diet and lifestyle factors. (1)

Unfortunately, cardiovascular disease is one of the most misdiagnosed and mistreated conditions in medicine. We’ve learned a tremendous amount about what causes heart disease over the past decade, but the medical establishment is still operating on outdated science from 40-50 years ago.

For the last half century, the medical establishment has vigorously promoted the notion that high cholesterol is a primary risk factor for coronary heart disease, and that a diet high in saturated fat and cholesterol causes heart disease. These hypotheses are widely accepted as fact by many physicians and the general public alike, despite the overwhelming body of evidence that suggests otherwise.

In this eBook, I will review over fifty years of research demonstrating that:
1. High cholesterol is not the primary cause of heart disease.
2. Diets high in saturated fat and cholesterol don’t cause heart disease.
3. Consumption of so-called “heart healthy” vegetable oils is linked to heart disease, cancer and many other conditions.
4. Statin drugs don’t reduce the risk of death for most people, and have dangerous side effects and complications.

I will also discuss the latest theories on what causes heart disease and a truly “heart healthy” approach to diet and lifestyle that is supported by both modern science and centuries of traditional wisdom.

**Cholesterol and Saturated Fat Are Not the Enemy**

Most of us grew up being told that foods like red meat, eggs and bacon raise our cholesterol levels. This idea is so deeply ingrained in our cultural psyche that few people even question it. But is it really true?

The diet-heart hypothesis—which holds that eating cholesterol and saturated fat raises cholesterol in our blood—originated with studies in both animals and humans more than half a century ago. However, more recent (and higher quality) evidence doesn’t support it.

On any given day, we have between 1,100 and 1,700 milligrams of cholesterol in our body. 25% of that comes from our diet, and 75% is produced inside of our bodies by the liver. Much of the cholesterol that’s found in food can’t be absorbed by our bodies, and most of the cholesterol in our gut was first synthesized in body cells and ended up in the gut via the liver and gall bladder. The body tightly regulates the amount of cholesterol in the blood by controlling internal production; when cholesterol intake in the diet goes down, the body makes more. When cholesterol intake in the diet goes up, the body makes less.

This explains why well-designed cholesterol feeding studies (where they feed volunteers 2-4 eggs a day and measure their cholesterol) show that dietary cholesterol has very little impact on blood cholesterol levels in about 75% of the population. The remaining 25% of
the population are referred to as “hyper-responders”. In this group, dietary cholesterol does modestly increase both LDL (“bad cholesterol” and HDL (“good cholesterol”), but it does not affect the ratio of LDL to HDL or increase the risk of heart disease. (2)

In other words, eating cholesterol isn’t going to give you a heart attack. You can ditch the egg-white omelets and start eating yolks again. That’s a good thing, since all of the 13 essential nutrients eggs contain are found in the yolk. Egg yolks are an especially good source of choline, a B-vitamin that plays important roles in everything from neurotransmitter production to detoxification to maintenance of healthy cells. (3) Studies show that up to 90% of Americans don’t get enough choline, which can lead to fatigue, insomnia, poor kidney function, memory problems and nerve-muscle imbalances. (4)

What about saturated fat? It’s true that some studies show that saturated fat intake raises blood cholesterol levels. But these studies are almost always short-term, lasting only a few weeks. (5) Longer-term studies have not shown an association between saturated fat intake and blood cholesterol levels. In fact, of all of the long-term studies examining this issue, only one of them showed a clear association between saturated fat intake and cholesterol levels, and even that association was weak. (6)

Moreover, studies on low-carbohydrate diets (which tend to be high in saturated fat) suggest that they not only don’t raise blood cholesterol, they have several beneficial impacts on cardiovascular disease risk markers. For example, a meta-analysis of 17 low-carb diet trials covering 1,140 obese patients published in the journal Obesity Reviews found that low-carb diets neither increased nor decreased LDL cholesterol. However, they did find that low-carb diets were associated with significant decreases in body weight as well as improvements in several CV risk factors, including decreases in triglycerides, fasting glucose, blood pressure, body mass index, abdominal circumference, plasma insulin and c-reactive protein, as well as an increase in HDL cholesterol. (7)

If you’re wondering whether saturated fat may contribute to heart disease in some way that isn’t related to cholesterol, a large meta-analysis of prospective studies involving close to 350,000 participants found no association between saturated fat and heart disease. (8) A Japanese prospective study that followed 58,000 men for an average of 14 years found no association between saturated fat intake and heart disease, and an
inverse association between saturated fat and stroke (i.e. those who ate more saturated fat had a lower risk of stroke). (9)

That said, just as not everyone responds to dietary cholesterol in the same manner, there’s some variation in how individuals respond to dietary saturated fat. If we took ten people, fed them a diet high in saturated fat, and measured their cholesterol levels, we’d see a range of responses that averages out to no net increase or decrease. (If dietary saturated fat does increase your total or LDL cholesterol, the more important question is whether that’s a problem. I’ll address that next.)

Another strike against the diet-heart hypothesis is that many of its original proponents haven’t believed it for at least two decades. In a letter to the New England Journal of Medicine in 1991, Ancel Keys, the founder of the diet-heart hypothesis said (10):

>Dietary cholesterol has an important effect on the cholesterol level in the blood of chickens and rabbits, but many controlled experiments have shown that dietary cholesterol has a limited effect in humans. Adding cholesterol to a cholesterol-free diet raises the blood level in humans, but when added to an unrestricted diet, it has a minimal effect.

In a 2004 editorial in the Journal of American College of Cardiology, Sylvan Lee Weinberg, former president of the American College of Cardiology and outspoken proponent of the diet-heart hypothesis, said (11):

>The low-fat, high-carbohydrate diet... may well have played an unintended role in the current epidemics of obesity, lipid abnormalities, type 2 diabetes, and metabolic syndromes. This diet can no longer be defended by appeal to the authority of prestigious medical organizations.

We’ve now established that eating cholesterol and saturated fat does not increase cholesterol levels in the blood for most people. Now, I’ll debunk the myth that high cholesterol in the blood is the cause of heart disease.
High Cholesterol is Not the Cause of Heart Disease

Part of the confusion about cholesterol and its role in heart disease is caused by imprecise terminology. So, before I explain why high cholesterol is not the underlying cause of heart disease, we have to cover some basics.

Cholesterol is not technically a fat; rather, it’s classified as a sterol, which is a combination of a steroid and alcohol. It's crucial to understand that you don't have a cholesterol level in your blood. Cholesterol is fat-soluble, and blood is mostly water. In order for cholesterol to be transported around the body in the blood, it has to be carried by special proteins called lipoproteins. These lipoproteins are classified according to their density; two of the most important in cardiovascular disease are low-density lipoprotein (LDL) and high-density lipoprotein (HDL).

I know this can get confusing quickly, so let me use an analogy to make this more clear. Imagine your bloodstream is like a highway. The lipoproteins are like cars that carry the cholesterol and fats around your body, and the cholesterol and fats are like passengers in the cars. Scientists used to believe that the number of passengers in the car (i.e. concentration of cholesterol in the LDL particle) is the driving factor in the development of heart disease. More recent studies, however, suggest that it's the number of cars on the road (i.e. LDL particles) that matters most.

Coronary arteries are essentially hollow tubes, and the endothelium (lining) of the artery is very thin—only one cell deep. The blood, which carries lipoproteins like LDL, is in constant contact with the endothelial lining. So why does the LDL particle leave the blood, penetrate the endothelium and enter the artery wall? The answer is that it’s a gradient-driven process. Going back to our analogy, the more cars there are on the road at one time, the more likely it is that some of them will “crash” into the fragile lining of the artery. It’s not the number of passengers (cholesterol) the cars are carrying that is the determining factor, but the number of cars on the highway.

The significance of this in terms of determining your risk of heart disease is profound. When you go to the doctor to get your cholesterol tested, chances are he or she will measure your total, LDL and HDL cholesterol. This tells you the concentration of
cholesterol (passengers) inside of the lipoproteins (cars), which is not the driving factor behind plaque formation and heart disease. Instead, what should be measured is the number of LDL particles in your blood.

LDL cholesterol levels and LDL particle number are often concordant (i.e. when one is high, the other is high, and vice versa), and this is probably why there is an association between LDL cholesterol and heart disease in observational studies. The elevated LDL cholesterol was more of a proxy marker for elevated LDL particle number in these cases. But here’s the kicker: they can also be discordant. In layperson’s terms, it’s possible to have normal or even low cholesterol, but a high number of LDL particles. (12) If this person only has their cholesterol measured, and not their particle number, they will be falsely led to believe they’re at low risk for heart disease.

Even worse, the patients that are the most likely to present with this pattern are among the highest risk patients: those with metabolic syndrome or full-fledged type 2 diabetes. The more components of the metabolic syndrome that are present—such as abdominal obesity, hypertension, insulin resistance, high triglycerides and low HDL—the more likely it is that LDL particle number will be elevated. (13)

On the other hand, patients with high LDL cholesterol (LDL-C) and low LDL particle number (LDL-P) are not at high risk of heart disease. In fact, studies suggest they’re at even lower risk than patients with low LDL-C and low LDL-P. (14) Yet they will often be treated with statin drugs or other cholesterol lowering medications, because the clinician only looked at LDL-C and failed to measure LDL particle number. This is a concern for two reasons. First, statin drugs aren’t harmless. Second, studies suggest that low cholesterol can increase the risk of death, especially in women and the elderly.

In one study of over 52,000 Norwegians, researchers found that women with total cholesterol levels below 195 mg/dL had a higher risk of death than women with cholesterol levels above that cut-off. (15) And a study published in the American Journal of Medicine found that people over 70 years of age with total cholesterol levels below 160 mg/dL had twice the risk of death than those with cholesterol levels between 160-199 mg/dL. (16) Low cholesterol is also associated with increased risk of disease—especially mental health and brain disorders. For example:
A study in the Journal of Psychiatric Research found that men with low total cholesterol levels were 7 times more likely to die prematurely from unnatural causes such as suicide and accidents than other men in the study. (17)

A 1993 study published in The Lancet found that depression was 3 times more likely in men over 70 with low cholesterol than in those with normal or high cholesterol. (18)

A Swedish study found that women with the lowest cholesterol suffered significantly more depressive symptoms than other women in the study. (19)

A study in the journal Neurology showed that low cholesterol is associated with increased risk of dementia. (20)

A paper published in the European Journal of Internal Medicine linked low cholesterol levels with Alzheimer’s disease. (21)

It's important to note that all of these studies were observational, which means that they don’t prove that low cholesterol was the cause of the increased risk of death or disease that was observed. It’s possible, for example, that these patients had another disease that caused both the lower cholesterol and increase in disease or mortality. However, given what we know about the important roles of cholesterol in the body, it’s certainly plausible that low cholesterol is capable of contributing to these problems directly.

I’d like to point out that although LDL particle number is superior to LDL cholesterol as a marker for heart disease, it’s still just that—a marker. A marker is not a disease. It’s a risk factor for a disease. Having a risk factor for a disease does not guarantee that you will get that disease—it just increases the chance that you will. There are still several gaps in our knowledge about LDL-P and its usefulness in a clinical setting. For example:

Imagine two people with an LDL-P above 2,000, which puts them in the highest risk group. Person A follows a Paleo diet and lifestyle, gets plenty of sleep, manages stress and has no other significant risk factors for heart disease. Person B eats a Standard American Diet, doesn’t exercise, doesn’t get enough sleep, is stressed out and has several other risk factors for heart disease. Logic would dictate that Person A would be at much lower risk for heart disease than Person B, but there isn’t any comparative data to quantify the difference in risk and it’s unlikely such a study will ever be done. (Who would pay for it?)
Imagine two people following a healthy Paleo-type diet and lifestyle. Person C has no conventional risk factors for heart disease. Person D has no conventional risk factors either, but does have an LDL-P of 2,000. Logic here would dictate that Person D is at higher risk than Person C, but again, we don’t have actual data to quantify the difference in risk.

Heart disease is a complex, multifactorial process. The likelihood that we’ll have a heart attack depends on numerous factors, including genetics, diet, lifestyle and living environment. The purpose of this eBook is not to suggest that LDL-P is the only risk factor that matters, or that other risk factors shouldn’t be taken into consideration. It is simply to point out that existing evidence suggests that LDL-P is a much better predictor of heart disease risk than LDL or total cholesterol, and that it appears to be one of the better markers available to us now.

Conventional medicine is primarily focused on suppressing symptoms. If your blood pressure is high, you take a medication to lower it. If your blood sugar is high, you take a medication to lower it. If your cholesterol is high, you take a medication to lower it. In most cases there is rarely any investigation into why these markers are high in the first place, with the possible exception of some basic (but often incorrect) counseling on diet and exercise.

On the other hand, functional medicine—which is what I practice—focuses on treating the underlying cause of health problems instead of just suppressing symptoms. If your blood sugar, blood pressure or cholesterol are high, the first question a functional medicine practitioner will ask is “why?” If we can identify the root cause of the problem, and address it at that level, medication is often unnecessary.

To use a simple analogy, if you have weeds in your garden, what happens if you just cut the weeds from the top? They grow right back—and sometimes faster than before! If you really want to get rid of them once and for all, you have to pull them up by their roots.

With this in mind, let’s look at some of the potential causes of elevated LDL particle number. If your LDL-P is high, it makes sense to test for and treat any of the conditions below (with the exception of the last, which is genetic and thus can’t be treated) before—or at least along with—taking pharmaceutical drugs.
What Causes Elevated LDL Particle Number?

LDL particles don’t just carry cholesterol; they also carry triglycerides, fat-soluble vitamins and antioxidants. You can think of LDL as a taxi service that delivers important nutrients to the cells and tissues of the body.

As you might expect, there’s a limit to how much “stuff” that each LDL particle can carry. Each LDL particle has a certain number of cholesterol molecules and a certain number of triglycerides. As the number of triglycerides increases, the amount of cholesterol it can carry decreases, and the liver will have to make more LDL particles to carry a given amount of cholesterol around the body. This person will end up with a higher number of LDL particles.

Consider two hypothetical people. Both have an LDL cholesterol level of 130 mg/dL, but one has high triglycerides and the other has low triglycerides. The one with the high triglyceride level will need more LDL particles to transport that same amount of cholesterol around the body than the one with a low triglyceride level.

Numerous studies have found an association between increased LDL particle number, and metabolic syndrome. One study measured ApoB, a marker for LDL particle number, in a group of 1,400 young Finns with no established disease. The participants with the highest LDL particle number were 2.8 times more likely to have metabolic syndrome than those with the lowest levels of LDL-P. (22) A much larger study of over 300,000 men also found a strong association between LDL-P and metabolic syndrome and its components (i.e. insulin resistance, abdominal obesity, high blood pressure, etc.). (23)

Poor thyroid function is another potential cause of elevated particle number. Thyroid hormone has multiple effects on the regulation of lipid production, absorption, and metabolism. It stimulates the expression of HMG-CoA reductase, which is an enzyme in the liver involved in the production of cholesterol. (As a side note, one way that statins work is by inhibiting the HMG-CoA reductase enzyme.)

Thyroid hormone also increases the expression of LDL receptors on the surface of cells in the liver and in other tissues. In hypothyroidism, the number of receptors for LDL on
cells will be decreased. This leads to reduced clearance of LDL from the blood and thus higher LDL levels. Hypothyroidism may also lead to higher cholesterol by acting on Niemann-Pick C1-like 1 protein, which plays a critical role in the intestinal absorption of cholesterol. (24, 25)

Studies show that LDL particle number is higher even in subclinical hypothyroidism (high TSH with normal T4 and T3), and that LDL particle number will decrease after treatment with thyroid hormone. (26)

Another cause of high cholesterol profile is infection. Multiple studies have shown associations between bacterial infections like *Chlamydia pneumoniae* and *H. pylori*, which is the bacterium causes duodenal ulcers, and viral infections like herpes and *cytomegalovirus* and elevated lipids. (27) For example, *H. pylori* leads to elevated levels of total cholesterol, LDL cholesterol, lipoprotein (a), ApoB or LDL particle number, and triglyceride concentrations as well as decreased levels of HDL. (28)

Several mechanisms have been proposed to explain the association between infections and elevated blood lipids. Some evidence suggests that viral and bacterial infections directly alter the lipid metabolism of infected cells, and other evidence suggests that lipids increase as a result of the body’s attempt to fight off infection. Other evidence suggests that LDL has antimicrobial properties and is directly involved in inactivating microbial pathogens. This has been confirmed by studies showing that mice with defective LDL receptors—and thus very high levels of LDL—are protected against infection by gram-negative bacteria like *H. pylori*. (29)

One of the primary functions of the intestinal barrier is to make sure that stuff that belongs in the gut stays in the gut. When this barrier fails, endotoxins such as lipopolysaccharide (LPS) produced by certain species of gut bacteria can enter the bloodstream and provoke an immune response. Part of that immune response involves LDL particles, which as I mentioned above, have an anti-microbial effect. A protein called LPS-binding protein, which circulates with LDL particles, has been shown to reduce the toxic properties of LPS by directly binding to it and removing it from the circulation. (30) Studies have also shown significant increases in LPS-binding protein (and thus LDL particles) in cases of endotoxemia—a condition caused by large amounts of circulating endotoxins. (31)
Though more research is needed in this area, the studies above suggest that a leaky gut could increase the level of LPS and other endotoxins in the blood, and thus increase LDL particle number as a result. I have seen this in my practice. I recently had a patient with high LDL-P and no other risk factors. I tested his gut and discovered *H. pylori* and small intestine bacterial overgrowth (SIBO). After treating his gut, his LDL-P came down to normal levels.

The final cause of elevated LDL-P is genetics. Familial hypercholesterolemia, or FH, involves a mutation of a gene that codes for the LDL receptor or the gene that codes for apolipoprotein B (ApoB). The LDL receptor sits on the outside of cells; the LDL particle has to attach to the LDL receptor in order to deliver the nutrients it’s carrying and be removed from the circulation. ApoB is the part of the LDL particle that binds to the receptor. If we use a door lock as an analogy, apolipoprotein B would be the key, and the LDL receptor is the lock. They both need to be working properly for LDL to deliver its cargo and to be removed from the bloodstream.

Homozygous carriers of FH have two copies of the mutated gene. This condition is very rare. It affects approximately 1 in a million people. And people that are homozygous for this mutation have extremely high total cholesterol levels, often as high as 1000 mg/dL. And unfortunately they usually die from severe atherosclerosis and heart disease before the age of 25.

Heterozygous carriers, however, only have a single copy of the mutated gene, and the other copy is functioning normally. This is much more common. The prevalence is between 1 in 300 to 1 in 500 people, depending on which study you look at. These heterozygous carriers of FH have total cholesterol levels that often range between 350 and 550 mg/dL, along with very high LDL particle number. They have about three times higher risk of death from heart disease than people without FH if it goes untreated.

It’s important to note that people with FH have primarily large, buoyant LDL particles, and yet are still at much higher risk for cardiovascular disease. While it’s true that small, dense, oxidized LDL particles are more likely to cause atherosclerosis, large, buoyant particles can also be harmful when their concentration is high enough. This is one reason why LDL particle number is a superior marker to LDL particle size.
Statins Don’t Save Lives in People Without Heart Disease

Statins have been hailed by many in the conventional medical establishment as wonder drugs, with some physicians going as far as suggesting they should be added to the water supply. (The doctor that made that particular suggestion is named John Reckless – I kid you not.) But are statins really the wonder drugs they’ve been made out to be?

Before we dive into the statistics on statins, I need to briefly explain the difference between relative and absolute risk reduction. Researchers and pharmaceutical companies often use relative risk statistics to report the results of drug studies. For example, they might say “in this trial, statins reduced the risk of a heart attack by 30%”. But what they may not tell you is that the actual risk of having a heart attack went from 0.5% to 0.35%. In other words, before you took the drug you had a 1 in 200 chance of having a heart attack; after taking the drug you have a 1 in 285 chance of having a heart attack. That’s not nearly as impressive as using the 30% relative risk number, but it provides a more accurate picture of what the actual, or “absolute” risk reduction is.

With that in mind, let’s take a closer look at the efficacy of statins in two broad groups of people: those with pre-existing heart disease, and those without pre-existing heart disease. In the medical literature, these groups are referred to as “secondary prevention” and “primary prevention”, respectively.

SECONDARY PREVENTION (THOSE WITH PRE-EXISTING HEART DISEASE)

There’s little doubt that statins are effective in reducing heart attacks and deaths from heart disease in people who already have heart disease. Several large controlled trials including 4S, CARE, LIPID, HPS, TNT, MIRACL, PROV-IT and A to Z have shown relative risk reductions between 7% on the low end in MIRACL and 32% on the high end in 4S, with an average risk reduction of about 20%.

However, absolute risk reductions are much more modest. They range from 0.8% in MIRACL on the low end to 9% in 4S on the high end, with an average of 3%.

An analysis by Dr. David Newman in 2010 which drew on large meta-analyses of statins found that among those with pre-existing heart disease that took statins for 5 years (32):

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■ 96% saw no benefit at all
■ 1.2% (1 in 83) had their lifespan extended (were saved from a fatal heart attack)
■ 2.6% (1 in 39) were helped by preventing a repeat heart attack
■ 0.8% (1 in 125) were helped by preventing a stroke
■ 0.6% (1 in 167) were harmed by developing diabetes
■ 10% (1 in 10) were harmed by muscle damage

A heart attack or stroke can have a significant negative impact on quality of life, so any intervention that can decrease the risk of such an event should be given serious consideration. But even in the population for which statins are most effective—those with pre-existing heart disease—83 people have to be treated to extend one life, and 39 people have to be treated to prevent a repeat heart attack.

Moreover, these results do not apply to all populations across the board. Most studies have shown that while statins do reduce cardiovascular disease (CVD) events and deaths from CVD in women, they do not reduce the risk of death from all causes (“total mortality”). (33)

Nor do these results apply to men or women over the age of 80. Statins do reduce the risk of heart attack and other CVD events in men over the age of 80, and especially at this age, these events can have a significant negative impact on quality of life. However, the bulk of the evidence suggests that statins don’t extend life in people over 80 years of age, regardless of whether they have heart disease, and the highest death rates in people over 80 are associated with the lowest cholesterol levels. (34), (35)

**PRIMARY PREVENTION (THOSE WITHOUT PRE-EXISTING HEART DISEASE)**
Statins do reduce the risk of cardiovascular events in people without pre-existing heart disease. However, this effect is more modest than most people assume. Dr. Newman also analyzed the effect of statins given to people with no known heart disease for 5 years (36):

■ 98% saw no benefit at all
■ 1.6% (1 in 60) were helped by preventing a heart attack
- 0.4% (1 in 268) were helped by preventing a stroke
- 1.5% (1 in 67) were harmed by developing diabetes
- 10% (1 in 10) were harmed by muscle damage

These statistics present a more sobering view on the efficacy of statins in people without pre-existing heart disease. They suggest that you’d need to treat 60 people for 5 years to prevent a single heart attack, or 268 people for 5 years to prevent a single stroke. These somewhat unimpressive benefits must also be weighed against the downsides of therapy, such as side effects and cost. During that hypothetical 5 year period, 1 in 67 patients would have developed diabetes and 1 in 10 patients would have developed muscle damage (which can be permanent in some cases, as we’ll see later in this section).

In addition, while statins do moderately reduce cardiovascular events such as heart attack in people without heart disease, they’ve never been shown to extend lifespan in this population. This is true even when the risk of heart disease is high. In a large meta-analysis of 11 randomized controlled trials by Kausik Ray, MD and colleagues published in the Archives of Internal Medicine, statins were not associated with a significant reduction in the risk of death from all causes. (37)

This trial included 65,000 people without pre-existing heart disease but with intermediate to high risk of heart disease. It was important because it was the first review that only included participants without known heart disease. Previous studies suggesting that statins are effective in reducing death in people without pre-existing heart disease included some people that did have heart disease, which would have skewed the results.

The lack of significant effect on mortality is even more interesting in light of the fact that LDL cholesterol levels did decrease significantly in the statin group; the average LDL level in those taking placebo was 134 mg/dL and the average in the statin-treated patients was 94 mg/dL—roughly 30% lower. Yet in spite of this marked reduction in LDL cholesterol in the statin group, there was no difference in lifespan between the two groups. This is yet another line of evidence suggesting that the amount of cholesterol in LDL particles is not the driving factor in heart disease.
A meta-analysis of statin trials in people without heart disease by the prestigious Cochrane Collaboration came to a similar conclusion. (38) They also observed that all but one of the clinical trials providing evidence on this issue were sponsored by the pharmaceutical industry. This is significant because research clearly indicates that industry-sponsored trials are more likely than non-industry-sponsored trials to report favorable results for drugs because of biased reporting, biased interpretation, or both. (39)

ADVERSE EFFECTS OF STATINS

If statins were harmless and free, then it wouldn’t matter how many people need to be treated to prevent a heart attack or extend someone’s lifespan. But statins are not free, nor are they harmless. Statin use has been associated with a wide range of side effects, including myopathy (muscle pain), liver damage, cataracts, kidney failure, cognitive impairment, impotence and diabetes.

Unfortunately, studies show that physicians are more likely to deny than affirm the possibility of statin side effects, even for symptoms with strong evidence in the scientific literature. (40) Assuming that physicians would likely not report the adverse reaction in these circumstances, it’s probable that the incidence of statin side effects is much higher than the reported rates.

One of the most troubling side effects of statins that has only recently become apparent is their potential to increase the risk of diabetes, especially in women. A study by Dr. Naveed Sattar and colleagues published in The Lancet in 2010 examined 13 randomized clinical trials involving over 90,000 patients taking statins. They found that statin use was associated with a 9% increased risk in developing diabetes. Note that this is a relative risk, so the absolute risk of developing diabetes while taking a statin is very low. That said, observational data from the Women’s Health Initiative found a 48% increased risk of diabetes in healthy women taking statins after adjusting for other risk factors. (41)

To summarize:

■ The only population that statins extend life in are men under 80 years of age with pre-existing heart disease.
In men under 80 without pre-existing heart disease, men over 80 with or without heart disease, and women of any age with or without heart disease, statins have not been shown to extend lifespan.

Statins do reduce the risk of cardiovascular events in all populations. A heart attack or stroke can have a significant, negative impact on quality of life—particularly in the elderly—so this benefit should not be discounted.

However, the reductions in cardiovascular events are often more modest than most assume; 60 people with high cholesterol but no heart disease would need to be treated for 5 years to prevent a single heart attack, and 268 people would need to be treated for 5 years to prevent a single stroke.

Statins have been shown to cause a number of side effects, such as muscle pain and cognitive problems, and they are probably more common than currently estimated due to under-reporting.

My intention here is not to suggest that statins have no place in the treatment of heart disease, but rather to give you the objective information you need to decide (along with your doctor) whether they are appropriate for you. The decision whether to take them should be based on whether you have pre-existing heart disease, what your overall risk of a heart attack is, how healthy your diet and lifestyle is, what other treatments you’ve already tried, and your own risk tolerance and worldview.

It’s clear that statins reduce heart disease as well as the risk of death in those that have already had a heart attack, so if you’re in this group and you’ve already tried diet and lifestyle interventions without much impact on your lipid or inflammatory markers, you are more likely to benefit.

How to Prevent and Reverse Heart Disease Naturally

Ben Franklin said, “An ounce of prevention is worth a pound of cure.” Heart disease is no exception. According to the INTERHEART study, which examined cardiovascular risk factors in 51 countries, 9 out of the 10 strongest risk factors for heart disease are modifiable by changes in diet and lifestyle. (42)
While taking action now does not guarantee that you'll never get heart disease (as age is perhaps the strongest risk factor), it does vastly improve your chances of avoiding it or at least delaying it significantly.

When most people hear the phrase “heart-healthy diet”, they think of egg-white omelettes, a salad with no dressing or similar low-fat, low-cholesterol fare. But after reading this eBook, you know better. The dietary approach I’ve written about in my book, *Your Personal Paleo Code*, is an excellent starting place. The diet I outline in this book includes all of the necessary micronutrients in their most bioavailable form, emphasizes an optimal balance of fats, eliminates highly processed and refined foods, and reduces other food toxins that interfere with nutrient absorption. On the other hand, the American Heart Association’s “heart healthy” diet emphasizes nutrient-poor foods such as whole grains and vegetable oil, and unnecessarily restricts nutrient-dense foods like red meat, animal fat and cholesterol.

But which version of the Paleo diet is best for preventing heart disease? In this series we’ve been focusing on LDL particle number as one of the primary drivers of atherosclerosis. We also discussed the five main causes of elevated LDL-P, including insulin/leptin resistance, genetics, poor thyroid function, infections and leaky gut. If you have elevated LDL-P while on a Paleo diet, the key is to first discover what’s causing it and then tailor your diet accordingly. In this eBook, I’ll focus on insulin/leptin resistance and genetics, since those are the two most common causes of elevated LDL-P that I see in my practice.

In the case of insulin/leptin resistance, the best approach is often a low-carb Paleo diet. When I say low carb, I generally mean between 10–15 percent of total calories per day (roughly 65–100 grams for a moderately active male eating 2,600 calories, and 50–75 grams daily for a moderately active female eating 2,000 calories) in the form of fruit and starchy vegetables like sweet potatoes, potatoes, plantain, yuca and taro. I do not count non-starchy vegetables toward the carbohydrate intake, because I don’t believe they make a significant enough contribution to matter. The purpose of this approach is to improve insulin and leptin sensitivity and promote weight loss, which will in turn decrease LDL-P.
If you have high LDL-P, but normal triglycerides, HDL, small LDL-P and your lipoprotein insulin resistance (LP-IR) score on the NMR LipoProfile is normal, and you’ve ruled out thyroid problems, infections and leaky gut, than it’s very likely that you have one of the many genetic variants that can lead to increased LDL particle number. In this case, a low-carb Paleo diet will often increase—rather than decrease—LDL-P. In my practice I will often recommend what I call a “Mediterranean Paleo diet” in these cases. This means following the basic Paleo approach, but reducing intake of fat and increasing intake of fruit and starchy vegetables. You can still eat fat as it naturally occurs in food, but try not adding as much additional fat to meals, and using more monounsaturated fat than saturated fat. In many cases this will decrease LDL-P quite significantly.

The trickiest situation is when someone has both insulin and leptin resistance and a genetic issue. A low-carb diet will usually drive up LDL-P in that situation, but it will improve many other markers that are also risk factors for heart disease, including triglycerides, HDL, fasting insulin, fasting glucose, etc. So I will usually recommend a low-carb diet for these patients, and if their LDL-P goes up, try to use natural therapies to bring it down.

**LIVE A HEART-HEALTHY LIFESTYLE**

Exercise has been shown to reduce LDL particle concentration even independently of diet. (43) Regular exercise prevents the development and progression of atherosclerosis, improves lipids, and reduces vascular symptoms in patients that already have heart disease. The benefits of exercise are related to maintenance of body weight or weight loss, blood pressure control, return of insulin sensitivity, and beneficial changes in lipids, all of which in turn promote endothelial stabilization and vascular health.

In addition to distinct periods of exercise, it’s also important to sit less and stand and walk more. In fact, some research suggests that this “non-exercise” physical activity may have a greater impact on our cardiovascular health than exercise. Dan’s Plan has some fantastic recommendations for physical activity, as well as a great software and hardware-based tracking system.

Chronic sleep deprivation is one of the most pernicious—yet under-recognized—contributors to the modern disease epidemic. Sleep deprivation has been associated with weight gain, insulin resistance, increased appetite and caloric intake,
overconsumption of highly palatable and rewarding food, decreased energy expenditure and a reduced likelihood of sticking with healthy lifestyle behaviors.

Sleep duration and quality are inversely associated with blood pressure in epidemiological studies, and high blood pressure is one of the strongest independent risk factors for cardiovascular disease (CVD). \(44\) Finally, the Nurses Health Study found that those who reported fewer than 5 hours of sleep at night had a 38% greater risk of coronary heart disease (CHD) than those reporting 8 hours of sleep. \(45\)

Stress increases the risk of cardiovascular disease in numerous ways. It increases intestinal permeability, impairs blood sugar control, depresses immunity (which increases the risk of infection), contributes to fat storage in the liver, and promotes consumption of comfort and junk foods. But perhaps the most significant contribution stress makes to CVD is that it promotes inflammation. Stress has been shown to increase circulating inflammatory markers like C-reactive protein (CRP) and interleukin-6 (IL-6), both of which are associated with heart disease \(46\). On the other hand, stress management can have a profound impact on heart disease risk. One recent randomized trial showed that regular meditation decreased the risk of death from heart attack, stroke and all causes by 48%—a much greater reduction than what is observed with statins even in the highest risk population. \(47\)

**BOOST YOUR HEART-HEALTHY NUTRIENTS**

In addition to the basic heart-healthy versions of the Paleo template I mentioned above, there are several specific foods/nutrients that have been shown to improve cardiovascular health.

Cold-water, fatty fish are an excellent source of EPA and DHA, long-chain omega-3 fats with several cardiovascular benefits. An analysis of randomized trials since 2003 suggests that regular fish consumption or consumption of fish oil would reduce total mortality or deaths from all cause by 17%. \(48\) This is remarkable when you consider the fact that statin drugs only reduce total mortality by 15%, and even then, only in certain populations.

Monounsaturated fats have been shown to reduce LDL and triglycerides and increase HDL. They also decrease oxidized LDL, reduce oxidation and inflammation in general,
lower blood pressure, decrease thrombosis, and they may reduce the incidence of heart disease. (49) The best sources of monounsaturated fat are olives, olive oil, macadamia nuts, and avocados.

Antioxidant-rich foods protect against heart disease in a number of important ways. Our antioxidant defense system is what protects us from oxidative damage, which as you now know is a major risk factor for heart disease. Strengthening this system has two sides: reducing our exposure to oxidative stress and increasing our intake of antioxidant-rich foods.

When most people think of antioxidants, they think of fruits and vegetables like dark, leafy greens and fruits like berries. But while it’s true that these foods are rich in antioxidants, what a lot of people don’t know is that red meat and organ meats are also very rich in important antioxidants that aren’t found in significant amounts in plant foods, like CoQ10 and retinol, which is preformed vitamin A. To increase your antioxidant consumption, a good rule of thumb is to eat the rainbow, choosing a variety of colors of fruits and vegetables, as well as organ meats, meats, eggs, and grass-fed dairy.

Polyphenols are a diverse class of molecules made by plants, certain fungi, and a few animals. They serve a lot of purposes including defense against predators and infections, defense against sunlight damage, chemical oxidation, and coloration. The color, in fact, of many fruits and vegetables like blueberries, eggplants, red potatoes, and apples comes from polyphenols. Some of the best studied polyphenol-rich foods are tea, especially green tea; blueberries; extra-virgin olive oil; red wine; citrus fruits; hibiscus tea; dark chocolate; coffee; turmeric; and other herbs and spices. Polyphenol-rich foods have been shown to have a number of beneficial health effects.

For example, dark chocolate has been shown to lower blood pressure and LDL cholesterol and improve insulin sensitivity, red wine has been shown to prevent the increase in oxidized fats that occur after consuming a meal high in oxidized and potentially oxidizable fats, several studies have shown that hibiscus tea lowers blood pressure in people with hypertension, and blueberries have been shown to lower blood pressure and oxidized LDL in men and women with metabolic syndrome. (50)
Some studies have shown that nut consumption may reduce the risk of cardiovascular disease. In a recent analysis of NHANES data from 1999 to 2004, investigators found that nut consumption was associated with a decrease in a wide range of cardiovascular disease risk markers, including body mass index, waist circumference, and systolic blood pressure, compared to non-consumers of nuts. (51) This is observational data so we can’t be sure that it was the nuts, rather than some other factor that wasn’t adequately controlled for, that led to the improvements.

That said, a review of five large prospective studies (including NHANES) as well as clinical trials examining the effects of nut consumption on lipid parameters found similar results. (52) I favor macadamia nuts, almonds and hazelnuts because they are lower in omega-6 linoleic acid, which research suggests may contribute to CVD when consumed in excess.

In the NHANES study, subjects followed for more than 19 years with the highest quartile of dietary soluble fiber intake had a 15% lower risk of heart disease and had a 10% lower risk of cardiovascular events. (53) Soluble fiber binds bile acids or cholesterol; upregulates LDL receptors in the liver; increases clearance of LDL; inhibits fatty acid synthesis by producing short-chain fatty acids like acetate, butyrate, and propionate; improves insulin sensitivity; and increases satiety with lower overall energy intake. (54) Soluble fiber is found in vegetables like brussels sprouts, turnips, carrots, sweet and white potatoes, squash and asparagus, and fruits like apricots, prunes, pears, oranges, grapefruit and mangoes.

**Summary**

I hope you’ve enjoyed this eBook, and that the information I’ve presented will help protect you and those you love against heart disease. I’ve done my best to cover the most important steps you can take, both in terms of diagnosis and treatment. That said, cardiovascular disease is a complex, multifactorial process and it’s difficult to give it the attention it deserves in an eBook.

That’s why I created the [High Cholesterol Action Plan](chriskresser.com). It’s a 9-week, digital course that goes into much more depth on these topics than I was able to go into here, including...
additional tests that help determine your risk, natural alternatives to statins, and a step-by-step framework that helps you determine your own, customized “action plan”.

[Click here](#) to learn more about it and sign up.