Why Your Doctor is Wrong About Cholesterol

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STORY AT-A-GLANCE -

- Cholesterol is a waxy substance found in nearly every cell of your body and is essential to good health. It plays a role in hormone production, digestion, the manufacture of vitamin D following sun exposure, and helps protect your cell membranes
- The most concerning risk factors for cardiovascular disease are insulin resistance, Type 2 diabetes and the chronic inflammation associated with these conditions

- Other factors at play as well, such as how you eat — whether you're rushing or taking your time — and other stress-related factors, both physical and psychological
- Damage of the interior layers of your arteries precedes heart disease, and this damage can be induced by a number of factors, including smoking, high blood pressure, elevated blood sugar and inflammation
- Once the artery is damaged, cholesterol-rich plaque begins to build up as a protective mechanism. Problems arise when the rate of damage and resultant blood clot formation outpace or outstrip your body's ability to repair

Cholesterol is a waxy substance found in nearly every cell of your body and is essential to good health. It plays a role in hormone production, digestion and the manufacture of vitamin D following sun exposure, and helps protect your cell membranes.

As noted by Zoe Harcombe, Ph.D.,[1] "It is virtually impossible to explain how vital cholesterol is to the human body. If you had no cholesterol in your body you would be dead."

Your liver manufactures most, about 80 percent, of the cholesterol your body requires, which in and of itself suggests your body cannot survive without it. The remaining 20 percent comes from your diet. However, dietary cholesterol is absorbed at a rate of 20 to 60 percent, depending on the individual,[2] and if you consume less, your body will compensate by making more and vice versa.

Animals use cholesterol in much the same way, hence beef, pork and chicken have similar levels of cholesterol, averaging 25 milligrams of cholesterol per ounce.[3]

Cholesterol has long been vilified as a primary cause of cardiovascular disease (CVD), yet numerous studies refute this hypothesis, demonstrating that cholesterol has virtually nothing to do with **heart disease** — at least not in the way conventional medicine presents it.

As noted by Harcombe, the notion that there is good and bad cholesterol is also wrong. Low-density lipoprotein (LDL) and high-density lipoprotein (HDL) are not actually cholesterol; they're carriers and transporters of cholesterol, triglycerides (fat), phospholipids and proteins.

"LDL would more accurately be called the carrier of fresh cholesterol and HDL would more accurately be called the carrier of recycled cholesterol," she says.[4] What's more, dietary cholesterol has no impact on the cholesterol level in your blood, so how could dietary cholesterol pose a health risk?

Does High Cholesterol Cause Cardiovascular Disease?

In an April 16, 2018, podcast (embedded at the top of this article), Christopher Kelly with Nourish Balance Thrive interviewed Dr. Malcolm Kendrick, a British physician and author of "Doctoring Data: **How to Sort Out Medical Advice from Medical Nonsense**," "The Great Cholesterol Con," and "A Statin Nation: Damaging Millions in a Brave New Post-Health World."

An outline[5] and transcript[6] of the interview can be found on the Nourish Balance Thrive website. In this interview, Kendrick discusses the true cause of cardiovascular disease and "the specific environmental and psychosocial factors that cause the most harm." You can also view the video above, in which Kendrick delivers a half-hour lecture on "Why Cholesterol Does Not and Cannot Cause Heart Disease."

In short, the most concerning risk factors for cardiovascular disease are actually insulin resistance, Type 2 diabetes and the chronic inflammation associated with these conditions. However, Kendrick argues there are other factors at play as well, such as how you eat.

Americans are notorious for rushing through their meals, while some other countries, such as France, encourage a slower, more leisurely pace during meals, which may affect how the food is metabolized.

> "*I still think that probably is partly the case because clearly if you eat in a rushed fashion and you gobble it down and then you rush around immediately afterwards, your body is not in the correct metabolic rate to digest your food,*" Kendrick says,[7] adding:
>
> "*I was looking at some studies*[8] *from Japan where they got people to eat a meal whilst being read a boring lecture and asked to eat the same meal whilst a comedian was telling jokes. And when the comedian was telling jokes, the blood sugar level was considerably lower.*"

Stress Increases Your Cardiovascular Disease Risk

Kendrick also notes the links between stress and elevations in insulin levels (as insulin is counteracted by stress hormones) and blood clotting factors, and cites data showing that death rates from cardiovascular disease parallel stressful events that affect an entire nation.

For example, in 1989, the heart disease rate skyrocketed in Lithuania, the same year the Soviet Union collapsed. This pattern can also be seen in other European countries. Meanwhile, the LDL hypothesis has failed to hold in a number of studies.

As just one example, Kendrick cites a BMJ study where they looked at LDL levels in people over 60, finding that those with the highest LDL levels actually had the lowest overall mortality, including CVD mortality.[9]

For Cardiovascular Disease to Occur, There Must be Arterial Wall Damage

Kendrick has written an extensive series of articles on the cardiovascular disease process. A summary of this voluminous work can be found in "What Causes Heart Disease — Part 59,"[10] posted on his blog November 27, 2018. In it, he dissects the fallacies inherent in the conventional LDL/cholesterol hypothesis, and explains the thrombogenic theory of cardiovascular disease as a more reliable counterhypothesis. He writes in part:[11]

> "*For the LDL hypothesis to be correct, it requires that LDL can travel past the lining of the artery, the endothelial cells, and into the artery wall behind. This is considered the starting point for atherosclerotic plaques to form.*
>
> *The problem with this hypothesis is that LDL cannot get into any cell, let alone an endothelial cell, unless that cell wants it to. We know this, for certain, because the only way for LDL to enter any cell, is if the cell manufactures an LDL receptor — which locks onto, and then pulls the LDL molecule inside. There is no other passageway. This is an inarguable fact ...*
>
> *Others have argued that ... the LDL simply slips through the gaps between endothelial cells and that is how it gets into the artery wall. Again, this is impossible. There are no gaps between endothelial cells.*

Endothelial cells are tightly bound to each other by strong protein bridges, known as 'tight junctions.'

These tight junctions can prevent the passage of single ions — charged atoms — which makes it impossible for an LDL molecule to slip through, as it is many thousands of times bigger than an ion. This, too, is an inarguable fact.

Ergo, the initiation of an atherosclerotic plaque (the underlying problem in cardiovascular disease) cannot be triggered by LDL leaking into an undamaged artery wall. Which means that, if you want to get LDL (or anything else) into the artery wall, you first must damage the endothelium/lining of the artery."

The American National Heart, Lung, and Blood Institute admits that damage of the interior layers of your arteries precedes heart disease, and that this damage can be induced by a number of factors, including smoking, high blood pressure, elevated blood sugar, inflammation and, as Dr. Zach Bush and Stefanie Seneff, Ph.D., promote: glyphosate. Once the artery is damaged, cholesterol-rich plaque begins to build up as a protective mechanism.

"*However, it is what happens next, where we rapidly diverge in our thinking,*" Kendrick writes.[12] "*The mainstream believes that, after damage has occurred, it is LDL, and only LDL, leaking into the artery wall that triggers a whole series of downstream reactions that lead to plaques forming.*

However, once you have damaged the endothelium there is no longer a barrier to stop anything getting into the artery wall. So, why pick on LDL? You also have proteins, red blood cells, platelets and Lp(a) and VLDL. Indeed, anything in the bloodstream now has free entry."

The Thrombogenic Theory

Kendrick presents the thrombogenic theory — initially suggested by Karl von Rokitansky in 1852[13] — as a counter-theory to the flawed LDL/cholesterol hypothesis. In summary, the thrombogenic theory goes as follows:

1. Endothelial damage occurs, resulting in the formation of a blood clot
2. The resulting blood clot is minimized in size by plasmin, an enzyme that breaks up blood clots
3. A new endothelial layer grows over top of the remnants of the blood clot, thereby incorporating it into the artery wall
4. Macrophages (white blood cells specializing in repair) break down and digest any remnants of the blood clot. The macrophages are in turn broken down along with their contents, and eliminated from your body through your lymph glands.

While this process leads to inflammation, it is a healthy response and not a disease state per se. Problems only arise "when the rate of damage, and resultant blood clot formation, outstrips the ability of the repair systems to clear up the mess," Kendrick explains.

So, while endothelial damage and resulting blood clotting is at the heart of cardiovascular disease, the only time this natural repair process will cause problems is when damage outstrips repair.

Factors That Raise Your Cardiovascular Disease Risk

As noted by Kendrick, "For good health, you want to maintain a balance between the blood being too ready to clot, and the blood not clotting when you need it to."[14] So, what factors might lead to a situation in which the arterial damage is

greater than your body's ability to repair it? Kendrick's "short list" includes over 30 factors, and there are many others. On this list are:

- Use of certain drugs, including oral steroids, omeprazole, Avastin and thalidomide
- Diseases such as Cushing's disease, Kawasaki's disease, **rheumatoid arthritis**, **systemic lupus erythematosus**, chronic kidney disease and acute renal failure, sickle cell disease, **malaria** and Type 2 diabetes, as well as bacterial and viral infections
- Acute physical and mental stress, and chronic mental stress
- Heavy metal exposure, including lead and mercury
- Certain nutritional deficiencies, including vitamins B and C deficiencies

Next there are factors that promote blood clot formation and/or inhibit the dissolution of blood clots, all of which also raise your CVD risk. Again, there are many factors that can do this, including but not limited to:

- Raised levels of lipoprotein (a), blood sugar, very low density lipoprotein (VLDL) and fibrinogen (fibrinogen binds tightly to the blood clot, creating a tough plug; with excess fibrinogen in your blood, you may end up with larger, more tough to dissolve blood clots)
- Dehydration
- Stress hormones such as cortisol
- Nonsteroidal anti-inflammatory drugs
- Acute mental and/or physical stress

Lastly, there are factors that impair your body's repair system, i.e., the formation of a new endothelial layer over the blood clot, and the clearing of debris from the

blood clot, and these also raise your risk for CVD. Factors that prevent new endothelial cells from being formed include but are not limited to:

- Certain drugs such as Avastin, thalidomide, omeprazole and any drug that lowers synthesis of nitric oxide (conversely, anything that **increases nitric oxide** in our body will reduce your risk of heart disease[15])
- Old age
- Chronic kidney disease
- **Type 2 diabetes**
- **Inactivity** (lack of exercise)

Factors that impair clearance of debris from within the arterial wall include:

- Use of oral steroids, immunosuppressant drugs, certain anti-inflammatory drugs and many anticancer drugs
- Age
- Chronic negative psychological stress

The Role of Inflammation in Cardiovascular Disease

According to Kendrick, while chronic inflammation is recognized as a contributing factor for cardiovascular disease, not all inflammation is bad. In fact, the use of anti-inflammatory drugs (which can impair useful acute inflammation) has been linked to an increase in cardiovascular disease risk, "Which suggests that if you interfere with the healing response to arterial injury, you are going to make thing worse — not better," he says, adding:[16]

> *"[T]he real reason why [chronic] inflammation is being seen as a possible cause of CVD is because inflammatory markers can be raised*

in CVD. To my mind this just demonstrates that in people with CVD, lots of damage is occurring, therefore there is more repair going on, so the inflammatory markers are raised.

However, the mainstream has decided to look at this from the opposite side. They see a lot inflammation going on and have decreed that the inflammation is causing the CVD — rather than the other way around. Frankly, I think this is bonkers. But there you go.

Anyway, where has all this got us to? I shall try to achieve a quick summary. The LDL hypothesis is nonsense, it is wrong, and it does not remotely fit with any other factors known to cause CVD.

The thrombogenic theory, on the other hand, fits with almost everything known about CVD. It states that there are three, interrelated, processes that increase the risk of CVD:

- *Increased rate of damage to the endothelial layer*
- *Formation of a bigger or more difficult to remove blood clot at that point*
- *Impaired repair/removal of remnant blood clot.*

Any factor that does one of these three things can increase the risk of CVD ... You need to have three or four, maybe more, and then things really get going ... All of which means that — in most cases — CVD has no single, specific, cause. It should, instead, be seen as a process whereby damage exceeds repair, causing plaques to start developing, and grow — with a final, fatal, blood clot causing the terminal event."

More Evidence Cholesterol Is Not the Enemy

The Minnesota Coronary Experiment was a study performed between 1968 and 1973 that examined the relationship between diet and heart health.[17] The researchers used a double-blind randomized trial to evaluate the effect of vegetable oil (high in **omega-6 linoleic acid**) versus **saturated fats** in coronary heart disease and death.

The results were left unpublished until 2016, when they appeared in the BMJ. An analysis of the collected data revealed lowering your cholesterol levels through dietary intervention did not reduce your risk of death from coronary heart disease. According to the researchers:[18]

> "*Available evidence from randomized controlled trials shows that replacement of saturated fat in the diet with linoleic acid effectively lowers serum cholesterol but does not support the hypothesis that this translates to a lower risk of death from coronary heart disease or all causes.*
>
> *Findings from the Minnesota Coronary Experiment add to growing evidence that incomplete publication has contributed to overestimation of the benefits of replacing saturated fat with vegetable oils rich in linoleic acid.*"

The researchers found that for every 30-point drop in total cholesterol, there was a 22 percent increase in the risk of death from cardiac disease. Upon autopsy, the group eating vegetable oil and the group eating saturated fat had the same amount of atherosclerotic plaques in their arteries, but the group eating saturated fat experienced nearly half the number of **heart attacks** as the group eating vegetable oil.

Similarly, a scientific analysis[19] of three large reviews published by statin advocates (which attempted to validate the current belief that statin treatment helps prevent cardiovascular disease) concluded the three studies in question failed to satisfy criteria for causality and drew faulty conclusions. Specifically, the authors found:[20]

- There was no association between total cholesterol and the degree of atherosclerosis severity
- Total cholesterol levels are generally not predictive of the risk of heart disease and may be absent or inverse in many studies
- In many studies LDL was not associated with atherosclerosis and in a large U.S. based study of nearly 140,000 patients who suffered an acute myocardial infarction, LDL levels at the time of admission were lower than normal
- Adults over the age of 60 with higher LDL levels generally live longer
- Few adults who experience familial hypercholesterolemia die prematurely

The researchers concluded that high cholesterol levels cannot be the main cause of heart disease as those with low levels have nearly the same degree of sclerosis as those with high levels, and the risk of having a heart attack is the same or higher when cholesterol levels are low.

They believe the hypothesis has been kept alive by reviewers using misleading statistics and excluding results from unsuccessful trials while ignoring numerous contradictory observations.[21] For a description of other studies debunking the saturated fat myth, often linked closely to increasing cholesterol levels, see my previous article, "**The Cholesterol Myth Has Been Busted — Yet Again**."

Why Statin Drugs Are Ill Advised for Most

While the dietary guidelines for Americans no longer focus on reducing dietary cholesterol to protect your heart, and U.S. cholesterol treatment guidelines have stopped using total cholesterol as a measure of heart disease risk, honing in on elevated LDL cholesterol instead, we're still far off the mark when it comes down to how to best prevent heart disease.

Refined sugar and processed fructose are in fact the primary drivers of heart disease, so that's where the focus needs to be — not on driving down your cholesterol with the aid of a statin drug (and/or avoiding healthy saturated fats in your diet).

The only subgroup that might benefit from a statin are those born with a genetic defect called familial hypercholesterolemia, as this makes them resistant to traditional measures of normalizing cholesterol.

There are many important reasons why you should not take a statin drug unless you have this genetic defect, including but not limited to the following. For more information about each of these, see "**5 Great Reasons Why You Should Not Take Statins**."

1. **They don't work as advertised** — A 2015 report[22] published in the Expert Review of Clinical Pharmacology concluded that statin advocates used a statistical tool called relative risk reduction to amplify statins' trivial beneficial effects. If you look at absolute risk, statin drugs benefit just 1 percent of the population. This means that out of 100 people treated with the drugs, one person will have one less heart attack.

   Another systematic review[23] published that same year concluded that in studies where death was the primary prevention endpoint, statins taken for

up to six years postponed death by 5 to 19 days. In secondary prevention trials, death was postponed by 10 to 27 days.

Median postponement of death for primary and secondary prevention was three and four days respectively. When you consider the many health hazards associated with these drugs, this minuscule benefit hardly warrants statin treatment.

2. **They deplete your body of CoQ10** — Statins block HMG coenzyme A reductase in your liver, which is how they reduce cholesterol. This enzyme also makes CoQ10, which is an essential mitochondrial nutrient that facilitates ATP production.

   As noted in a 2010 study,[24] "Coenzyme Q10 is an important factor in mitochondrial respiration," and "Primary and secondary deficiencies of coenzyme Q10 result in a number of neurologic and myopathic syndromes." Since they impair your mitochondrial function, they could potentially affect any number of health problems, as without well-functioning mitochondria, your risk for chronic disease increases significantly.

3. **They inhibit the synthesis of vitamin K2**, a vitamin that protects your arteries from calcification.

4. **They reduce ketone production**[25] — If you take CoQ10 while on statins you still have not solved the problem, as the same enzyme (HMG coenzyme A reductase) also inhibits your liver's ability to produce ketones, water-soluble fats that are essential to keep your body metabolically flexible. Ketones are also important molecular signaling molecules. So, statins make it virtually impossible to achieve **nutritional ketosis**.

5. **Because of 2, 3 and 4, statins increase your risk for other serious diseases**, including:

Heart failure — Primarily due to statin-induced CoQ10 deficiency[26,27]
Cancer — Research[28] has shown that 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 carcinoma.
Diabetes — Statins have been shown to increase your risk of diabetes via a number of different mechanisms, two of which include increasing your insulin resistance, and raising your blood sugar.
Neurodegenerative diseases
Musculoskeletal disorders and motor nerve damage — Research[29] has shown that statin treatment lasting longer than two years causes "definite damage to peripheral nerves."
Cataracts
Liver problems[30,31]

Crucial and Recommended Nutritional Supplements if Taking a Statin

If you decide to take a statin, make sure you take CoQ10 or ubiquinol (the reduced form) with it. One study evaluated the benefits of CoQ10 and selenium supplementation for patients with statin-associated myopathy.[32]

Compared to those given a placebo, the treatment group experienced significantly less pain, decreased muscle weakness and cramps, and less fatigue. A vitamin K2 supplement is also highly recommended. MK-7 is the form

you'll want to look for in supplements; it's extracted from the **Japanese fermented soy product called natto**.

Newer Cholesterol Drugs Are Not Safer

Also, beware of a newer class of cholesterol absorption inhibitors called **PCSK9 Inhibitors**.[33] PCSK9 is a protein that works with LDL receptors that regulate LDL in the liver and release LDL cholesterol into the blood. The inhibitors block that protein, thus lowering the amount of LDL circulating in your blood; in clinical trials, these drugs lowered LDLs by about 60 percent. However, as discussed earlier, LDL has no direct bearing on your CVD risk.

What's more, while these drugs are being touted as the answer for those who cannot tolerate some of the side effects of the other drugs, such as severe muscle pain, trials have already discovered that PCSK9 inhibitors can produce "neurocognitive effects," with some patients experiencing confusion and attention deficits.[34] There's evidence suggesting these drugs may actually be even more dangerous than statins.

Assessing Your Heart Disease Risk

As you evaluate your risk of cardiovascular disease, there are specific ratios and blood level values that will tell you much more than your total cholesterol number. For example, an **NMR LipoProfile**, which measures the size of your LDL particles, is a far better assessment of your risk of heart disease than total cholesterol or total LDL. The following tests will also give you a better assessment of your potential risk for heart attack or CVD:

- **Cholesterol ratios** — Your HDL/cholesterol ratio and triglyceride/HDL ratio is a strong indicator of your risk. For your HDL/cholesterol ratio divide your

HDL by your total cholesterol and multiply by 100. That percentage should ideally be above 24 percent. For your triglyceride/HDL ratio divide your triglyceride total by your HDL and multiply by 100. The ideal percentage is below 2 percent.

- **Fasting insulin level** — Sugar and carbohydrates increase inflammation. Once eaten, these chemicals trigger a release of insulin, promoting the accumulation of fat and creation of triglycerides, making it more difficult for you to lose weight or maintain your normal weight. Excess fat around your midsection is one of the major contributors to heart disease.[35]

  Your **fasting insulin level** can be determined by a simple, inexpensive blood test. A normal fasting blood insulin level is below 5 microunits per milliliter (mcU/ml), but ideally, you'll want it below 3 mcU/ml. If your insulin level is higher than 3 to 5, the most effective way to optimize it is to reduce net carbs.

- **Fasting blood sugar level** — Studies have demonstrated people with higher fasting blood sugar levels have a higher risk of heart disease.[36] In fact, when your fasting blood sugar is between 100 and 125 mg/dl, your risk of coronary artery disease increases to 300 percent more than those whose level is below 79 mg/dl.[37,38]

- **High sensitivity C-reactive protein (HS-CRP)** — This is one of the best overall measures of inflammation and an excellent screen for your risk of heart disease. Ideally your level should be below 0.7 and the lower the better.

- **Iron level** — Iron creates an environment for oxidative stress, so excess iron may increase your inflammation and increase your risk of heart disease. An ideal **iron level** for adult men and non-menstruating women is between 40 and 60 nanograms per milliliter (ng/ml). You do not want to be below 20 ng/ml or above 80 ng/ml.

The simplest and most efficient way to lower your iron level if elevated is to donate blood. If you can't donate, then therapeutic phlebotomy will effectively eliminate the excess iron. Heavy metal detoxification will also naturally reduce high iron.

How to Avoid Heart Disease

In closing, remember that high total cholesterol and even high LDL are insignificant when trying to determine your heart disease risk, and dietary cholesterol and saturated fat are not contributing factors.

Probably the best predictor for CVD is your insulin sensitivity. Considering how insulin resistance drives chronic disease in general, not just heart disease, I strongly recommend measuring your fasting insulin on a regular basis and taking immediate action if you find yourself inching toward insulin resistance.

As for preventing or reversing insulin resistance, the following general guidelines will set you on the right track:

1. Dramatically reduce your net carbs and eliminate processed fructose, as this is what set this cascade of metabolic dysfunction into motion in the first place. Replace the lost calories with higher amounts of healthy fats, not protein. My **optimized nutritional plan** can guide you through this process.
2. Normalize your omega-3-to-omega-6 ratio. Most get far too little omega-3, found in fatty fish such as **wild Alaskan salmon**, sardines, anchovies, fish oil and krill oil, and too much omega-6, as it is plentiful in processed vegetable oils and hence processed and fried foods.
3. **Optimize your vitamin D level** by getting regular, sensible sun exposure. Other nutrients of importance include magnesium and vitamins K2 and C.

4. Get eight hours of high quality sleep each night to normalize your hormonal system. Research has shown sleep deprivation can have a significant bearing on your insulin sensitivity.
5. Get regular exercise, as it is a powerful way to help normalize your insulin sensitivity.