

A Special Interview with Chris Kresser

By Dr. Joseph Mercola

DM: Dr. Joseph Mercola

CK: Chris Kresser

Introduction:

DM: Welcome, everyone. This is Dr. Mercola, and I'm joined today by Chris Kresser, who is an acupuncturist and a licensed integrative medicine clinician. He graduated about three years ago, and he's been active in the field.

One of his passions is examining risk factors for heart disease and looking at a relatively novel way of making that assessment with LDL particles and such. He's actually writing a book about this topic for the Paleo ancestral health community.

So, we're delighted to have you today, Chris. And hopefully, you'll expand our knowledge base on this and help us develop a deeper appreciation of how to really accurately predict a person's risk for one of the most common causes of death, which is heart disease. So, welcome and thank you for joining us.

CK: Thank you, Dr. Mercola, for having me. It's a pleasure to be here.

DM: Well, maybe you can just start by explaining your journey and how you got to this point.

CK: Sure.

DM: Because each [of us] have our own unique journey, and understanding that perspective is helpful in appreciating the information you're going to share.

CK: Yeah, I got interested in this topic several years ago actually when my grandfather died of heart disease. His case was pretty badly mismanaged, and that was clear to all of us who were involved at that time. And so, I ended up wanting to learn a lot more about what causes heart disease, because already at that point, I was a little skeptical of the mainstream perspective.

And then a few years later, when I was in graduate school studying integrative medicine, I did a semester-long research project on the relationship between cholesterol and heart disease in my nutrition class. Since then, I've read about 750 peer-reviewed studies. I've consulted with numerous experts in the field. I've challenged just about everything I thought I knew about the role of cholesterol in heart disease. And over the last several years, I've been sharing that information on my blog, in my podcast, and in educational seminars and program.

So, here's the problem, in my opinion, with the mainstream approach in a nutshell. We've been told basically for the last 50 years that eating saturated fat and cholesterol in the diet raises cholesterol levels in the blood. But more recent research over the past 10 or 15 years suggests

that neither of those statements are true. What's more is that the typical cholesterol tests that we get when we go into our doctor that are supposed to measure our risk for heart disease don't actually do a very good job of predicting our risk.

So, some people with low or normal LDL or total cholesterol can actually be at high risk from heart disease. And other people with high or, you know, normal total or LDL cholesterol can actually be at low risk for heart disease. What this means is that some people are not getting enough treatment, and other people are getting too much. In either case, that can put them on unnecessary risk.

The truth is we've learned a lot about what causes heart disease over the past 10 years. But unfortunately, that knowledge hasn't really trickled down into the mainstream yet. So, your average general care physician, primary care nurse, or even science writer that's writing for the mainstream media is still operating on information from the old paradigm.

And the other issue is that the current dietary guidelines that are offered for how to reduce your risk for heart disease are based on this information that's still 30 to 50 years old. And they're clearly not working. As you mentioned, cardiovascular disease is still the number one killer. One out of every three deaths is due to this cardiovascular disease, and it affects about 65 million people in the U.S. alone.

This is a real shame, because the INTERHEART study, which looked at heart disease risk factors in over 50 countries around the world, found that 90 percent or nine in 10 cases of heart disease is completely preventable just by modifying diet and lifestyle factors. So, we really need a new approach that's based on more current evidence.

But the problem, of course, that we face is that the old paradigm is so entrenched. The idea that cholesterol and saturated fat are bad for us is so deeply engrained in our society that a lot of us don't even question that anymore.

One of the main problems there is the massive conflicts of interest in the medical profession. We have a situation where two-thirds of medical research is sponsored by pharmaceutical companies. Eight out of nine of the doctors who are on the National Cholesterol Education program that write the guidelines for cholesterol receive money from pharmaceutical companies.

DM: Before we go there, can we just... What I'd like you to do is discuss or just sort of briefly summarize, and then we go into the details. We can certainly go on to the topic you were just mentioning. But if we can go on the specific diagnostic strategy that you're recommending as a result of this new knowledge... Just briefly mention that and then discuss it.

CK: Right.

DM: And then we can go into more details in a little bit.

CK: When you go in to get your cholesterol test, the doctors, they're usually going to test your total cholesterol, your LDL cholesterol, and maybe your HDL cholesterol and triglycerides. But we now know those are not accurate predictors for cardiovascular disease risk. Instead, the more accurate predictor is testing your actual LDL particle number.

I know this can be a little confusing at first. So, to use an analogy: if you imagine your bloodstream's like a river, the LDL particles are like the boats that carry the cholesterol and fats around the body. The cholesterol and fats are like cargo in the boats.

Right now doctors are usually measuring the amount of cargo or cholesterol in the LDL particles. But what we should be measuring is the number of LDL particles or the number of boats in the rivers, so to speak, because that's a much more accurate risk factor for heart disease.

The biggest problem about this, as I mentioned, is it's possible to have normal total or LDL cholesterol, but have a high number of LDL particles. These people are being completely missed by the medical establishment right now. You know, they might leave the doctor's office feeling really good about their lipid profile when, in fact, they're at higher risk for heart disease.

And then on the other hand, you have a group of people who have a high total cholesterol or LDL cholesterol, and they're being prescribed a statin drug when they may actually have normal LDL particle number, which means they do not have additional risk for heart disease. That's really the crux of the problem.

I would like to see – and actually, it's already happening in some groups like the National Lipid Association. They're really starting to shift the focus toward LDL particle number instead of total and LDL cholesterol.

DM: If people watching this were interested in finding out what that number is, what is the name of the test that they would ask their physician to do? Because obviously, this is a test that's not done at home.

CK: Sure. Yeah. There are several ways to test for it actually, but the best way is called the NMR LipoProfile. It's offered by a lab called Liposcience. This is the FDA-approved technology for testing LDL particle number. It's the one that's used in most of the scientific studies. It turns out that all the different lipoproteins have a unique magnetic signature, and this test uses a nuclear magnetic resonance technique to pick up on that signature. It can correctly identify the number of particles in each case.

The great thing is it's not an exotic or weird test that's hard to get. All the major labs offer it, like LabCorp and Quest. Insurance usually covers it. And even if your doctor won't order it for some reason, there are now third-party intermediaries like Direct Labs, or you can order the test yourself online, and go get the blood drawn locally. It's a pretty easy test to get.

DM: Yeah. Many people watching this may not realize that NMR was the first name that was given to MRI scanners.

CK: Right.

DM: And then they changed it just for... It sounds like they just kept the name "NMR" with the lipoproteins and did an MRI of the lipoproteins.

CK: That's essentially what they did.

DM: Yeah. So, more people may have a better understanding. Because NMR sounded too frightening, I guess, so they had a public relations disaster, and then they changed that name. I think in the late or the early '80s, they changed it over.

CK: Right.

DM: Yeah. Actually, the very first paper I published was on NMR for corneal transplants.

CK: Yeah.

DM: An interesting tidbit of history.

CK: Sure.

DM: So, that's a pretty fascinating story. I'm wondering if you can give us your perspective on how we got into this whole sort of deluded, delusional perspective on the danger of fats. When in fact, you know, I think we both are pretty much in agreement that healthy fats are really some of the most foundational important strategies that you can have if you want to live a healthy life. And to avoid them, you are just asking for disaster.

CK: Absolutely. Yeah, I think it was a combination of some early misunderstanding. This research of, example... There were early studies that show that saturated fat raised cholesterol levels in the blood. But the problem with those studies is they're almost always short-term.

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They only lasted for maybe two to 13 weeks or something. Since then, much larger observational studies for a much longer period of time have been done. And there has been no relationship between saturated fat intake and blood cholesterol levels in those studies. We're talking about some really big, well-known studies that cover tens of thousands of people.

In fact, there was research published in the *American Journal of Clinical Nutrition* that covered about 350,000 people in a follow-up period of five to 23 years. And there was no relationship at all between saturated fat intake and heart disease. And then a large Japanese study of about 58,000 people actually found an inverse association between saturated fat intake and strokes. So, in other words, the people who were eating the most saturated fat actually had the lowest levels of stroke.

And then in terms of dietary cholesterol, you had some early experiments....

DM: Wait, before we go into dietary cholesterol, let's go on the saturated fat.

CK: Sure.

DM: Because what you said, I agree with, but certainly, the vast majority – maybe over 95 percent of the media and conventional physicians – do not accept that. In fact, the last time I was in Dr. Oz, we've got a battle about this, because he's a firm believer in the danger of saturated fat.

CK: Yeah.

DM: So, there's a large number of studies that sort of support, conclude, and summarize that there's a danger there. My take on those studies – and I would be interested in yours – is that there was damage that occurred with the saturated fats, but it was because of the compounding variable of the trans-fats. That variable was never separated out, and that was what's responsible for those studies that really provided a support for that recommendation.

CK: Yeah. I think you're right, Dr. Mercola. And I would add another factor, which is just that there are now massive conflicts of interests in the medical profession, which makes it really hard to shift out of an old paradigm for a new paradigm. Because two-thirds of medical research is sponsored by drug companies, and eight out of nine of the doctors who write the National Cholesterol Educational Program guidelines receive money from pharmaceutical companies.

And as Upton Sinclair [inaudible 12:30] said, "It's difficult to get a man to understand something when his salary is dependent upon him not understanding it."

So, if we have a hypothesis that is padding the salaries of researchers and doctors around the world and generates billions of dollars of profit for Big Pharma and Big Food, it's a lot harder. That's not going to turn around overnight, in spite of the fact that, as you pointed out, there's a lot of really, really good solid research suggesting that eating saturated fat does not increase the risk of heart disease.

DM: Yeah. I couldn't agree more with the conflict of interest. The only challenge with that is most of the studies that I'm referring to were sort of pre-1980, which preceded the advent of most of the hypercholesterolemia medications. They had very few back then. And certainly, the statins didn't exist. High-dose niacin was rarely used, and they had some – I forgot some of the ones that were used. It wasn't really acknowledged.

When I first went into practice in the mid-80s, I believed this, and I was confused about the dangers of it, of course. I didn't understand it completely or at least as well as I do now. And I would tell people that they had a cholesterol of 270. They would go back to their old doctor and say, "My doctor said it was okay."

CK: Right.

DM: It was within the normal range. That transition didn't really occur until like the mid to late '80s, where this phobia of cholesterol came about. I mean, I believe it's largely related to the fact that they had drugs that they could sell.

CK: Right.

DM: That could lower them.

CK: Yeah. And I want to be clear. I don't think this is some massive conspiracy or something; I think it was partly a product of just scientific methods improving over time and, like you said, learning to separate trans-fats from saturated fats.

And then another factor, for example, there's a study published just yesterday that went back and looked at some data. You know, some of the early data suggested that replacing saturated fat with industrial seed oil or polyunsaturated fat would lower cholesterol and lower the risk of heart

disease. But what they didn't realize back in the '60s when they were doing those studies: they thought all polyunsaturated fats were the same – omega-6 and omega-3. And so, they would lump them together in the studies.

But now, of course, we understand that omega-6 has very different effects from omega-3. When you study them separately, you see that omega-6 actually doesn't reduce the risk of heart disease and may increase the risk of heart disease when it's studied independently of omega-3s.

I think there were just a lot of misunderstanding early on that has now become clear that didn't necessarily have to do with conflict of interest, but just with, you know, continual advancement of science and our comprehension of this issue.

DM: I couldn't agree more. I saw that study, too; that was in the *British Medical Journal*. I was delighted to see it. We're actually going to do a lead story on that one. But it really isn't new information. We've known that omega-6s in excess are really bad. I mean, we need them. They are essential fats. But it's pervasive in our diets, so almost everyone gets too much.

CK: Yeah. You don't have to try very hard to get enough.

DM: Yeah. And then the danger that's in almost all of the omega-6s now? I mean, this didn't exist 10 or 15 years ago. They're all GMO. I mean, for the most part, canola – well, canola's not omega-6, but – soy and corn oil, they're all [GMO]. So, you've got that added into the mixture, which makes it even worse. Even if they were organic, they'd still be a problem, but then you've got that added.

So, maybe you can share with us your perspective on the top myths of cholesterol and heart disease.

CK: Sure. Yeah, I would say, by far, the top myth is the diet-heart hypothesis, which is the idea that eating cholesterol and saturated fat (which we've already been talking about a little bit) raises cholesterol levels in the blood.

And then the second part of the diet-heart hypothesis is that high cholesterol in the blood is what drives the risk for heart disease. So, we already just talked about saturated fats. What about dietary cholesterol? You know, some of the early animal studies suggested that eating cholesterol like in egg yolks will raise your cholesterol levels in the blood. But we now know that that's actually not true. That's been pretty thoroughly disproven in the scientific literature.

We have between 1,100 to 1,700 milligrams of cholesterol in our body at any given time. But only 25 percent of that actually comes from the diet, and 75 percent is internally produced primarily through the liver. Why would that be? Because cholesterol is so important to the proper function of the body that the body tightly regulates its production. If you don't eat enough cholesterol, the body will make more. It's needing more cholesterol [inaudible 17:34] less.

The other thing that most people don't know is that only free or unesterified cholesterol can be absorbed from the diet through the intestines. Most foods have esterified cholesterol that can't actually be absorbed. This probably explains why egg-feeding studies (where they take volunteers and they feed them between two to four eggs a day)... In 75 percent of cases, there's no change at all in the blood cholesterol levels even in people who were eating up to four eggs.

In 25 percent of cases, they're what are known as hyper-responders. But even in those cases, they experience an increase in both LDL and HDL. Their LDL to HDL ratio doesn't change. That means that there's no clinically significant effect there. They get a mild increase in their cholesterol, but it doesn't affect their risk of heart disease at all.

So, the second part of the diet-heart hypothesis, which is the idea that high-cholesterol levels in the blood are what drive heart disease risk, is a little bit more complicated. We need to understand the basic terminology to really break this down.

The first thing to understand is we don't have a cholesterol level in our blood, actually. Cholesterol is fat-soluble, and the blood is mostly water. For it to be transported around the blood, cholesterol needs to be carried by a protein, specifically by a lipoprotein. These lipoproteins, as many of your viewers know, are classified by density. So, you have very low-density lipoprotein or VLDL, low-density lipoprotein or LDL, and high-density lipoprotein or HDL, which are the main ones that we'll talk about.

I mentioned before the analogy that our bloodstream is like a river. Remember that the lipoproteins are like boats that carry the cholesterol and fats around the body. The cholesterol and fats are like cargo in the boats.

So, here's the really crucial point: up until about 10 or 15 years ago, we thought that it was the concentration of cholesterol in the lipoprotein or the amount of cargo in the boat that was driving the risk of heart disease. But recent research indicates that it's the number of boats or the number of LDL particles that's really the driving factor.

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This is so important to get; I just want to repeat it again. If you're going in to your doctor to get your cholesterol tested, they're not measuring the number of boats or LDL particles; they're measuring the concentration of cargo in those boats.

I guess we could say that the second part of that hypothesis is both true and untrue. It's untrue in the sense that the amount of cholesterol inside of the LDL particle is not the main risk factor for heart disease. But it is true in the sense that if you have too many of those cholesterol-carrying LDL particles, that can increase the risk of heart disease.

DM: Well, let's stick with cholesterol for a bit, because there are two points I want to ask you on that. One is that many believe that it's not the regular cholesterol that's an issue, but the cholesterol that's oxidized or damaged that is essentially the problem with the free radical. So, I'm wondering if you can comment on the oxidation, which typically happens when you heat food. One of the dangers of heating foods excessively, of course.

CK: I definitely agree that oxidized LDL can be a greater risk factor for heart disease. Oxidative damage is a natural process of energy production and storage in the body. But when oxidative stress is high because of a poor diet (too much industrial seed oil, for example), not getting enough exercise, chronic stress, and things like that, or when you're antioxidant capacity is low (again usually because of a poor diet), then we become subject to this oxidative damage.

Oxidized LDL is more harmful than normal [non-]oxidized LDL, because it's smaller and denser. That makes it likely to penetrate the fragile lining of the artery, which is only really one-cell deep. And then oxidized LDL stimulates collagen formation, and collagen is what forms the fibrous plaque, which is associated with heart disease. It also weakens the stability of those plaques, which makes them more likely to rupture. That's the precipitating event in a heart attack.

The more LDL particles you have, the more likely you are to have some oxidized LDL, and they can be more atherogenic.

However, oxidized LDL loses their predictive value when it's adjusted for LDL particle number. That suggests that LDL particle number may be an even more important risk factor and may need a high number of LDL particles before oxidation becomes a big problem.

DM: Terrific. I'm wondering, is there a test that measures oxidized LDL?

CK: Yeah. There are three different tests that measure it in a research setting. But there is only one test that's available from one lab in New York that I'm aware of that measures oxidized LDL. It's not really easily available, unfortunately, in patients yet. And I think part of the reason for that is there's still some controversy among researchers about what the best way to measure oxidized LDL is.

Different studies use different techniques. So, that's actually part of the reason why we might be seeing different results in these studies about the predictive value of oxidized LDL, because they're using different ways to measure it. I think we have a few years to go before we can really [inaudible 23:34-35].

DM: And it's not something you recommend anyway from your investigations. It doesn't seem to be as potent predictor as the LDL particles.

CK: That's right.

DM: Okay. And I'm wondering, too. We now have a deeper appreciation of the value of vitamin K2, especially with atherosclerotic plaque. K2 is briefly responsible for transporting the calcium out of the plaque back into the bones. I'm wondering if that's, from your perspective, the major issue. Or are there any other interaction between the K2 levels and the LDL atherogenicity?

CK: That's a good question. As you mentioned, K2 helps regulate calcium metabolism, keeps it out of the soft tissues, and puts it in the bone, and that would affect plaque formation. I think it does have a relationship there. But I haven't seen any specific research on the relationship between K2 and LDL particle.

I do actually want to go back and add a couple of things to your question about oxidized LDL, because I think there are some ways to indirectly get a sense of oxidative damage and inflammation through some tests that are currently available. One is high-sensitivity C-reactive protein. That's a measure of systemic inflammation in the body. And then there's another marker called Lp-PLA2. This is a more specific way to measure cardiovascular inflammation.

Whereas CRP is a measure of system-wide inflammation in the body, Lp-PLA2 measures specifically cardiovascular inflammation.

In my patients, if I see that their LDL particle number is elevated, I'll also run a CRP test, an Lp-PLA2 test, and then maybe a lipoprotein (a) test, which is another marker of inflammation. If all of those are elevated, then I will suspect that they're under significant oxidative stress, and we might, of course, make dietary recommendations, maybe some supplements that would raise glutathione levels, and something like a water-soluble form of curcumin that can help reduce inflammation.

DM: But you mentioned the CRP test is a high-sensitivity test. That's a relatively new test that's commercially available, because that typically wasn't available in the past.

CK: Yeah.

DM: Okay. Do most labs have that available?

CK: They do now. Yeah.

DM: Okay, good. One last question on the cholesterol. Because some people, certainly a minority, but a significant number of people (and actually I'm one of them) who have a low cholesterol. Previously, that was, "Oh, that's great. You're not going to have heart disease." But we know there are other problems with low cholesterol, because it's so essential for the function of the human body as a precursor for all these important hormones.

So, I'm wondering what strategies you have. One of the ones I've encountered and which seems to be pretty effective is increasing coconut oil. I don't know the mechanism of why it works, but it seems to be pretty darn effective at helping to improve or to increase the cholesterol levels – total cholesterol levels.

CK: Yeah. So, kind of the good news-bad news about the fact that we don't absorb – most of us – much cholesterol from the diet is... The good news is if you're worried about your cholesterol and you've been eating egg-white omelets, you can definitely, in most cases, even go back to eating the egg yolks. And you should, because the egg yolks are what contain most of the nutrients in an egg. But the bad news is if you have low cholesterol, eating a whole bunch of cholesterol may or may not increase your cholesterol levels.

And it's true, coconut oil, which is a medium-chain triglyceride, does seem to help raise cholesterol levels in many people whose cholesterol levels are low.

But another thing to investigate is if someone has low cholesterol levels, one of the first things I will look at is their liver function, because as I mentioned earlier, the liver is where most of the endogenous cholesterol is produced. A lot of factors of the modern lifestyle – or aspects of the modern lifestyle such as our exposures to environmental toxins (which is growing every year), food toxins, alcohol, and things that are quite toxic to the liver – can really impair our liver function.

And so, that's one of the first places to look, and maybe using some botanicals and nutrients that help support healthy liver detoxification and liver function, like milk thistle, for example. I've

had pretty good success with just a natural liver thing program. That will often bring the cholesterol levels back up if they're very low in a person.

DM: That's interesting. In my case, I don't think it's the liver, although it might be secondarily, because it's clearly related to hereditary hemolytic anemia with beta-thalassemia. I don't know why, but it's very clear. And it's even not widely mentioned in the literature, but it's very clear that that's associated with low cholesterol levels.

CK: Right.

DM: I don't know the mechanism. What type of dosage of coconut oil do you use in your patients for it to normalize cholesterol?

CK: Yeah. That kind of depends on what else is going on with them, but you know... As we might talk about later in the interview, one of the main causes of elevated LDL particle number is insulin and leptin resistance. In patients who have that, I will typically put them on a lower-carbohydrate form of a Paleo or ancestral diet. And I'll recommend anywhere between two and six tablespoons of coconut oil a day, depending on how low-carb they are and how low their cholesterol is.

DM: Yeah. That's an interesting recommendation and certainly not commonly advocated, but I certainly agree with that. I'm wondering what strategies you found, because you're a clinician – you're still seeing patients actively, and you've got a lot of good feedback going on. So, have you found any particular strategies to palatably increase that amount? Because obviously, [for] people, just consuming six to eight teaspoons is not... You need to include it in something. So, what have you found to be helpful?

CK: Yeah. I mean, there are a couple of ways. One is making smoothies with coconut milk.

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And oftentimes, patients are able to tolerate it a little bit more that way if it's in a liquid form. Another strategy – and this is mixed in terms of people's response for palatability – is using MCT oil, because the part of coconut oil that is probably having an effect on the cholesterol levels is in the MCT, and that's the part that's ketogenic as well.

MCT oil is 100 percent MCT, whereas coconut oil is about 50 percent. Sometimes adding some MCT, which is a liquid, can be used in salad dressings and things like that, and tastes different from the 100 percent virgin coconut oil, is a better option.

One more is if someone drinks tea or coffee, some people find that putting some in the tea or coffee is pretty palatable as well.

DM: Yeah, because it will be a liquid. Because the problem with coconut oil is it's solid for about 75 degrees. In the winter, that's common to be solid for most people's homes.

So, the other 50 percent of coconut oil that's not MCT, would that be lauric acid? Or is lauric acid an MCT also?

CK: I don't actually know the answer to that question. I think it's an MCT, but I believe there are some long-chain saturated fats in coconut oil as well.

DM: Yeah, because I was surprised. I didn't realize... I've always promoted coconut oil as being beneficial because of the lauric acid. But then I realized one day, it's like over 50 percent lauric acid, it's just not a little. I mean, it's significant.

CK: As you've written, it has antimicrobial benefits. It's present in mother's milk and has some pretty unique properties above and beyond its role as a fat.

DM: Now, I'm also particularly fond of coconut oil because I believe it's a very useful therapeutic agent to help people in their attempt to implement something called intermittent fasting, which I suspect you're a fan of, too. And I think that's probably one of the most valuable stealth strategies to get people healthy, largely I believe because it really helps people transition to radically improve their insulin and leptin resistance.

CK: That's right.

DM: I'm wondering if you can comment on that and your experience, especially with respect to cholesterol levels.

CK: Yeah. As you mentioned, insulin resistance and leptin resistance are pretty widespread problems. And I just mentioned a little while ago that that's one of the main driving forces in elevated LDL particle number. The reason for that is that LDL particles can only carry a certain... They carry not only cholesterol, but also triglycerides, fat-soluble vitamins, and antioxidants. Each LDL particle can only carry so much.

If someone has high triglycerides, which they often will when they have insulin or leptin resistance, then that means a given LDL particle can carry less cholesterol, because it's stuffed full of triglycerides. The liver will then have to make more LDL particles to carry that same given amount of cholesterol around the tissues and cell in the body.

That's why people with insulin and leptin resistance can have normal or low total or LDL cholesterol, because their LDL particles are cholesterol [inaudible 33:30]. But they can have a high LDL particle number because the triglyceride-rich particles make it. So, the liver has to make many more, several more, of them.

Intermittent fasting is one of many ways to improve insulin sensitivity and leptin sensitivity, because there are certain processes in the body that engage after you haven't eaten for a period of time. They're all evolutionary mechanisms that are designed to help us survive in periods of food scarcity. You have an upregulate in metabolism basically, and it becomes easier to, you know... Your insulin sensitivity improves. Your leptin sensitivity improves.

It's a really good way for people to lose weight, which again will improve insulin and leptin sensitivity, because obesity is both a cause and an effect of leptin resistance. I think it's a really great strategy for most people; I do use it in my practice a lot. The only kind of caution might be in people who have pretty severe fatigue, you know, or are suffering from some kind of chronic illness, and need to eat more [inaudible 34:50]. But for most people, I think it's great.

DM: Yeah. I've noticed, too, that the transition is the challenge. [But] once you're in that fat-adapted zone, it's pretty easy. The worse insulin and leptin resistance you have, the longer it's going to take.

CK: That's right.

DM: So, in that transition period, it seems that's when coconut oil is most beneficial, because it will not upset insulin and leptin resistance. It's neutral, but yet it's metabolized so rapidly and provides a source of energy, because the fatigue is the real challenge in the hunger. So, it can be a really useful tool, especially if implemented in the suggestions that you mentioned earlier.

CK: Yeah. I know you had Paul Jaminet on your show a while back. He recommends – and I agree with him – that you can actually have some coconut oil during the fasted period. So, let's say, you're compressing all your food intake between 12:00 P.M. and 8:00 P.M. If you wake up in the morning and have a cup of coffee, you can add some coconut oil to it, because coconut oil will not interrupt the beneficial processes that are happening while you're fasting. It's really more of protein and carbohydrate that will interrupt those processes.

You can have a little bit of coconut oil in the morning, and that might help you make it through until your first meal.

DM: Okay, great. So, let's step back a little bit and help people understand better, so that they could explain it to their friends or relatives why statins aren't the solution. So, why? The shortest answer, of course, is that it doesn't treat the cause. As we mentioned and you certainly mentioned, the cause is insulin and leptin resistance. Why don't we step back and explain what causes the insulin and leptin resistance, and then what's secondary to that that actually raises those LDL particles that increases the risk for heart disease.

CK: Insulin and leptin resistance, I think, has a number of causes. I really try to summarize them with a simple phrase, which is "Insulin and leptin resistance equals modern lifestyle [inaudible 37:02] genetic nutrition." I'm sure we all know someone who eats a terrible diet, doesn't exercise, and is under a lot of stress, and they still remain lean. That doesn't mean they're not insulin- or leptin-resistant, because you can be lean and still have those problems.

But there is some genetic predisposition to this condition, I think, that affects our likelihood of getting it. But what I mean by modern lifestyle, I'm talking about a diet that's full of processed and refined carbohydrates, sugars, refined flours, industrial seed oils (which are a huge problem that I think we're only recently beginning to fully understand), and all of the foods in boxes and bags that really comprise a large percentage of calories for many Americans now. So, there's the toxic food system.

There's a decrease in non-exercise physical activity, which I think is an increasingly big factor. What I mean by that is it's becoming clear that too much sitting has harmful effects that are completely independent of how much exercise we've got. People who are getting plenty of exercise and who are even marathon runners and training – you know, running for miles and miles a week – if they're sedentary most of the rest of the time, that's going to cause biochemical changes that predispose them to insulin and leptin resistance. That's another big problem.

And then we have chronic sleep deprivation. It's a kind of stealth issue that is more and more on the radar. There are studies that show that even one night of disturbed sleep can decrease your insulin sensitivity the next day and cause pretty intense cravings and overeating. I think that's another big problem.

And then we have issues like exposure to environmental toxins. There are a lot of studies in mice now that show that exposure to BPA, which is in some plastics, in the lining of cans, receipts, and things like that, can disrupt the brain regulation of weight.

And then we have the gut and gut health, which is really, again, another one of these factors that's becoming more and more clear in its effect on stimulation. Some studies show that the gut flora, the beneficial bacteria that live in our gut, if there's an imbalance there (we have too much bad bacteria or too much of one kind of bacteria versus another), that can actually predispose us to obesity and insulin and leptin resistance.

It's really a combination of factors, in my opinion, that contribute to this. But they're all very common and really kind of epidemic in our modern lifestyle.

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DM: Well, and the end result of these, the culmination of the synergistic effect of all those or the majority of them going the wrong way, as I understand: it puts this pressure on the liver to actually increase the production of cholesterol.

CK: That's it. Yeah. It puts pressure on the liver to increase the production of lipoproteins, more specifically of low-density lipoproteins. And then there's one other way that leptin resistance contributes. Leptin is kind of the master signal in the body that communicates whether we're in a state of abundance, meaning whether we have all the nutrients and nourishment that we need.

When the cell signals that it needs more cholesterol for whatever function it's performing, or when leptin signals to the body that we're in a state of abundance, the LDL receptor activity will be ramped up. Now, the LDL receptor sits on the outside of cells, and its job is to dock or attach to the LDL particles that are floating around in the blood, so that the LDL particles can deliver the nutrients that they're carrying inside of the cell. That's the way that the normal mechanism works.

The problem is if someone is leptin-resistant, they won't get that message, because the receptors for leptin are not sensitive enough, and then the LDL receptor activity won't be increased. And then you'll have these LDL particles that are just floating around in the blood in contact to a cell and be taken out of the circulations. That will increase the LDL particle number directly as well.

So, there are a couple of different mechanisms by which insulin resistance and leptin resistance increase LDL particle number.

DM: Thank you for clarifying that. I also want to comment on one that we may or may not be aware of, and that's the issue of the sleep. But there's a really great objective tool that's relatively inexpensive, it's called the Zeo. Have you heard of the Zeo?

CK: Yes.

DM: Do you use it in your practice?

CK: I've used the Zeo. That's another one that renews, the sleep clock or sleep monitor that I like. It's a pretty exciting field. It's a lovely tool.

DM: Yeah. I've been using it every day for about the last two years. It's a sensor that you attach to your head. Actually, I stopped for a while, because the initial sensors were actually transmitting to a bedside clock, which I didn't want to do because of the EMF issue. Now it just records and you download the data later.

But it tells you how much deep sleep you're having, how much REM sleep, how long you're sleeping (the length of time), and then it gives you this score that's pretty objective. You can actually rate your sleep, so that you can check the variables in your lifestyle that might be contributing to that, because it's this mystery essentially. Otherwise, there's no way to figure it out. I've just found it enormously useful, personally. It's so inexpensive that it seems foolish not to use it.

CK: I think that's a fantastic tool. And while we're on the subject, there are also now pedometers that can measure the number of steps you take in each day. They're really small. They're like the size of an AA battery. You just slip them through your pocket. One of them is called the Fitbit. If you want, it will automatically upload the data to a great tracking tool. There's a free web application called Dan's Plan that will post all that data and show you a chart of how many steps you're taking each day. There are some really exciting new tools that can help you implement these changes now.

DM: Yeah. I've known about Fitbit for a while, but I've never been really too intrigued with it, because my impression is that it really is trying to objectify or quantify the amount of exercise you're doing. But you know, walking, I never really considered as a great exercise. Because really, I'm a big fan of the high-intensity Peak fitness type of approach, where you're just going up to 100 percent of the total maximum heart rate, but only doing that a few times a week. That's something that Fitbit really doesn't integrate.

CK: It's interesting. I mean, there's a lot of research that suggests that a combination of that kind of exercise that you're talking about and lower-intensity exercise is optimal.

From an evolutionary perspective, when you think about the kind of movements that our ancestors did, there was probably a combination of both when they were either hunting, you know, really deep in the chase, or they had to run away from predators, a fight, or something like that. And then [there's] just more anthropological evidence that the average number of steps taken by our ancestors [inaudible 44:45] hunter-gatherers is around 10,000 steps a day – anywhere between 7,000 or 8,000, and 13,000 steps per day.

For me, the Fitbit is actually less about measuring the number of steps; it's more of a proxy way of measuring how much time you're not sitting on your butt.

DM: Oh, that's a good frame. I mean, I haven't thought of that from that perspective.

CK: And there are actually studies that show that pedometers are the easiest and most accessible way of measuring sedentary behavior.

DM: Interesting.

CK: If you look down at your Fitbit and it says 3,000 steps at the end of the day, chances are, you've been sitting way, way too long. So, that's the way I use it.

DM: I've never used it, because I've just never been enthralled with it. But now, I'm interested, because of the different perspective you've given me. So, if you could provide a range of objective ways of what's poor, good, average, and exceptional?

CK: I counsel my patients to try to really avoid any days less than 5,000 steps. Because when you look at the evidence in both anthropological studies and animal modern studies, healthy human beings are taking between 7,000 to 13,000 steps a day. So, I would say, below 5,000 is poor; 5,000 to maybe 7,000 or 8,000 is okay; and then above 8,000 is good.

DM: Okay. Well, thank you. I'll probably be picking up a Fitbit and see where I fit in that pattern. I always love this... Especially, you know, I'm a gadget geek.

CK: Right.

DM: That's one of the things I like about how technology advances so rapidly. We could take the value and the benefits of that and apply to health to give us this information, which is really cool.

CK: There's the [inaudible 46:31] scale. Have you heard of that? Which is a scale that not only measures your weight, but it estimates your body fat percentage. It's Wi-Fi enabled, so it automatically uploads your data to something like Dan's Plan if you're using it.

DM: Yeah. I've got the EatSmart one. It doesn't upload it, but I think it's still... It's bioimpedance. That's the way it determines it.

CK: Right.

DM: It may or may not be that accurate [compared to] some of the other techniques. But what is accurate is that it's very consistent, that if you see a difference, it does it within a tenth of a percent, which I think [inaudible 47:03].

CK: Yeah. So, you asked earlier about other causes of elevated LDL particle number. And we talked about insulin and leptin resistance, but there are a couple of others that should probably at least be briefly mentioned.

DM: Sure.

CK: One of those is poor thyroid function, which is something that's pretty common, a lot more common than you will believe. The reason for that is that T3 hormone (which is the most active form of thyroid hormone) is required to activate the LDL receptor, which as we discussed is what takes LDL out of the circulation.

If you have poor thyroid function or low T3 levels, then your LDL receptor activity will be poor, and you'll have a higher number of LDL particles. I do see this quite regularly in my practice.

The good news behind that is that if you address the thyroid problem, then the LDL particle number will come down.

And this gets back to your question about statins. If someone were to go into their doctor, they had high cholesterol or even high LDL particle number, and there's no investigation to what the underlying cause of the LDL particle number is, then there's an opportunity missed there. If they just get a statin to reduce the particle number, then they now have to find out that they have that thyroid problem, and that they're taking a drug unnecessarily that they don't have to take.

DM: Yeah, that's a good point. You may or may not be aware, but prior to 1980, that was one of the popular forms of treating high cholesterol. It was thyroid drug.

CK: Even in people who didn't necessarily have thyroid problems. That's my understanding.

DM: Yeah. So, what I haven't had an opportunity to look at clinically is the opportunity to evaluate that benefit of thyroid on the ability of people to optimize their weight and improve their ability to normalize the insulin and leptin resistance, and actually even integrate something like intermittent fasting. What has been your experience clinically when you find these people? And maybe even mention the testing strategy you would use, because that's a bit of a controversy, too.

CK: Yeah. My experience – and also from my reading of the literature – is that in terms of weight, normalizing thyroid function sometimes has a pretty profound impact on weight and sometimes doesn't. It's really kind of a mixed bag. It ultimately depends on what the primary cause of the weight gain was. However, I have a good response if someone has poor thyroid function, I almost always see a decrease in LDL particle number.

The tests I use to determine thyroid function are thyroid-stimulating hormone (TSH) (which is the standard one that's often done), but I also use free T3 and free T4 (the unbound forms of thyroid hormones), which is more accurate than the total thyroid hormone that are often measured.

[----- 50:00 -----]

And then there's another marker called reverse T3, which is a kind of decoy or inactive form of thyroid hormone that can block the thyroid receptors from being activated by true active thyroid hormones like T3. If someone has high levels of reverse T3 and even a normal or low normal level of free T3, then they might still be suffering from this effect, because their active levels of T3 will essentially be low.

Thyroid biology is a little complex, but the TSH, free T3, and reverse T3, I would say, would be the most important markers.

DM: Yeah. And addressing someone who is hypothyroid certainly can be done with, I believe, natural thyroid hormone best, as opposed to synthetic like Synthroid or Levothyroid. But it's still in some ways similar to using a statin for high cholesterol, because it's not treating the cause.

So, my understanding is that some of the most common causes would be the use of fluoride in your environment. It's similar to iodine. It displaces the iodine out of the thyroid. So, looking at that, but then also exposure to soy. Those are two of the most common ones.

For some reason, I don't know why, but maybe you might, it seems to affect women far more than men. It seems to be some sex hormone.

CK: Yeah, because of its effect on hormones and because of its effect on estrogen is why it affects women more. Women have more estrogen in their bodies naturally.

I would also add to your list bromide toxicity, which has a similar effect. Bromide can block the thyroid hormone receptors that block the uptake of iodine in the body. If you have excess bromide, which is a pretty ubiquitous chemical in pesticides like methyl bromide... It's in [inaudible 52:05] like in mattresses, furniture, and in sodas like Mountain Dew. That can be a problem. And I see that pretty regularly in my practice.

But interestingly enough, this is another case where inflammation fears [inaudible 52:20], because the conversion of T4 to T3 in a more active form of thyroid hormone happens primarily outside of the thyroid gland. The thing that impairs that conversion the most is inflammation. Specifically, inflammatory cytokines that are produced will cause T4 to be converted into reverse T3 instead of active T3.

So, let's say, for example, you have gut inflammation. You have irritable bowel syndrome, leaky gut, intestinal permeability, and dysbiosis causing this constant state of low-grade inflammation. As I'm sure you'll agree, Dr. Mercola, that's a really common problem that people are suffering from today. The inflammatory cytokines that are produced in that condition would essentially reduce the conversion of T4 to active T3, and increase the conversion of T4 to reverse T3.

It's really all about identifying the underlying causes and addressing them at that level as the first step and using medication as the last resort when those initial efforts aren't successful, in my opinion.

DM: Yeah, it's interesting that treating cholesterol may be best suited to addressing those issues that you just mentioned – the gut inflammation. And who would have thought the gut inflammation had this domino effect that would affect the thyroid that affects the cholesterol?

CK: That's right. And gut inflammation can actually even affect cholesterol more directly. There are some studies that show that lipopolysaccharide, which is an endotoxin that can be found in some types of bacteria in the gut... If the intestinal barrier is permeable, which shouldn't be, of course, some of that lipopolysaccharide can get into the bloodstream. LDL particles actually have an antimicrobial effect. So, LDL particles will increase if there is some endotoxin going into the bloodstream.

There are actually studies of mice that show that mice that have defective LDL receptors and mice with really high levels of LDL particles are almost easily infected from gram-negative bacterial infection. We not only have the indirect effect of that inflammation on lipoprotein or on LDL particles via the thyroid. We also have a more direct effect from bacterial toxin seeping into the bloodstream and causing a direct increase of LDL particles.

DM: That's actually where coconut oil can be useful, too, because of the lauric acid as an antimicrobial.

CK: Exactly.

DM: So, I just have one comment on bromine. Because I think from the general population, probably not most of the people watching this, that maybe one of the most common sources might be brominated flour, which is what they use to whiten the flour. The general population has loads of that. That may be something that has to be eliminated from the diet, too.

Now with respect to the bromine, I believe, of course, it's an important component. But in addition to the list you mentioned, I think most of the people in the country – not people that are watching this video – may be taking white flour. White flour is typically bleached with bromine. That might be another important source to eliminate. It doesn't do anyone any good. It's actually, I believe, even more potent or as potent as fluoride with respect to that detrimental effect.

CK: Yeah, I agree. I test people for iodine and bromide levels. Even in people who aren't eating bread, not an insignificant percentage has excess bromide.

DM: Chris, this has been absolutely delightful. I really greatly appreciate you taking the time and effort to share with us your knowledge on this and help us have a deeper appreciation of the complexities involved here, so that we can get some really useful strategies. I'm wondering if you could summarize for us your highlights and strategies on how to, first of all, know if people are having a problem, and then some of the strategies to address it if they have a challenge with their LDL cholesterol.

CK: Sure. Yeah. I think the most important test we can get to determine your risk is this NMR lipoprofile that measures your LDL particle number. And actually, something I didn't mention is that it has some other markers that are probably the best way of determining if you have insulin resistance.

Because as I mentioned earlier in the show, if you have insulin resistance or leptin resistance, you'll have an increase in LDL particle number and specifically the number of small LDL particles. This test measures that, too. And it gives you a really objective score called the lipoprotein insulin resistance score or LDIR. If that's high above the recommended reference range, the chances are very high that you have leptin and insulin resistance. That's a fantastic test and the most important one, in my opinion, for determining your heart disease risk.

DM: What's the cost of that test?

CK: Well, it depends. I mean, like I said, insurance usually covers it. But if you order it yourself from Direct Labs or Access Labs, it's about 100 bucks.

DM: Okay. That's not too bad. It's pretty reasonable.

CK: Yeah. And if you get it through a healthcare provider. For example, some prefer to come to me; clinicians usually get lower rates. It only costs me 65 bucks, so I pass that on to my patients. It's definitely affordable, and I think worth the expenditure.

So, if your LDL particle number is high, then in my opinion, the next step is to start investigating causes. We talked about two of the major ones – insulin and leptin resistance – which are also reported on that same test. So, you'll know right then if you have that. And then another cause was poor thyroid function. A couple that we didn't get the chance to talk about as much are genetics and chronic infections. But you would investigate the causes, see if any of those [inaudible 58:38] are present, and then address those.

Primarily, the way to address those is changes in diet, shifting toward the nutrient-dense-food-based diet with a lower carbohydrate intake in general. It will be helpful if you have that in insulin resistance. Things like intermittent fasting, as you mentioned, can be really useful tools. Making sure you're sleeping enough, making sure you're getting some of that high-intensity exercise that helps improve insulin and leptin sensitivity.

On the other hand, making sure you're not sitting too much, which can have a direct adverse effect on insulin and leptin sensitivity. Minimizing your exposure to environmental toxins as much as possible.

And a really important thing: addressing your gut health. So, eating fermented foods, soluble fiber that enriches the beneficial gut flora, and avoiding food toxins and things that harm the gut are really important.

Perhaps not surprisingly, the things that you would do to treat your high LDL particle number are the same things that you would do to promote optimal health in other areas. To me, that just makes sense. The things that keep us healthy in one department or one area are more likely to make us healthy in another. And this is no different from that.

[----- 1:00:00 -----]

DM: Okay, great. I'm wondering also if you can provide us some information if people wanted to know more details about the type of services you offer. Or if you have a blog, because I think actually you do have a blog. It's in the top 25 in the whole world. Because we keep a list of those, and you're right up there. So, congratulations.

CK: I didn't actually know that. Thanks for telling me.

DM: Yeah. You're in the top 25 natural health websites in the world.

CK: Good. So, my website is ChrisKresser.com. I also have an Internet radio show called Revolution Health Radio, where I talk a lot about these topics. And then I have some educational programs on my website that you can check out. I have one called the High Cholesterol Action Plan that goes into a whole lot more detail on the subject. It's a nine-week self-guided multimedia course. There's a lot more information there if somebody wants to learn more about this topic.

DM: How do people access your podcast or the radio show?

CK: You can find it on my website, and it's also on iTunes.

DM: Okay, perfect. All right. Well, thanks for all you're doing. We need more people like you out there that really are delving into the new information and providing some basics and strategies that really herald back to our ancestors essentially.

CK: Right.

DM: But at least providing a more comprehensive academic appreciation of why those strategies were working, and some important finetuning that we can do with new technology.

CK: Thank you so much for having me on the show, Dr. Mercola. It's been a real pleasure.

[END]