The 5 Cholesterol Claims Putting “Healthy” People at Risk Transcript

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Intro: Cholesterol Misinformation and the Top Myths

Alright, stop. Hold up a second. If you've heard any of these claims online, things like butter fixes cholesterol. L-deal is a scam. Don't scroll. Because cholesterol misinformation, it doesn't just confuse people.

It can cost lives. That's why today, I'm reacting to the top five influencer cholesterol myths that have gone completely off the rails. And myth number four, that's the one that looks healthy right up until it does it.

I'm Dr. Columbus Patisse. I am an interventional cardiologist and a lifestyle advocate. When prevention fails, I one of the ones in the cath lab at 2 a.m.

trying to open blocked arteries to save heart muscle. So first myth number one, cholesterol doesn't cause heart disease. It's only inflammation. Now this one sounds smart, but it skips the main driver.

it gets traction because inflammation is real. You've heard this, i have too.

Myth 1: Cholesterol vs. Inflammation

Cholesterol is innocent. Inflammation is the cause. Yes, I'll admit, inflammation matters. But here's the simplest way to think about it. Inflammation is solely the fire.

April B, that's a fuel, a log, the stuff that keeps it going. If you have less fuel you'll have fewer fires. So remember that cars near the wall idea?

Here is the biology version. Some April b particles, they can slip under the lining and get stuck. Now once they're under the surface, they stick to the artery wall like Velcro.

They get damaged. they get oxidized, which is like rusting. The clump up and their overall their change. So your immune system, it sees this and it rushes in and says that doesn't belong here.

And it sends the cleanup crew to eat it up. It like Pac-Man and then it turns it into foam cells, Which becomes the origin story, the beginning for plaque.

Now these foam cells don't stop there, they trigger more inflammation and the plaque continues to grow as long as the trigger stays there. Now here's why we trust this hypothesis.

When therapies lower April B type particles, heart attack risk drops. So this week, I want you to ask your clinician for April B or at least non-HDL. I Want you treat it like something measurable that you can reduce because you Can.

Fire is real, but don't ignore the fuel. All right, what's up next? Myth number two. My LDL is fine because my HDL Is high. Now this one sounds reassuring and that's why it's dangerous.

This is the HDL shield myth and it tricks a lot of people as well as doctors. My HDl is high so I'm good, right? Well here's the truth. HDO, it doesn't cancel out an LDL.

They operate through different pathways. Your LDL, it delivers cholesterol to the artery wall and it builds plaque. Now HDL it can help move cholesterol back to liver, but that doesn't erase the damage already done by LDO.

LDl and April B, they remain the primary drivers of atherosclerotic cardiovascular disease, regardless of your HDl level. But here's what we used to believe.

The higher HD l means more protection. Here's with the data actually tells us. Very high HD L is not a guaranteed win. Actually, in some studies, extremely high HDL was actually linked to higher risk.

So more isn't always better.

Myth 2: High HDL Cancels Out LDL

I say that more is not always be better, but here's the part that changed everything in cardiology. We try HDO raising drugs. The lab numbers improved and we got excited, the heart attacks, they didn't.

For example, to aim high, trial nice and raise HD on paper, But it did not reduce cardiovascular events. Now genetic studies they tell the same story, that simply having higher HDLC it isn't a reliable, a casual shield, the way April B is.

Now HDLC is a marker, not a mechanism we can count on. So here's the catch, your lab number, HDL-C, it's not HDO quality. People can have high HD, but HD that doesn't do its job well.

Same uniform, broken tools. One more thing most people miss. When HDL is low, there's often an unrecognized excess LDL particles, and those particles carry less cholesterol each, so standard LDLC can underestimate your true particle count.

That's why April B matters. HDl isn't a shield. April b, that's the scorecard you need to track. This week, don't celebrate HDLL alone. Keep your focus on LDL and April B.

That's where the proven benefit lives. Pair it with blood pressure and blood sugar. Remember, HDL is a clue, but April b is the traffic and we all need the Traffic Report.

All right, quick question. Have you ever been told that your HDl is high so you don't have to worry? Let me know in the comments. and then let your doc know what I'm talking about here.

Alright, you still with me? Good. Because this next one is a myth that doctors hate the most. Myth number three. It's not dangerous if it's large fluffy LDL.

Small LDl is the only problem. Now this one half true, then it goes off the road. If you've ever heard large, fluffy, LD l, listen close. I've had patients tell me, doc, small LD L is bad, large LD is safe.

Here's what's true. and what's misleading. Small dense LDL is more atherogenic per particle.

Myth 3: Large, Fluffy LDL Is Harmless

It doesn't get cleared as fast, so it hangs around longer. Its more prone towards that oxidation, and it can penetrate the artery wall much easier. And in many studies, small dense LDO, it tracks with higher coronary risk, especially in insulin resistance.

So yes, small LDL is a problem, but here's where the whole myth begins to fall apart. All LDl particles are atherogenic regardless of size. So consider familial hypercholesterolemia where you're born with high cholesterol.

Now these patients have large LD l particles and they still get heart attacks in their 30s or 40s. Large LD L did not protect them. Now there was a study called the CARE trial and it showed the same thing, that large cholesterol-rich LDL particles still predict coronary events.

So here's some of the key insights. When researchers adjust for April B levels, triglycerides, and insulin resistance, the association between small LDl and heart disease often weakens or disappears.

Small LD l may be more of a marker of metabolic dysfunction, but April b, it captures the actual risk. That's why

professional societies don't recommend chasing particle size.

The hierarchy is clear. LDLC, non-HDLC. APLB or LDL particle number. Not fluffy versus dense. Your arteries don' grade you on size, they respond to how many particles are hitting that wall, day after day, year after year.

Arteries don''t argue size only particle count. So this week, if you're worried about fluffy vs. small, simplify it. Get that APB, LDl particle, number and focus on lowering the count Okay, quick reset.

Remember, LDL size is the type of car, right? Small versus large. LDLC is a passenger count, but April B is car count. Are you ready? The next one matters a whole lot.

Myth number four. High LDl on keto or carnivore? It's just harmless adaptation.

Myth 4: Keto or Carnivore LDL Spikes Are Harmless

Now this myth, it ruins healthy people who feel amazing and it's the one that gets the most under my skin. And here's why. This is where the internet gets loud and your arteries stay quiet, but increasingly lethal.

This the one that I see getting healthy looking people in real trouble. So here's the part that's true about keto. Keto can improve triglycerides, HDL, blood sugar, insulin, pressure, and body weight.

And that can feel like a win. It is when you're moving away from a standard American diet. But here's what we keep seeing. When researchers pooled lots of studies and we kept seeing ketogenic and low carb, high fat diets raise LDL in April B across diverse populations including healthy, normal weight people.

And the biggest spikes? They tend to happen in people with genetic predisposition to high cholesterol. So you can't out vibe your way out of genetics.

Alright, so here's the part that influencers skip. Some population data, it suggests that low carb, high fat patterns can be linked to higher atherosclerotic cardiovascular disease events.

Especially when LDLC is very high, like above 190. So some, they will call this physiological adaptation, the body just adjusting to new dietary fats.

That is a hypothesis. It does not establish the elevation is safe, at least long term. And we know that long-term outcome data in metabolically healthy people with diet-induced high LDL, we don't have data on that yet.

Trials are still ongoing. So here's what we do know. Here's the what the research confirms. Reductions in triglycerides in lipoprotein A do not erase increased risk signal from elevated April B.

Better triglicerides don' erase high particle count. As a matter of fact, the American Heart Association has expressed concerns about dietary eating patterns surrounding ketogenic diets, especially when they include a lot of saturated fat and very little fiber.

Now I want to tell you what happened to Marcus. Marcus was healthy looking. He was an executive. But as April B, it was on the up trend. It was high. And he told me, listen, Columbus, we're friends.

I feel fine. Don't worry. Then one random Tuesday, his chest started tightening. He went in and the EKG wasn't normal. By the time he was taken to the cath lab and they looked inside, he had a dangerous blockage.

that did not care how confident the internet sounded, the stories that were told, his HDL level in isolation. It cared about the April B level. If you're keto or carnivore, I'm not here to shame you.

I am here coach you, help you lower your risk. A diet label doesn't protect the artery wall. AprilB does. So this week, I want you to ask for April B or LDL particle.

Inspect what you expect. Just check it out. If your LDLC exceeds 190, that's a conversation you need to have with your provider. You may well consider running a six to eight week fat swap experiment, then rechecking it.

Now one practical move if you want to stay low carb but lower risk, remove the garbage. I call it garbage is that ultra processed refined carbohydrates but prioritize plant-based foods.

No butter, coconut oil, fatty, processed, or red meats, but eat olive oil nuts, seeds, avocado, and fiber-rich plants. Keto can improve some of your markers, But you still have to measure your April B and digest.

Okay, one more myth. And this is the one that everyone wants to believe. Myth number five. Dietary, cholesterol, saturated fat don't matter. This one sounds like great news, it's only half the story.

And it gets shared because people want it to be true, but the data is more nuanced. And I hear this every week in clinic or the cath lab. Doc, eggs don't matter.

Time Magazine says butter doesn't matters. Saturated fat doesn' matter, it's all just calories.

Myth 5: Saturated Fat and Dietary Cholesterol Don't Matter

Well, here's the truth. Saturated fat, it matters because it raises LDL. And in many people, erases April B as well. That's the primary mechanism of harm.

More atherogenic particles driving athrosclerosis. But here's what most people miss. It's not just about cutting that saturated fat. it's about what you replace it with.

We can't just live in a cycle of what we're not eating is what are we eating for our health. This is where the data gets powerful. When saturated is replaced with unsaturated fats like nuts, seeds, plant oils, cardiovascular events, they drop meaningfully.

And here's the swap matters idea. Replace just 5% of calories from saturated fat with unsaturated fat, risk drops. So use this replacement hierarchy. The best?

Swap butter, fatty meats for nuts, seeds, olive oil, and fiber-rich plants. Good? Maybe you swap it for avocado, olives, or other mostly unsaturiated fats.

No Win? swap for refined carbs like white bread and sweets. The worst, keep the saturated fat high and the fiber low. Remember, saturated fats, it raises the car count for a lot of people.

So this week, run the real experiment. Check your April B, or at least non-HDL. Make a smart swap for six to eight weeks. No butter, no red meat, and no processed meats.

Adopt the nuts, seeds, avocado, then recheck your numbers. That's how you turn nutrition into something measurable and personal just for you. Here's my simple model.

Plaque risk, equals April-B exposure. times the artery wall stress times time. Now I'll tell you what, April B exposure is the particle count. Arterial wall, stress, that's your high blood pressure, your higher blood sugar, you're smoking, the insulin resistance.

And time or duration, That's the part no one wants to hear because plaque, it's a slow thief. It's silent operator. Don't wait for symptoms. So here's what I want you to ask your provider for.

Catch this. Can we check my April B? What's my non HDL cholesterol? Should we checked lipoprotein A one time? Now, if you're in a gray zone, ask you doctor if a calcium coronary score makes sense for you so you can have additional evidence about if already have manifestations of atherosclerosis.

All right. Here's the 22nd version upgraded. Three takeaways. One, track the April B or at least non-HDL. Two, lower that artery wall stress, blood pressure, and blood sugar.

And don't smoke. Three, be consistent long enough for time to work for you and not against you. Here's the global move, clean and simple. Run that April b plus smart swap experiment so you can lower your risk and stay out of the cath lab.

Because if staying off the cath lab table means being present for your kid or your grandkids graduation or life, this isn't about winning an argument, it's about keeping your life on schedule.

Marcus didn't need a better debate, he needed better data and a plan. My goal is never to win an arguments, my goal to share the data, My Goal is to keep you out the Cath lab because cholesterol misinformation it doesn't just confuse people, It can cost lives.

I want you to drop the next cholesterol claim you've heard online, and I'll react to it with some science in the episode, or at least a future one. Remember, if you want to go fast, go alone.

If you wanna go far, then go together. Let's go Far Together by building healthier hearts in stronger communities. I need you like it, share it and send it.

Save a life. See you next time.