Paleo Solution - 400

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Robb: Hey, folks, welcome to another edition of the Paleo Solution podcast. One of the hottest topics, I think, that ever come up in this health and wellness story is one of cholesterol levels, lipoproteins, the likelihood of developing cardiovascular disease. I just had a fascinating conversation with a brilliant guy, Dave Feldman.

Dave is an engineer by training. He, due to some health challenges, started exploring this Paleo low carb keto way of eating but noticed some really whacky responses to his blood lipid levels. Armed with his engineering background and this orientation towards systems, thinking and distributed networks and energy metabolism and whatnot, Dave has just come up with some really fascinating explanations behind some of what we see in this lipoprotein cholesterol world and the implications for our overall health.

We had a fascinating conversation. Unfortunately, we had some technical difficulties and the podcast dropped off partway through and we weren't able to resolve that in a timeline that worked for both of us. What we're going to do is have Dave back on in a couple of weeks. I think you are all likely to have some significant questions for Dave. Enjoy the chunk that we have here today and then we will get Dave back on in a couple of weeks. I'll ask you guys for some questions and we'll circle back around and have another conversation with him soon.

Hey, folks, Robb Wolf here. Just making sure our technology side of the recording is working. Dave, how are you doing, man?

Dave: I'm doing fantastic. Thanks for having me on, Robb.

Robb: Cool. Dave, we met at, let's see here, the Ketogains Seminar in Las Vegas. Is it coming up on two years ago now? Has it been that long?

Dave: Wow. Yeah, 2016, October of 2016. That's right.

Robb: Yeah. Holy smokes, man. That's been a while. I had been following your work prior to meeting you in person but I got to say, just had my hair blown back. If I wore a toupee, it would have been a mile behind me by the end of your talk. It's interesting. You're another one of these folks that I feel like had been moving the needle on this health conversation in a way that's really remarkable.

You, Marty Kendall, Ted Naiman, these folks that come into this story with an engineering background and really no biases as to which horse in the race, which dog in the fight is the ultimate favorite with regards to what's going to work best for performance, health and longevity. You arrived on the scene, again, with this phenomenal engineering background, great data analysis, and you got super geeked out on the topic of cholesterol and lipoprotein and everything related to that. Could you share a little bit of that process with folks?

Dave: Absolutely. I mean, it's amusing to be finally talking to you now because, you're right, you were there at the very first public presentation of my data. What had happened--

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Robb: And you were still collecting data. That day, you were like, "Okay, I've got to go eat this food because I've got to take a blood test here in an hour." Yeah. I remember that.

Dave: It was a very amusing event. Yeah, because, basically, what happened was just by the year before that I got my first cholesterol test after being keto for seven months. My cholesterol had shot through the roof. This was surprising to me because both my biological dad and my biological sister, my only other sibling, had also been inspired and gone on keto at around the same time but their cholesterol didn't. It bumped a little bit but mine went way high.

I then, being a software engineer, as I was learning about lipids primarily out of fear because I was concerned about what this could really mean, I found that it looked in many ways like a network, something I see in my industry quite a bit. I stand by that to this day. There's really enormous numbers of things in common with how your cholesterol moves around in your body and the objects that it carries it in called lipoproteins.

A year later I finally locked down this inversion pattern, as I call it, which is an intuitive and unlike anything you typically read out there which is actually the more fat you eat the lower your total and LDL cholesterol tends to go. Likewise, the less you eat, the higher it tends to go, at least on a ketogenic diet. At that conference that we met at, I wrapped, I intentionally wrapped an experiment around the conference. It's part of why it makes it a little--

That's why it gives me a little bit of nostalgia just even talking about it because, basically, you saw me in a presentation present this slide where I had my blood taken from two days earlier and I had a picture of it on one side and on the other side I had a picture of a silhouette because it hadn't happened yet. I said, "Look,

I'll bet my reputation and all my data that what you're watching right now is my LDL cholesterol plummet."

And you're watching it as I'm eating huge amounts of food all through this conference. I had a cooler next to me. I'm downing a whole bunch of fatty meats and cheeses and fat shakes and so forth. Sure enough, I get my blood drawn the day after that presentation and that's exactly what happened. My LDL cholesterol just absolutely fell through. I mean, it drops 73 milligrams per deciliter. My LDL-P dropped 1,115. It was astonishing but it was as I expected at that point knowing the inversion pattern as I did.

Robb: Dave, the thing that was -- I mean, there were lots of things that were mind-blowing on that but one of the biggest, and I took this back to our clinic which I sit on the board of directors of a lipidology clinic and very smart people, solid folks, love the work that they do, but when I came back after that weekend, I was like, "Hey, guys, what generally is the consensus on how long it takes for lipoproteins to have a real significant delta, for a real significant change to happen?"

It came back anything from weeks to months. It's like that, like finger popping deal**,** leaning back and I was like, "Well, what if I want to suggest that there's some folks out there doing a little bit of N equal one stuff and it might change on the course of hours leaning towards days?" They were, "That would really odd and we would have to rewrite the textbooks."

I actually floated them some of the material you shared with me and all I got back was crickets. I funneled this thing up the chain of command. I mean, I'm not going to throw anybody under the bus again because I love these folks and they do amazing work but it was such a controversial counterintuitive curveball that I threw at them by sharing your data that then even to this day I don't know that they're really sure what to make of the whole thing.

Just so that people can appreciate this, and maybe stepping back again, in general, when we're talking about cholesterol levels and blood lipid levels and stuff like that, it's generally been accepted that it takes a decent amount of time for this stuff to change in a significant way. We're fairly comfortable with the idea that blood glucose levels could go up and down over a 24-hour period and could vary by a decent amount, but it's thought that these blood lipids particularly the cholesterol and lipoprotein fractions, those things are pretty static.

What you were doing was taking these things and sticking them up into the stratosphere on the one hand, and by doing that you were generally eating less food and less fat and then you were plummeting these things.

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Just for folks to appreciate, you were getting a thousand point LDL particle count change. Many people only have a 1,000 LDL particle number. You were changing your number by a factor that oftentimes represents the total pool of lipoproteins in some folks. That's a pretty accurate characterization, right?

Dave: Yeah. I mean, I dropped it. It was a little above 2,000. Effectively, I dropped it in half. You and I both know that there are many articles that are out there right now that give five, six different little things that you can do to tweak your LDL-P to hopefully bring it down further. I think this is the point that cannot possibly be missed. It's not that I dismiss that there aren't other factors that impact your lipoprotein number. That might be epigenetic. That may be related to environment or the nature of the foods you have or particularly the nature of the fat types you have.

But all of that said, I definitely feel stronger than I ever had in my life that the single greatest influencer on your existing LDL cholesterol and thus your LDL-P, the particle count, is the nature of your energy metabolism, how much of it is fat based and how much existing energy stores you have? Between those two, I would raise against any pharmacological impact there is. Like right now, I would love to take anybody -- Anybody who wants to set up a race between me and your favorite drug of choice, I'll take that on right now.

Robb: Right. Even for me, I'm still grappling with what the implications of all this stuff really are. Again, in our clinic, we really go after, we're looking for the insulin resistant individual particularly within police, military and fire populations. We find this discordance process frequently within these populations, the sleep deprivation, the hypervigilant state, elevated cortisol and catecholamines seem to produce a scenario where LDL cholesterol may not be particularly high.

These folks maybe don't have a super squirrely triglyceride to HDL ratio. They don't look really insulin resistant. They're maybe a little bit. If you've got an educated eye, you're kind of like, "Not really liking this trend here." But in general, these folks are passing through general physical examinations but then when we run their advanced testing looking at their LDL particle count and doing something called LPIR score, a lot of these folks within the police, military and fire, in particular, they have what's called discordance.

They have comparatively low cholesterol levels, very high particle count, and people get geeked out on the large versus small. I think as time has gone on we've understood that maybe isn't as big a factor. Basically, that just plays itself out in the wash. But these are the folks that appear to be having cardiac events.

They're 35 years old. They're doing triathlons. And then they keel over and die from some sort of occlusive cardiac event.

What is your position on the role of lipoproteins in that process? I think we would both agree that it's a matrix driven scenario that there's lots of different inputs that tweak these variables, everything from glycemic load to thyroid status and all this different stuff. We've seen folks like yourself who have super high cholesterol and lipoprotein levels but you do coronary calcium scan, you've done some of the 3D imaging and as far as we can tell, if you're going to die from something, it doesn't look like it's going to be from a cardiac event, at least based off of some of these things of looking directly for vascular pathology. I guess, my long winded thing is what the heck is going on there? What's your take on all that?

Dave: Sure. Well, definitely a lot to unpack. I want to say the very first thing that matters in my opinion more than ever, and should to your listeners, if there's any one thing I'd want them to take away, it's that I now when I see any lipid panel, and particularly just the basic lipid panel, I am absolutely looking first to triglycerides and very close second to HDL.

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Those two, I've now got two extremely strong studies that showcase just how when those two are taken into account, LDL becomes nearly meaningless. Not meaningless, but nearly meaningless. A good analogy would be that LDL is like a scale. If you had no information, you couldn't see the patients or anything, but all you had was a weight scale, you would have a loose association between poor health and high weight.

Certainly, somebody who is 400 pounds is probably not in a good way health wise. But that's not going to be a large number in your sample. What about somebody who is 200 pounds, 250 pounds? For some, that would be unhealthy. For some, it wouldn't be. It really would help if you had more information. I think HDL and triglycerides is like having also height and weight circumference.

If I were to say, "Robb, I want you to diagnose based on either having a scale or having height and weight circumference," it would be no contest at all, right? In that sense, I have actually codified a challenge that I have online that hasn't yet been met, which is that I ask, it's called the Low Carb Cholesterol challenge. The hashtag is LCcholesterolchallenge.

I said, "Look, a lot of people seem to be comforted by having high LDL so long as they have low triglycerides and high HDL cholesterol." And I have yet to find a study, a non-drug study, so just a normal population of people who have high

rates of cardiovascular disease even if they have high LDL so long as they have low triglycerides and high HDL.

I pinged a whole bunch of lipid lowering proponents, bigwigs all over the place that have never been able to produce one study that meets this criteria. Not one study. But conversely I managed to find studies that showed the opposite, that you can have extremely high levels of LDL cholesterol and yet be in excellent shape so long as your triglycerides are low and your HDL is high.

So, why am I emphasizing this? This makes it make sense as to why it is that that means you're metabolizing fat effectively. The triglycerides are the cargo in the boats of the lipoproteins. And if those triglycerides are low in your bloodstream, it suggests that there's neither a broken metabolism, a broken energy metabolism, because we tend to see with high triglycerides more cardiovascular events. We tend to see more all cause mortality.

But it also means that there needed to be that much LDL-C and LDL-P, the boats that carry the cargo, in order to meet the demands. We are seeing it as a lot of empty boats. So, getting to the point you were talking about where you do see some people who have a lot of empty boats but they're small boats. There's other processes that can be involved that usually show an also the HDL as well. I'm willing to be that a lot of people you're seeing with a small boat also have unusually low levels of HDL, right?

Robb: Right. Just really quickly. This dovetails back into Gerald Reaven's original observations around the metabolic syndrome, this increase in triglyceride, decrease in HDL as two of the key features of that.

Dave: Exactly, and not LDL. If you're really watching triglycerides and HDL, they actually provide a very powerful window into your health. That's why there's a lot of people who will come to the site and it's really a small subset of people who are on a low carb diet. But there's some outcome to the side and they go, "Oh, yeah, my LDL raised but from your research I gather that I shouldn't worry at all about my lipids because it's pretty much meaningless."

And I'll go, "Now, hold on a moment. You're telling me your triglycerides are 200. That would concern me. I would like to actually know more about that and why it happens." Incidentally, by the way, the most common reason somebody on a low carb diet comes in with a high triglyceride count is because they were given the advice to not have a fasted cholesterol test, which I highly advise against.

I advise having a fasted cholesterol test because if you're on a low carb high fat diet and you have a fatty meal, say, a few hours before your cholesterol test, you

literally loaded triglycerides into your bloodstream and, therefore, will have spiked out your triglycerides for your cholesterol test.

Robb: We're basically testing the components of that meal, not your background status. Yeah, absolutely.

Dave: That's exactly right. It's unfortunate because that is a lot of people we get. I want to answer one other thing, which is people need to be mindful of coffee. That's really showed up on our radar a lot.

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There seems to be, not everybody, but a small subset of people who are on Paleo or low carb and for that matter on a carnivore diet who had this unexplained really massive jump in the triglycerides. We asked about coffee. Oftentimes they turned out to be coffee drinkers and when they cut off the coffee then the triglycerides go down.

I hate to be the bearer of bad news, but I don't know yet of any good reason or any case that can be made for having high levels of energy in the blood be it glucose, be it triglycerides. I even take some flak for this, but I believe that there probably is such an overage for ketones. The issue is you just need to think about it mechanistically. Energy, your body is running a buffet, not a diner. That means, as a buffet, it's trying to make just enough that's available on that buffet line which is your bloodstream to your tissues so your tissues have just enough energy that they can make use with probably a little bit more than is necessary but not much more.

When you see a rise in the energy in your bloodstream, that's a huge signal. That's very important to pay attention to because that usually means there's a downstream problem with your health.

Robb: Dave, you were one of the first people that, both you and Marty Kendall, again engineers, funny that you guys zeroed in on this, but making this case that in general, again in this background state of in between meals, overnight fast or what have you, you would really probably like to have the bulk of our energy substrates sequestered away.

The lipids tied up in fat, esterified into the fat cells. We'd like the glycogen or glucose stored in glycogen either in the liver, in the muscles. And then when we need that stuff, say you start exercising, you're doing different things, then we're also pretty crafty and efficient at mobilizing and getting that stuff out into circulation and being used as a substrate for energy.

Because there is a reality. Just glucose is an example. When it is stored as glycogen, it's pretty benign. It's not glycating anything. It's not causing all these metabolic problems. But once it's in circulation, it's game on. Some percentage of that is going to glycate proteins and we need some insulin to manage it and stuff like that. To some degree -- and then on the flipside with lipids, these things can oxidize, the cholesterol fragments can oxidize and whatnot.

You want it the hot side hot, cool side cool. You don't want a bunch of spillover. We could probably make a credible argument that when people are just generally in that overfed state, which can happen a lot of different ways, the body starts trying to stick nutrients just about anywhere. Some people end up getting these cholesterol plaques in the eyes because the body is literally -- it makes me think of some of the old prison movies where they're digging a tunnel and they'll take the dirt and put it in their pants and then go play basketball with the dirt dribble out their pants.

They're just trying to hide stuff anywhere it will fit. And so, again, this is one of the arguments whether you go high carb or low carb, if you're overweight and you have metabolic problems we really want to figure out a way of spontaneously reducing caloric intake so that we can offload that excess energy and then get back into this dynamic homeostasis of we use it when we need it and then otherwise it's stored.

Dave: Yeah. It cannot possibly be emphasized enough, that, I as an engineer, I'm looking at this absolutely astonishing, for lack of a better term, operating system, this human operating system. I mean, I have to tell you, Robb, I wrote my first line of code when I was 13. This is 30 years ago. I felt very dedicated to software engineering and had no interest in medicine or nutrition or anatomy, any of these things.

I now feel like this is such a powerful new linear transition and leaving that with engineering eyes and understanding design patterns and all the things that apply to that side of the world and taking it over to this side of the world because I have such respect, profound respect for how effective the body is in making use of energy and metabolism. Let me tell you, the whole system has that as its primary directive. If you look at how everything is structured, certainly there are many different systems in place but if you can't keep the lights on there's no point.

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So, it's very, very good in anticipating your energy needs. And even the different sample passes that goes back with certain amount. That's why there's this three-window with the inversion pattern. It makes perfect sense to me that the body

has effectively baked in the code to go, "Oh, okay, you seem to be exercising a lot more lately. I should probably prime a little bit more glucose in the morning. Oh, and I should also -- You seem to be eating a lot less lately. I should go ahead and mobilize more of these fatty acids since you tend to be fat-adapted more than--"

And it does this without much knowledge on our part. We don't actually feel the differences to the degree that you would think for how much it's changing over these substrates. So, getting to your point about fat being stored in places we wouldn't expect it to be stored in, people really don't respect our adipose tissue to the degree it really deserves. It is professional at grabbing fatty acids, esterifying them into triglycerides, storing them, and just releasing them a la carte into its locale very effectively to keep the lights on for the nearby tissues.

This is what's so neat about it, is the reason we see very high levels of LDL cholesterol with those people who tend to be leaner, is it's really just more of a global sharing of the fatty acids from the adipose tissue. Your fat that comes from your left forearm and your fat that comes from your right forearm can make it to the tissue in your right calf a lot easier since you don't have as fatty of a calf. Whereas if you had a fattier calf then you actually might have less LDL because you have less of the precursor VLDL that carries those fatty acids because it was already nearby in the fat that was near there.

That's all by design. So then what happens? What happens when tissues that are like the liver, the pancreas, even the heart are accumulating fat? What that says to me is we ran out of the degree of leverage we needed in the adipose tissue. We hit our personal fat threshold. And this makes a lot of mechanistic sense because there is a worse place for the fat to hang out than in the adipose tissue and that's the bloodstream.

That's a very scarce space. That's a very important freeway to virtually everything that needs to get anywhere in the body quickly. Of course, there's going to be affinity thresholds where, hey, if muscle tissue that needs it doesn't need it then, okay, then it goes to the adipose tissue. Does the adipose tissue have plenty of room? Yes, it has plenty of rooms even within a personal fat threshold. But if you max that out, then we have less desirable decisions down the road, the liver, the pancreas, et cetera, by golly, we'll put it there before we let it stack up too much in the bloodstream.

That's why when you see these high levels of fat in the bloodstream it's finding that higher level of, or that lower level of affinity which is why it's such a reflection of a poor metabolic system in the first place.

Robb: Really well said. I'll try to paraphrase that, and correct me if I get this wrong, but the case that you're making is that if somebody is carrying a significant amount of fat in general we might not be surprised to see more of this LDL-C and basically the lipoprotein because they may not need to shuttle this stuff around as much. In a lean individual, we're relying on more global fat distribution instead of localized fat distribution so you might see a little bit more shuttling.

But, Dave, how would we distinguish between the lean individual who is relying on more global fat storage versus the overweight individual that these lipoproteins are going up because it is spilling over in the blood? Like if we were to have each of those people side by side, what are the couple of things that are different there?

Dave: It's the HDL and the triglycerides. It sounds too simple to be true, Robb, but that's why I identified a profile about a year ago that I'm sure you've heard of by this point I call lean mass hyper-responder. If your followers haven't heard of hyper-responder before, that was a term that predates me, for just people who went on a low carb diet and saw their cholesterol go up, and that was all generally very unknown.

Last year, I kept identifying and pointing out that there's a particular pattern of people who go keto especially and tend to actually be very lean and/or fit and have the highest levels of LDL. But they likewise tend to have very low levels of triglycerides, often under 70, and I often even see them in the 40s of even 30s and typically have very high levels of HDL cholesterol typically over 80 and oftentimes I'll see them even in the hundreds.

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You can't even imagine this profile. But again, it's worth repeating this. Even for as high as their LDL-C is, and then we checked it out and their LDL-P is 2500, 3000, 3500, their triglycerides are just absolutely tiny at like 50 milligrams per deciliter. You have to think like an engineer with me for a sec. What are we seeing? We're seeing a whole bunch of boats that started out as VLDLs when they left the liver loaded with 2500 molecules of triglycerides, each of them.

Clearly, they're now LDL particles, their succeeding stage, because they dropped off all of these triglycerides successfully. And now they're the smaller boat that ends up afterwards that are now on a support role. They're succeeding at dropping off the energy. The big question then becomes, okay, so maybe this is the energy explanation as to why lean mass hyper-responders would have higher levels of LDL-C and LDL-P. Then that begs the question, is it still an independent risk?

Robb, I'm telling you, that's why I had the low carb cholesterol challenge. I cannot find any piece of data that supports that. Not one piece of data. And I'm open-minded. I absolutely, as politely as I can ask anybody, I want them to find me something, anything that shows if you have high levels of LDL but low triglycerides and high HDL, that you're at risk, not even just for all cause mortality but for cardiovascular reason, cardiovascular health in particular.

That's why I started a group just recently. I did a speech about a week and a half ago about lean mass hyper-responders. I have this Facebook group because I want to share this data and I want them to share it with each other. Are there any lean mass hyper-responders? You see the things you just talked about. You see fat deposits in their eyes, xanthomas, for example, that we see with people who have FH.

Do we see rapidly increasing carotid intima-media thickness from CIMT scores? Do we see rapidly increasing CAC? And while it's a small data set, it's just not there. I'm not seeing a whole bunch of these lean mass hyper-responders showing clearly progressive cardiovascular deleterious health. Now, I want to be a good scientist. Small data set. We don't know for sure. It's self-reported so it's possible that the ones who are having positive results are the ones who are coming out the woodwork to tell us about it.

But that's all the more reason why I want to be able to watch these people over time, maintain contact, and have them keep sharing the data with each other because it's pretty exciting, Robb. These guys, these lean mass hyper-responders may be the ones to really give a body blow to the entire lipid hypothesis.

Robb: Man, it's fascinating stuff. One of the things that's intriguing to me, and this maybe gets out in the weeds a little bit, but within this population of folks that go low carb or keto and we see pretty dramatic elevations in lipoproteins specifically LDL-P and LDL-C, oftentimes we trace this back to some issue with dairy in particular. Dairy seems to be one of these things that can really fire those numbers up and then if we shift folks to more of a monounsaturated fat, more olive oil, more nuts, we may see just a shocking decrease in the numbers.

That's APs. And then we have another cross-section of people that will get the same lipoprotein elevation. But if we take them from 30 grams of carbs a day to 75 grams of carbs a day we see those lipoproteins plummet. What's going on in those two scenarios? I'm particularly interested in the monounsaturated fat versus the dairy story.

In the back of my head I'm thinking about the role that lipoproteins play and innate immune response. There is a reality that short chain saturated fats are really good at moving lipopolysaccharide through the gut membrane into

circulation. Lipopolysaccharide can be proinflammatory agent and nothing clears lipopolysaccharide like lipoproteins do.

Again, I'm still in this situation where I'm like, okay, does that even matter? But there's a great mechanism there for why certain types of fat may elevate lipoproteins because it's actually the lipoproteins are detoxifying this lipopolysaccharide that comes off the outer surface, membrane surface of bacteria. What are your thoughts on that? I know that was a ton of stuff and not well articulated.

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What are your thoughts between that -- We have a situation of lean mass hyper-responders, lipoproteins can go up, this is probably a response to fuel partitioning needs. But then why do we have this situation where shifting either a little bit more carbs, a little bit less fat in general, or shifting fat quality dramatically shifts those lipoproteins?

Dave: I have theories that actually hit each of these in isolation. Let me start first with the higher levels of carbs. Again, I'm not sure how close you follow my research but literally the whole second phase of my research was isolating exactly what that threshold was within myself and determining where it was.

Imagine it this way. You have a gigantic walk in freezer in your body called adipose tissue. That's great. But the problem is, when you pull things out of it, you have a whole thawing out stage. The food is not just good to go. But then you have a refrigerator and the refrigerator is really small. We'll call the refrigerator your quick access food and that's going to be your glycogen stores. That food's pretty good to go. You throw it in the mike, it's ready.

The problem is, if that gets really low, if the glycogen stores get low then your body actually has a reason to be pulling more stuff out of the walk in freezer and already be thawing it, if you will, and getting it ready. So, I for a while there was playing with exactly what thresholds of carbs can I swap fat out for? So, keeping it isocoloric, I'm actually changing out the energy that I'm getting from fat on a ketogenic diet to specifically glucose in order to top up my glycogen stores.

I believe this is primarily in the liver. You have glycogen stores both in the liver and in the muscles but I believe that the liver, because it's a central regulator to all of the energy in your body. It's, of course, aware of its own stores. This is how I would design it if it were a network. If it were a network and I had a central server then if it were sending out this -- I'll take it back away from the server analogy for a sec.

If you're sending out all of this fuel into a dark maze, but the one rule you can count on is all of the containers of that fuel come back to you, well then you always have a decent gauge of what the distribution is based on the push-pull that's going out for those that are collecting the fuel that's out in that dark maze you can't see. But you have a short term energy supply that you need to be mindful of and you have the long term energy supply.

The short term is one that you have a lot of focus on. Well, you have a lot of glycogen stores in the liver then you don't feel as much need to mobilize as much of these fatty acids. Therefore, you don't have as much need to make, to put out as many of the VLDL boats so that tissues have the, of course on VLDLs, the fatty acids to make use of because you know you can respond in case the saber-toothed tiger comes around the corner because you're going to release tons of glucose. Therefore, less need. It's a load balancing thing, if that makes sense.

Now, I want to talk for a moment about the monounsaturated versus saturated. So, here's the catch. If you've watched fat profiles a lot of times if you're getting monounsaturated you're also gaining a little more polyunsaturated typically speaking. It's usually like saturated fat versus mono and some poly in a lot of these profiles and that's part of what I want to isolate down because we do know this, if you isolate in particular polyunsaturated fats and have a lot more of those, you will have a lower LDL.

But the problem is we now know more the physiology behind that and it stands to reason given a lot of these existing studies that part of the way you're reducing the LDL by having more PUFAs is that PUFAs, polyunsaturated fatty acids, have a unique capability of oxidizing LDL. And if you have oxidized LDL but gets modified, there are receptors in your body called scavenger receptors where they'll take it back out of the bloodstream because it's not supposed to be oxidized. I mean, it's actually a [inaudible] response.

Robb: It's the worst of the worst. Yeah.

Dave: Right. So, we don't have any capability, any easy capability in vivo to detect scavenger receptor activity. But we do have a capability to capture degree of oxidized LDL to some degree.

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I'm not super happy about the test that exists right now but bottom line is this, if the way by which you're getting a better blood test or having lower LDL-P is that you're oxidizing more LDL-P, I don't consider that a positive outcome. I don't want to--

Robb: Right. This is some of that whacky stuff that came up from the buried Ancel Keys research where they had done effectively metabolic ward studies on psych patients. This is pre-IRB stuff and so these people were fed higher saturated fat diet, higher monounsaturated fat diet, the high monounsaturated fat folks, their cholesterol plummeted and their all cause mortality was much higher. This stuff was round filed and just came up.

I think your point there is like, okay, yeah, we dropped the cholesterol, we dropped lipoproteins but the way that we did it is in this super oxidative proinflammatory process that is super -- like it's kind of the gnarliest shit that can happen in the vascular system. You do not want that stuff happening at all.

Dave: Right. To be sure, that experiment, the Minnesota coronary experiment, that experiment, it had some flaws to it but it was the only double blind experiment of its size and I was genuinely impressed at the nature of the data that they got because it couldn't have gotten spun in many ways which is why it wasn't really broadcasted very loudly at the time that its results came in.

But this gets back to the original point. I like this statement in engineering and it's kind of a crass one so I'll give you the PG version. Don't blame the mirror for how bad you look. That's how we look at it from networks. There's well-built networks and then they don't seem to be running well and so you then blame the network but a lot of engineers would be like, "Now, wait a sec. You decided to throw in this load of a million users into one that was only ever set to scale for 30. That's not on us. We designed it correctly against what it was."

This is where I want to get back to ancestral health which applies a lot more to your users. What has our operating system, the human operating system, had a lot of practice with in metabolizing energy? It had a lot of practice with fat, it had a lot of practice with protein, and it had a lot of practice with fibrous carbs. Lots of practice. Tons of it. Millions of years in the animal kingdom.

We have not had a lot of practice with exactly this, with energy that upon digestion can very quickly get into the bloodstream, much faster than our hormonal coding has been used to dealing with. And that's why I actually can bring this back to dairy. One of the things that I have people cut out if they're stalling or if they have very high levels of triglycerides, and this has been with my own family and friends, is I have them cut out liquid or refined forms of fat.

I have to cut it out because it is another means by which you can get energy quickly into the bloodstream. So, try for example to overeat a fat shake of a thousand calories where it's almost entirely heavy whipping cream and water with some stevia and unsweetened cocoa. It's pretty easy to do. Try to overeat a

thousand calorie of steak. Good luck with that. Try to overeat a thousand calories of eggs. Good luck with that. You just can't do it.

It's not that I think everybody should cut that out. It's just that those people who are having problems with, particularly having high triglycerides, it's one of the first things I'm looking at. How did this happen? How did you end up with triglycerides in the blood? Because I do want to tackle that right away because I do see that as a big threat.

Robb: Oh, it's fascinating. It's interesting just even on the empirical level like our good friends at Ketogains, in the beginning of their process, they really advocate for people to not use any dairy in the first iterations of their process and they just kind of empirically have noticed that it tends to stall people. They don't make as good a progress.

Dairy is damn tasty whether it's cheese or butter or cream or what have you, and I know it's not everybody's cup of tea, but man, I can put that stuff away. Even on the hyperpalatable side of this stuff -- Individually, the dairy is good. When you add dairy to something else, it makes both those things pretty amazing.

We're in that early stage of somebody being significantly overweight and we're trying to introduce some sort of a caloric deficit to offload this energy, we do want a low palatability food experience, as low as the person can manage and not freak out and go do a hookers-on-cocaine binge on that. It's interesting. There's this argument for really relying on whole largely unprocessed foods at least in the beginning of the story.

And then, I think, to your point, if we have somebody who is a hard charging athlete and doing some Pacific Crest Trail, they're backpacking 20 miles a day or what have you, yeah, okay, a bunch of more refined calories is probably going to be just fine. But if you count the number of steps you take in the hundreds per day because you go from your home to your car to your desk and back again then probably chewing your food is going to be really important relative to shooting it down in a shake or Bulletproof coffee.

Hey, folks. Just a reminder. I know that dropped off after I was jabbering but we had some technical difficulties with the recording with Dave. We're going to have him back on in a couple of weeks. I know that that was a massive chunk of material. Pencil down your questions. I'm going to circle back around you folks probably on Facebook, social media, and ask you for your questions related to all of this lipoprotein and cholesterol talk that Dave and I had.

Dave was honestly just getting going. There was a lot of other material that he wants to dig into. So, looking forward to part two with Dave Feldman.

[0:46:48] End of Audio