Superfuel: Ketogenic Keys to Unlock the Secrets of Good Fats, Bad Fats, and Great Health: A Special Interview With Dr. James DiNicolantonio By Dr. Joseph Mercola

JM: Dr. Joseph Mercola **JD:** Dr. James DiNicolantonio

JM: Welcome, everyone. This is Dr. Mercola, helping you take control of your health. Today we are joined by Dr. James Nicolantonio, who I've interviewed in the past for his book *The Salt Fix: Why the Experts Got It All Wrong and How Eating More Might Save Your Life*. He's written a number of other books, but the one we're going to talk about today is the upcoming one that he and I both wrote, which is *Superfuel: Ketogenic Keys to Unlock the Secrets of Good Fats, Bad Fats, and Great Health,* which talks about fats, the importance of them and how to discriminate between the good and bad fats, and what is really healthy fat. Welcome and thank you for joining us today.

JD: Thanks for having me.

JM: You had basically compiled most of the research on this and invited me to contribute to it. I was happy to, especially knowing the work you did with *The Salt Fix*, which was really an important book that helped change many people's views on the dangers of salt or the importance of salt for health. What motivated you to write this book?

JD: This is kind of like ketogenic diet 2.0, right? I mean everyone's doing keto diets now, where their diets are mostly 70 to 80 percent fat. It was kind of like, "How do we enhance that diet? What are the healthy fats? What type of fat should people be eating since it's making up the majority of their calories?"

I think a lot of people doing the ketogenic diet, they're doing some things right, but they're doing a lot of things wrong. This book is really to build off of a diet that is dramatically improving people's lives, but we can actually enhance their lives by selecting good fats versus bad fats. That really was the motivation for this book.

JM: Yeah. It's pretty sad, actually, because many people just simply summarize keto or paleo as being high-fat. While it is, it's really not the full picture. If you choose the wrong fats, you're going to actually run into more problems. If you replace those with healthy carbs, in general, I think there's a lot more danger from eating damaged fats than there is from processed carbohydrates.

JD: Yeah. Absolutely.

JM: The primary reason – Well, you can expand on that. I've got my guesses, but why don't you tell us why?

JD: Yeah. Really, it's almost matching what the fats we eat during Paleolithic times. So really, the three main changes that occurred in our intake in regards to fat was we started consuming industrial vegetable, also known as seed oils, which we never had in our past. Our intake of omega-6 doubled or tripled. Our intake of omega-3s both plant- and marine-based went down at least tenfold. We've had these dramatic changes in our fat intake. They actually changed us from the inside out.

Really, omega-6 isn't bad necessarily. It's when you isolate it and you adulterate it. That makes it extremely bad. We have these bottles of omega-6 now that are on shelves. They're 24/7 exposed to light, and then we cook with them. That oxidizes the oil further. Then we consume these isolated oils. They don't have the natural vitamins and minerals and antioxidants in the coatings around seeds and nuts that gives us omega-6 to protect them from oxidizing in our body.

When you consume these isolated oils, even if it's like a cold-pressed omega-6, the acid in your stomach will still oxidize those oils and create lipid hydro peroxides and aldehydes. We absorb these and they cause a ton of damage.

Really, like you said, the worst thing that's happening to our health is this increase in omega-6. The half-life of omega-6 is anywhere from one to two years, so it stays with you. Really, if you look at our fat stores of omega-6, they've gone from under 10 percent, about 9 percent in our fat tissue to 23 percent in the last 50 years. We're just stockpiling this inflammatory omega-6, which is just absolutely wreaking havoc on our health.

JM: Yeah. But omega-6 and omega-3 are both essential fatty acids – essential, meaning your body can't make them. You need the precursors to build them up and store them and have healthy biological function. The major danger from having the wrong oils, from my understanding, is that, as opposed to carbohydrates –

Carbohydrates are typically burned as fuel or stored as glycogen and burned as fuel shortly thereafter. I mean if you get it really in excess, it's going to be stored as adipose tissue. But it's not damaged. It basically takes the precursor and builds healthy fat. But when you're eating damaged fats, like most people do, then it's integrated in your cell membranes. Those cells stick around for a long time, some not for so long, but most of them are going to be around for a while. And then your cell membranes, if they're dysfunctional, because you're on fats, you're going to run into loads of complications.

JD: Absolutely.

JM: Yeah. The other point I want to mention, because I think the danger that many people have with respect to understanding this concept is that they say, "Well, I'm not buying that vegetable oil on the grocery store shelf. There's none of that in my house, in fact." But what they don't understand – and this is the key – is that there are stealth omega-6s.

I want to talk about that. Because unless you're the rare person who just simply never eats out, you're going to be exposed to this. That's why you have to be so ultra-careful when you're eating out, because that's where you're going to get loaded with it. It's just all – Of course there are restaurants that don't do this, but almost all of them do. I'd say, it's well over 95 percent who are

using these cheap vegetable oils that they use to cook with and make their food. Why don't you comment on that and the other issues that you think contribute to it?

JD: Yeah. No. I mean that's a great point. Most of these restaurants are cooking their vegetables and meats in these omega-6 vegetable oils. Even oils that we consider healthy, like canola oil, when you cook with that – I mean canola's like one of the worst oils you can cook with – it causes a ton of oxidation products, because they're so susceptible to heat, because of the double bonds. As you said, you're getting these in vegetable oils. I mean they put soybean oil in these omega-6 seed oils, in bread and in condiments. It's really everywhere. Cereals –

JM: Pastries, the desserts. When they show you the dessert menu, you can always be guaranteed, unless it's a fruit plate or something, there's going to be processed omega-6 in that dessert.

JD: Exactly. Like you said, it's literally transforming you from the inside-out, because these longchain omegas, they get integrated in the cell membrane. The oxidation products will oxidize those tails, those fatty acid tails. Then what happens is when you damage those tails in the lipid bilayer, they start to curl upwards. That actually creates a more permeable membrane. You get more things that aren't supposed to get into the cell and damage the mitochondria, damage the DNA. The fluidity of the cell membrane goes down.

The fluidity of the cell membrane is extremely important, because you have all these hormone transporters that sit in the cell membrane. When you don't get enough omega-3s, especially docosahexaenoic acid (DHA), the membranes become very rigid. What ends up happening is those membranes start pinching on the transmembranes. That brings in sodium, potassium, amino acids and glucose.

Instead of being able to come in and out very easily, because the membrane isn't fluid, it starts pinching on those membrane transporters. That starts affecting how things start flowing in and out of the cell. Your metabolic rate goes down, and you have damage in the cell. It's a huge issue.

JM: Yeah. To extend that illustration even further, it's not just the cell membranes, but it's the organelles or the functioning structures within the cells. The most important one, of course, being the mitochondria, because the mitochondria is not one but two membranes that are composed of the same lipids and fats. You run into the same problems that you just described. It's not just the cell. It's the organelles within the cell.

JD: Yeah. No. That's a great point. Actually, probably one of the greatest damages that happens is you have this inner mitochondrial membrane. The cardiolipin is supposed to be saturated in DHA. The reason why is because DHA is very susceptible to oxidation. It's actually the alarm system that our cells have to either survive or, if it's turning into a cancer cell, die. If you don't have the cardiolipins saturated with DHA, you can't signal caspase-3, and you can't signal apoptosis.

Literally not having your cell membranes and your cardiolipins saturated with DHA can increase, potentially, your risk of a cell from going from healthy and being damaged and flipping to apoptosis, versus a cell that can't do that anymore if you don't have enough DHA. It's very

important to saturate the cardiolipin or the inner mitochondrial membrane with DHA to program cell death if necessary.

JM: Yes, indeed. Can you describe in further detail the caspase-3 that you mentioned? Because many people don't know what that is.

JD: Yeah. It's basically the signal that cells use to trigger the self-destruction or self-kill mechanism. The cell has to have something to say, "Wait a second, damage is occurring, and it's gone to a point where we need to just get rid of the cell before it converts to a malignant cell."

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The signal is DHA, because it's something that our body has evolved off of for thousands or millions of years. It knows to utilize that DHA as the signal to either – as damage is going on – need to upregulate antioxidant enzymes or, "Too much. I need to kill this cell, so it doesn't turn into a malignant one."

In the brain, actually, we utilize DHA as well, as a signal to stimulate NRF2, stimulate heme oxygenase 1 and upregulate antioxidant enzymes. Omega-3s oxidizing in the body is bad, but our body actually kind of knows what to do with that signal, whereas omega-6, not so much, because we didn't have as much during Paleolithic times.

JM: Yeah. That's definitely one of the issues, no question about it. But there are other signals for apoptosis, such as mammalian target of rapamycin (mTOR) inhibition and also fasting, which will stimulate a cascade of metabolic events that actually catalyze apoptosis, appropriate apoptosis, and the senescent cells. The senescent cell is the cell that's defined as one that's aged and crippled or damaged and has lost the ability to reproduce. It essentially becomes useless.

Actually, it's worse than useless, because it clogs up the machinery. Unless you get rid of that senescent cell, you're going to run into problems. It's gunk in the machinery. It's just clogging up your system. You need a mechanism to remove those. A lot of people believe that's one of the keys to addressing some of the major hurdles of slowing down the aging process, are these senescent cells.

A big issue is lowering of omega-6, so these damaged omega-6s. Because as I said earlier, omega-6 and omega-3 are both essential. Your body needs them. It's really difficult to become deficient in omega-6, because of the abundance in our food supply. But you can, and I can think of people who are on intravenous (IV) nutrition because they're in a coma and they just cannot eat or they're on parenteral nutrition, essentially through IV or through oral (PO). Those individuals can run into omega-6 deficiency if it's not put into the feedings that they're getting.

JD: Yeah. I mean, you can. But the thing, like you said, is it's extremely difficult to be deficient in omega-6.

JM: Yeah.

JD: Really, the estimates right now would be most people living with 2 percent of omega-6. But really, when you look at the older studies, it really seems to be only about 0.5 percent. That's all you need. As long as you're getting sufficient omega-3, you may only need 1 or 2 grams of linoleic acid per day.

It blows my mind that the American Heart Association (AHA) and the dietary guidelines are recommending that we consume at least 5 to 10 percent of our calories as omega-6. That's coming from either vegetable oils or olive oil. Instead of actually recommending whole foods, they recommend consuming them via oils, which makes absolutely no sense.

JM: Yeah.

JD: When you have direct studies contradicting that you need 5 to 10 percent of your linoleic acid. If you look at the Lyon Diet Heart Study, it actually lowered linoleic acid from over 5 percent to about 3 and 1/2 percent. There was a 70 percent reduction in cardiovascular runs and mortality by lowering linoleic acid. There's actually no evidence to support the AHA and their dietary guideline: recommending or consuming high amounts of omega-6s from vegetable oils.

JM: Let's just back up here too. Linoleic acid – because it gets really confusing there – It actually also has the acronym ALA, alpha-linoleic acid, and then there's alpha-linolenic acid. Linoleic acid, I believe, is the omega-6, and linolenic acid is the omega-3.

JD: Right. It's commonly confused. I just wanted to make that clear. If you're confused, don't be surprised, because it's common. But anyway, you referenced the AHA, the American Heart Association. You shouldn't be surprised. Maybe we need to do a cognoscopy on you, because you're surprised. Because this is the same organization that last year, in 2017, said coconut oil was dangerous.

Now, of course, one of their disciples, a German researcher from Harvard, is saying that coconut oil is pure poison. But it really stemmed from the letter they sent out, the AHA sent out, to cardiologists last year, warning about the dangers of excessive coconut oil. I'm not sure what drove that, other than – maybe you have some other insights on this – just to concern, not to appear that their 30 to 40 years of recommendations were fatally flawed would be one of my biggest theories as to why they persist in this fantasy.

JD: Well, really, the AHA, they were no one until Procter & Gamble came along and gave them basically a 1.75-million dollar donation. Of course, Procter & Gamble were the ones who created Crisco, which is crystallized cottonseed oil, which is -

JM: That's a century ago now.

JD: That was 1911, over a century ago. The story is an interesting one and how that all came to be. We can go into that if you want.

JM: Yeah. I think that's interesting. Elaborate.

JD: Yeah. The downhill of our health really began with the invention of the cotton gin. In the United States, we were only able to produce about 600 pounds of cotton, and then along came the cotton gin. Within less than a decade, we were, in the late 1700s in the United States, we were producing 40 million pounds of cotton. Of course, for every 100 pounds of cotton, you get 160 pounds of cottonseed. You only need 5 percent of those seeds to plant the new crop. And so you have all this cottonseeds and you have nothing to do with it.

Procter & Gamble came along. They started producing candles and soaps, and started lighting homes with cottonseed oil. What happened is the oil industry came along in the mid-1800s and there goes the application of cottonseed oil for lighting homes. And so, you have all this cottonseed oil and you have no use for it.

A German chemist came along in the early 1900s. He discovered that if you took vegetable oils or a liquid oil, you added a catalyst, you added heat and you removed hydrogen, you could convert a liquid oil to a solid. He invented partial hydrogenation. I believe it was 1909 when he may have done that. This was Wilhelm Norman, who was a German chemist. Procter & Gamble bought his patent to partial hydrogenation in 1909.

They opened their own lab and created Crisco in 1911. Back then, you could advertise whatever you wanted. You could make Crisco – "You'll live longer. Your teeth with glow. Your skin will shine. Your kids will have no issues." No one monitored all of these things.

Once they had that, they created an entire recipe book. They were giving them all out for free. And, of course, all the recipes contained Crisco. They were making all these claims that say, "Mothers, they cook better cakes and pastries with Crisco," "Digestion improves," and all that.

It went from nothing, consuming absolutely nothing, until 1916 – in just one year – they sold 60 million pounds of Crisco, way back 100 years ago. That is a lot of Crisco. That was over 100 years ago. It just got integrated. And then Procter & Gamble, they did a radio show and gathered 1.75 million dollars, gave it to the AHA and launched them into the leading cardiovascular society that we know today. You're still kind of reading that bias.

JM: What year was that?

JD: I think it was –

JM: Around early 1900s?

JD: 1960s, I think they did that.

JM: Because that's not a lot of money really. It's 1.7 million, when you consider all the craziness that's being done. Literally, the billions, literally billions, with a B, of dollars in fines that many companies are getting now, drug companies included, for their blatant violations in harming humans and causing enormous suffering. Yeah. I mean it's a lot of money for some people, but for an organization to give to another organization, it's a relatively minor amount.

But they've been doing this for over a century. It was promoted largely because of its benefits. I don't think – Refrigeration back then was a challenge. I think that many people had iceboxes, but a lot of people didn't have electric refrigerators. I'm not even sure when that became more popular, but certainly not the early 1900s.

JD: Yeah.

JM: You didn't have to refrigerate this stuff, so that made it a lot more convenient.

JD: Right.

JM: Yeah. And it had a long shelf life.

JD: It was a solid fat. People are used to cooking with lard and tallow. Now, they have this weapon that they could say, "This could replace that." Really, you had the pressure of the Great Depression. You had World Wars going on. Really, actually, they were using glycerol to make bombs.

Actually, people – I think it was for – Man, I can't remember now, but it was one of the World Wars. I think it was World War II. They were being asked to give their animal fats for the glycerol to be used for making bombs. They were almost forced into consuming these vegetable oils because you could cook with them at the fraction of the cost. And then they were being marginalized, these animal fats, to go to the army. Really, it was almost forcing Americans in the '30s and '40s to switch over to vegetable oils.

JM: Well, glycerol, for those who aren't aware of it, is the backbone of triglycerides. It's where all the fatty acids attach to. But I'm curious why couldn't they just get the glycerol from the vegetable oil? Instead of getting them from animal fats. Was there an industrial process to do that?

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JD: Yeah. I don't know. I'm not sure. But I do know they were rationing animal fats, and at the same time, it was becoming extremely expensive compared to these cheap vegetable oils.

JM: When Procter & Gamble first made Crisco, was the fat that they used primarily cottonseed?

JD: It was cottonseed oil. Yup.

JM: Why don't you update us? Because to the best of my knowledge, cottonseed oil is not classified for human consumption, is it?

JD: Yes.

JM: I mean it's not a human consumption oil.

JD: I think they do use a little bit of cottonseed oil now. But like you're saying, it's dramatically gone down. It's really soybean oil that they use nowadays.

JM: Yeah. But I don't think for health reasons they didn't promote it. I mean I thought it was not recommended or advised. I know it's being used –

JD: I see what you're saying.

JM: But it's not a food product. It's an industrial crop. It's not designed for food. Cottonseeds are not food products.

JD: Correct. You can't squeeze a cottonseed and get oil out of it. It takes tremendous machinery, multi-million-dollar machinery, heat hexane. This was really part of the problem with these industrial seed oils. It's the processing that occurs to get them out of the seed. They have to deodorize the oil, because it's so toxic. By the time it makes it to the shelf, you've got all these oxidation products, and then you consume it and your body oxidizes it with the acid in your stomach.

These oxidation products are about a thousand-fold higher than the eicosanoids in your body. They are dramatically more harmful than any of the most harmful eicosanoids, like thromboxane a2, that you could ever think of, because they form aldehydes. These aldehydes, like 4-hydroxynoninole, which is formed off a linoleic acid, this is what actually causes oxidized low-density lipoprotein (LDL). It actually binds to the apolipoprotein B (apo B), all apo B-containing lipoproteins.

Now, these lipoproteins aren't recognized by the LDL receptors. They hang out in the blood. It's really the linoleic acid that gets integrated into high-density lipoprotein (HDL), LDL and very low-density lipoprotein (VLDL) that oxidizes and causes atherosclerosis.

I mean, linoleic acid itself can actually damage the endothelium and cause an increase in penetration of LDL and VLDL particles into the subendothelium. That's not even oxidized linoleic acid. That's just linoleic acid itself is toxic to the endothelium. And then when you get these oxidation products, it's dramatically more harmful. This is what's causing neurodegenerative diseases.

I mean these aldehydes can actually crosslink proteins, crosslink tau protein and create neurofibrillary tangles. This has been shown in animal studies, that these aldehydes can literally create neurofibrillary tangles that you see in Alzheimer's disease. This is what's called advanced lipoxidation end-products, ALEs. Most people know about advanced glycation end-products, but these advanced lipoxidation end-products, by far, are much more harmful. It's dramatically caused by these consumption of oxidized seed oils.

JM: Yeah. I might recommend that if you're a little bit confused with what was just said is to replay this, because that was really an important summary of the two most common diseases that affect us in our society, which is heart disease and neurodegenerative diseases, primarily Alzheimer's. Largely related to this excess consumption of damaged omega-6, linoleic acid.

JD: Correct.

JM: Linoleic. Yeah. I still get confused with those things. But anyway, it's linoleic. Too much of that is going to be counterproductive through the mechanisms that you just cited. You consolidated a lot of information in those few paragraphs, but that was a good summary. What's your best speculation as to why so-called experts in the AHA and people who promote their work don't understand or accept this? I mean, how are they so confused?

JD: Well, one is they hang their hats on LDL. Ansel Keys really showed that these vegetable oils can lower LDL and saturated fats raise LDL. That is still an issue, because it's very difficult for any scientist to come out and say that high LDL is fine and lowering LDL is not good. They obviously believe that high LDL is bad.

We do know that vegetable oils lower LDL, but they increase the susceptibility of LDL to oxidize. That's really what they need to understand. Don't worry about the number or what's actually happening to the LDL.

The second thing that has been confusing, prior to Dr. Chris Ramsden coming out with his 2010 meta-analysis and his 2013 meta-analysis, is all studies combined the omega-3 and omega-6 and showed that there wasn't benefit if you swapped out animal fats and you started consuming more omega fats. They never separated those studies that only gave and swapped out omega-6 with animal fats, and omega-3s. Chris Ramsden was the first scientist to do that.

When he separated the studies, he actually found that the studies where animal fats were replaced by these vegetable oils, there was an increase in all-cause mortality, coronary heart disease mortality and cardiovascular events. He was the first one to ever do that. That was as recently as 2010. He did an update. He looked at the Sydney Diet Heart Study. He updated it. He found additional evidence from that study, updated his meta-analysis and found the same thing.

And then you have the Minnesota Coronary Survey, which was hidden in a basement for 30 years. That was published just a few years ago. It really showed that those who actually had the greatest reduction in LDL were at the highest risk of heart disease from consuming these omega-6s. Actually, the autopsy study showed that the patients who switched from consuming animal fats to the vegetable oils, the autopsy study showed a significant increase in heart attacks. That was just a couple of years ago that study came out.

That's why, I think, it's so difficult for people, scientists and the lay public to understand. It's because evidence was buried. It's just starting to surface. There was kind of poor science being published, combining omega-3s and omega-6s and confusing the heck out of people. Now, you have all these studies underdosing omega-3s and saying omega-3s aren't beneficial anymore when they're still giving them to people who are consuming 20 to 30 grams of linoleic acid, and they're only giving 1 gram of omega-3 fish oil and thinking that's actually going to be beneficial.

They're forgetting all the old studies that actually tested Italians or Japanese who had a low intake of omega-6. Every study that has looked at omega-6 or omega-3 fish oils on a background of low omega-6 has always found dramatic benefit. We know the omega-3 index is super important and we know that getting to 8 to 10 percent on an omega-3 index reduces your risk of sudden cardiac death by 90 percent. There's a confusion. People are worried about omega-3s raising LDLs. It's

really this whole confusion of what's really happened with omega-3 and omega-6 and what's really beneficial.

JM: Yeah. A good point that's frequently overlooked is that when these studies looked at omega-3 supplementation, they don't look at the totality and see what the ratios are. But even if they did, you can still have the same problem with omega-3s that you have with omega-6. We go into that in the book. Because there are a lot of omega-3 products out there that are very similar to the industrially processed omega-6 vegetable oils that you grab off a shelf. But they do cost essentially the same problems, because they're processed almost identically. Why don't you go into that a bit?

JD: Yes. About 50 percent of the fish oil in the market has problems with oxidation. From harvesting and getting it into the pill and then what happens during when you store it, in the shelf life. Like you said, omega-3s and omega-6s are both very susceptible to oxidation. Really, you've got to get a product that tests the hydro peroxide levels. There's a certain – I don't think you want more than 5 percent. But the lower, the better, obviously.

A good, quality supplement will actually show you the testing of the hydro peroxides in the supplement. A lot of these fish oils aren't coming from wild fish. That's an important thing too. It's that you really want to supplement that from a wild-sourced fish, particularly from a fish from an ocean or waters that are clean, like Alaska or Canada, is another important part. But honestly, very few supplements source from a good source and use wild seafood to do it.

JM: Yeah. A very good point, because if you're getting farmed fish, they're frequently not giving them fish food that has been essentially raised on algae and other primary sources of food in the ocean, which concentrate the DHA and the eicosapentaenoic acid (EPA). If you're eating those types of fish, you won't have the omega-3 content that you need. But even more importantly, I think, is the issue of sustainability. We've got over 7 billion people on the planet. Every human needs omega-3. There really isn't enough fish in the ocean to supply them with it. There just isn't. We have to figure out some other resources. That's one of the reasons I like krill oil, because it is the largest biomass in the world.

JD: Yup.

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JM: You have to look at sustainability. I think it's The Convention on the Conservation of Antarctic Marine Living Resources (CCAMLR), and then there's another organization that escapes me right now, that monitors that very carefully to make sure that the krill isn't harvested more than they need to be for sustainability issues and to sustain the whale population too, because that's their source of food.

JD: Yeah. I think they're only allowed to take like 1 percent or less per year. That biomass is, like you said, the largest biomass in the world. Krill is great, because it has so many advantages compared to just regular fish oil, because the omega-3s are bound to phospholipids.

How we used to get our omega-3s back in Paleolithic times – Even inland, thousands of miles away from the ocean, the only animal that could actually obtain the brain and can break the skull besides a human is a hyena. We were scavengers. Sites were discovered over 2 million years ago, human sites with dozens of animal skulls cracked open around them. The brain is higher in DHA than salmon, so up to 30 percent more concentrated. Our ancient ancestors were able to access and scavenge skulls in African savanna and get tremendous amounts of DHA.

To give you an example, 4 ounces of brain can give you up to 1.5 grams of DHA. It's extremely saturated in cholesterol as well. This was a phospholipid-bound DHA that we were getting through this. The absorption, your brain doesn't absorb DHA without it being bound to phosphatidylcholine. This is lysophosphatidylcholine that the DHA is bound to. When you're consuming fish oil, you've got to esterify it. You've got to attach it to choline, and then you try to absorb it. But with krill oil, being bound to the phospholipids, you get two times the absorption in the brain of DHA.

JM: And other tissues too.

JD: What's that?

JM: And your other tissues.

JD: Yes. Exactly. Particularly the brain though. Krill oil has astaxanthin and cantaxanthin, which are extremely potent carotenoids. Astaxanthin can actually expand the entire cellular membrane. You can prevent oxidation from the inside, as well as the outside of the cell. It has vitamin A, vitamin E in it. I mean compared to Coenzyme Q10 (CoQ10), astaxanthin is just incredibly powerful on the Oxygen Radical Absorbance Capacity (ORAC) level, on squelching singlet oxygen. It puts CoQ10 to shame. It's like dramatically better than that.

JM: But they serve different purposes.

JD: They serve different purposes, yeah.

JM: CoQ10 has its role in the mitochondrial electron transport chain.

JD: No doubt.

JM: Astaxanthin has other roles too.

JD: Yup. And you get a good dose of choline from krill. I mean 1 gram of krill, you can get 75 milligrams of choline. It's super important for preventing fatty liver and things like that. We're used to getting our omega-3s bound to phospholipids. We're getting them through brain consumption a couple of million years ago. Really, too, how we used to get our -

JM: Those are people who lived inland. If they were living on a coast, they would get seafood.

JD: Correct. They were getting seafood. And then, also, our consumption of ALA was 10 times what it was today. How we used to get omega-3s if we weren't getting brain, we weren't getting seafood, is we were consuming a tremendous amount of plant material. We were getting 10 to 15 grams of ALA. We only get about 2 grams nowadays. If you look at a female, who is of childbearing age, they could convert over 20 percent of their ALA to EPA.

JM: The ALA being the alpha-linolenic acid, which is the omega-3.

JD: Yup. That's the parent omega-3 in plants. Yeah. Women of childbearing age can convert over 20 percent of that ALA, and they were getting 10 to 15 grams to EPA. They were getting 2 to 3 grams of EPA just from converting all of that ALA. They can actually convert almost 10 percent of that ALA to DHA, which is a long-chain marine omega-3. They were getting 1 to 1 and 1/2 grams of DHA just from the conversion.

As a fetus, you're getting all of that EPA and DHA through the ALA intake of the mother. And then back then we breastfed for up to 4 years. DHA gets into the breastmilk. That was a tremendous source. Nowadays, DHA in breastmilk is not even close to what it needs to be. To optimize a baby's DHA level, it should be anywhere from 0.8 to 1 percent DHA in breastmilk. Nowadays, the breastmilk only contains 0.3 to 0.6 percent, so about three times less than what it needs to be to optimize DHA in the fetus.

JM: Those are some interesting numbers on the conversion from the ALA to higher-chained omega-3 fats, EPA and DHA. Twenty percent for a pregnant woman, but that's not typical. What does a typical adult, non-pregnant adult – Would that conversion be a lot lower?

JD: It's a lot lower.

JM: It's a magnitude lower.

JD: Yup. For most people, you're only going to convert about 5 percent ALA to EPA and 0.5 percent from ALA to DHA. There are so many things now that are happening to us that we don't even realize as reducing the conversion. When you go from 15 grams of linoleic acid to 30 grams, which we're consuming nowadays, that reduces your conversion rate of ALA to EPA and DHA by 40 to 50 percent.

JM: Wow. So you knocked that 0.5 down to 0.25.

JD: Exactly. You also are required – Vitamins and minerals are required for these desaturase enzymes. There's a delta 6 desaturase that takes linoleic acid and ALA and starts desaturizing them and elongating them. There are enzymes that do this to bring them to the longer-chain omega-3 and omega-6 fats. They all require minerals and vitamins.

About 30 percent of the general population is magnesium-deficient. That is required for the first step in converting ALA and linoleic acid to a longer-chain omega-3. Delta-6 desaturase requires magnesium. And then, really, we're missing these healthy omega-6 conversions. gamma-Linolenic acid (GLA), which is found in borage oil, blackcurrant, evening primrose, those oils are very high

in what's called gamma-Linoleic acid. That is the elongation product of linoleic acid, so it's in omega-6, but it's healthy omega-6. There have been many studies showing that giving GLA or evening primrose, blackcurrant or borage oils to rheumatoid arthritis dramatically improves inflammation.

The reason is because GLA turns into dihomo-gamma-linoleic acid, DGLA. Off of that omega-6, you have a very healthy prostaglandin E1, which vasodilates and has anti-platelet effects and antiatherosclerotic effects. That conversion of DGLA to prostaglandin E1, which is very healthy and beneficial for us, requires vitamin C. It requires zinc. It requires calcium. The lack of vitamin C in our diets is dramatically reducing the conversion of DGLA to this beneficial prostaglandin E1.

This is at the heart of atopic dermatitis, eczema, rheumatoid arthritis, attention deficit and hyperactivity disorder (ADHD) and things like that. Our conversions, our healthy omega-6s and the prostaglandins that are formed from them is part of, you know, the fat fix and the superfuel and all those good things that you need to do that we're suffering from.

JM: Yeah. And another factor – You had mentioned 30 percent were magnesium-deficient. It's probably closer to 80 percent of the population that's magnesium-deficient. But a big one that also 80 percent of the population has is insulin resistance. That will clearly slow down and impair delta-6 desaturase from making that conversion.

JD: Yup. Exactly. Both desaturase enzymes are insulin-sensitive. Delta-5 desaturase too goes down when you're insulin resistant. When you have higher insulin levels, you actually form more arachidonic acid. You shunt DGLA instead of going to the healthy prostaglandin. The higher insulin level shunts it into more arachidonic acid. Really, arachidonic acid isn't bad. You can form -

JM: You need it. Yeah. If you have low levels, you have a problem.

JD: Exactly. It's the overall inflammation caused by linoleic acid that upregulates these cyclooxygenase and lipoxygenase that attack arachidonic acid and form all these harmful biochemical from them, which are called eicosanoids, like the 2- and 4-series eicosanoids, thromboxane A2, which are blood clots, and you have leukotriene B4, which is asthma and allergies and all those things.

JM: At the heart of all this is this shift in the fuel supply or development of the prostaglandins, which you have been referencing, which ultimately results in increased inflammation in the body.

JD: Yup.

JM: There's a simple test that you can take to see if you're at risk for this. It's called the highsensitivity C-reactive protein, or hs-CRP, for short. It's not that expensive. It's a test that I recommend for almost everyone. You'll be surprised with what your numbers are because odds are, unless you're super diligent, it's going to be 1.0. It might be 5, 6, 10 or even higher. But ideally, it should be below 0.5 and even lower if you can. JD: Yeah.

JM: If it's not, then you know that you've made some poor choices, either knowingly or unknowingly in your food, or you've got other issues going on too, because it's not just the fat that causes it. But that's the leading contributor, and I think the one with most people are confused about.

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JD: Yeah. You're absolutely right. hs-CRP, or high-sensitivity C-reactive protein, is kind of like a marker of chronic inflammation. You need to stop inflammation, right?

JM: Yeah.

JD: If you caught an infection, no doubt. But our body has a mechanism. You have to be able to resolve the inflammation. The way our body does that is through omega-3s. There are these resolvins and protectins. They're called SPMs, specialized pro-resolving mediators, that are formed off of omega-3s.

JM: They're relatively new, so if you haven't heard of these things, don't be disappointed, because these have only been known for a few years.

JD: Yup. Exactly. This is the body's way of actually resolving the important acute inflammation that you need to fight an infection. When you don't have enough – when your cells aren't saturated with omega-3s, you don't have the ability to suppress that inflammation. So you have chronic inflammation that drives a lot of the chronic disease.

You don't know if you have inflamed fat cells. You don't know. You just don't. That's the really harmful fat that's killing you. It's the ectopic. It's the fat that goes around the liver, the pancreas and the heart. If you have inflamed fat – and omega-3-deficient fat cells are inflamed fat cells – just bottom line, if you don't have them, you can't form the resolvins and protectins in the fat cell.

Omega-3s are really cool too because they can convert the harmful macrophages, Type 1. They can shift those to Type 2 anti-inflammatory macrophages. That's the problem that we have nowadays with "thin on the outside, fat on the inside." It's this inflamed fat, where these macrophages are coming in. They're turning inflammatory, and then your fat cells are releasing inflammatory cytokines, causing systemic inflammation.

If you don't have a high amount of omega-3s, like in Japan, where they have an omega-3 index of 10 to 12 percent, which probably is what we should be at, but it takes 4 grams of EPA and DHA, at least, to get to that level. You probably need 6 grams on a higher background of omega-6, like we're consuming.

This omega-3 deficiency is affecting well over 80 percent of the population. Very few people are getting 3 to 4 grams. That's really what you need to get the blood-pressure-lowering effects of omega-3s, the reduction of triglycerides, the antiplatelet effects, the plaque-stabilizing effects. You

don't get those at 1 or 2 grams. You've got to go 3 to 4 grams of EPA and DHA. That's why all these recent studies have been failing, as they're only giving 1 gram at most of EPA and DHA.

JM: Yeah. That's a good point. One of the reasons we collaborated on writing this book is that it's such a good addition to the *Fat for Fuel* book, which is my last book. Obviously, its focus is to helping your body relearn how to burn fat as your primary fuel, because most of the people in the United States have lost that ability. You're pretty much only burning carbohydrates, which is loaded with negative adverse health consequences.

I think you laid a pretty good argument for minimizing industrial-processed omega-6, but we do need some. As we mentioned, it's not hard to get. But I like my primary sources of omega-6 to be whole foods, I mean really whole foods, like seeds. I mean you can get it in vegetables. There's not a lot though. Seeds have a far higher concentration. I take a tablespoon of a whole variety of seeds. There are some seeds that don't have a lot of omega-6, like flax seeds, chia seeds and hemp seeds. But almost all the other plant seeds do have omega-6. That's one of my favorites. What are some of yours for the omega-6?

JD: Yeah. No. I mean seeds, like you said, absolutely.

JM: Nuts too.

JD: Yeah. Seeds and nuts.

JM: It depends on the nut. Walnuts may be higher in omega-3, but there's still plenty of omega-6.

JD: There's still plenty of omega-6. Yup. You're right. If you're getting omega-6, you're going to want it in a whole-food package, with vitamins, minerals and -

JM: The whole view, without the whole-processed oil.

JD: Yeah. In regards to the ketogenic diet, most people don't understand how omega-3s, how important they are to building muscle, improving exercise and burning fat. To give you an example, if you replaced just 6 grams of visible fat in your diet, let's say on this fatty steak, and you take 6 grams of fish oil, one study showed just in three weeks they lost 2 pounds of fat and gained half a pound of muscle.

The reason is because omega-3s, particularly DHA, is the pacemaker of the cell. The reason why hummingbirds can beat their wings 80 times a second is because they can saturate their wings with DHA. DHA makes the cell membrane so fluid that molecules, like amino acids, glucose, sodium, potassium, they fly in and out of the cell. The same thing happens in humans. When you consume a high amount of omega-3s, about 3 to 4 grams, as what's needed, you create a cell membrane that is super saturated DHA, very fluid.

Now, your basal metabolic rate goes up 15 percent. Your beta-oxidation in the liver during exercise, your fat burning during exercise goes up by 30 percent, consuming high amounts of omega-3s. Even at rest, your beta-oxidation goes up by 20 percent.

Long-chain omega-3s are important for ketogenic diets, because you become a better fat-burning machine. It's affecting the machinery, the beta-oxidation in the liver. It's improving that by activating genes. And then the other omega-3, the plant omega-3, alpha-linolenic acid, is a ketogenic substrate, so it doesn't get stored like the marine omega-3s. It can be converted into ketones. Medium-chain triglycerides (MCT) oil is great too, but a lot of people don't realize plant omega-3 kind of serves a little bit similarly in that way.

JM: It's a much bigger molecule.

JD: Yeah.

JM: I mean it's 18 carbons, whereas MCTs are 8 and 10 carbons.

JD: Exactly. Yup. MCTs are great too for fat loss. Meta-analyses or randomized studies show that MCTs, compared to long-chain saturated fats – we're talking about heavy cream and butter – it does significantly reduce waist circumference and visceral adiposity, because it doesn't get stored. It gets burned for ketones.

JM: Yeah.

JD: The reason why omega-3s are good for fuel is because it suppresses inflammation in the brain. What happens in a cognitive-declining brain is you're not able to utilize glucose well, because of the inflammation. DHA helps squelch the inflammation. Your brain is able to utilize glucose better when you're consuming more omega-3s. You're actually able to produce more ketone bodies when you're consuming both parent omega-3 and the long-chain EPA and DHA. You become a better ketogenic machine, in a way, when you're consuming high amounts of omega-3s.

JM: What's the mechanism there?

JD: It upregulates genes that activate beta-oxidation in the liver, so you burn fat better with omega-3s. Your basal metabolic rate goes up, because the cell membrane is so fluid. Your ability to get amino acids and glucose in and out of the cell are better. Your inflammation goes down, so you become a better fat-burning machine.

Actually, omega-3s help synthesize protein better too. Muscle protein synthesis dramatically goes up when you consume 3 to 4 grams of omega-3s, because amino acids, again, are flying in and out of the cell way faster when they're saturated with DHA.

Studies have shown in middle-aged adults, as well as in the elderly, consuming 3 grams of DHA increases muscle strength, increases your maximum amount that you're able to rep. Your grip strength is improved. This is an important fat to help prevent sarcopenia. This is a very big issue, where elderly people are not even able to carry a milk carton throughout the grocery store. Really,

the omega-3s are what's going to hopefully help prevent a lot of the sarcopenia that's happening or muscle lost during aging.

JM: Yeah. That's a major issue when you're frail. That's one of the things that if you're in your '60s or '70s that you really want to – Actually, you should do it in your 40s and 50s. It's to focus on it, as you really want to avoid being frail. The loss of muscle will be probably the primary contributor, and then you lose your mobility too. That's a 1-2 bad combination.

Where do we get these omega-6s? We've already talked about the concern about many of the fish oils and other sources of omega-3 supplements with the krill. But there are other sources too. There are, of course, people who are vegetarians or vegans. They will refuse to have fish or fish sources of it, so they're really stuck with essentially algae sources. I'm wondering if you can comment about that, especially the distribution of the EPA and DHA and how that might be problematic.

JD: Yes. You're looking for sources of -

JM: Yeah. Non-fish sources of omega-3.

JD: Omega-3?

JM: Right.

JD: Okay. You kind of mentioned chia, hemp and flaxseeds are all very high in omega-3.

JM: Yeah, well. Okay. I'm sorry. Of course from the plants. I was actually more referring to the EPA and the DHA. Because, as you mentioned earlier, unless you're a pregnant woman, you're only going to be converting 0.5 percent of that ALA to DHA.

JD: Yeah.

JM: We need a lot more of that. You have to have a load of omega-3, of ALA, to produce therapeutic levels.

JD: Yeah. That's a good point. If you're not eating seafood, how do you get EPA and DHA?

JM: Right. Yes.

JD: It's difficult. You can get it from algal oil, right? From algae. That's probably your best way to get it, to be honest.

JM: Does that have EPA and DHA in it?

JD: Yeah. I think it's higher in EPA. I think most algae have like a 2:1 ratio of EPA to DHA. But, of course, it depends on the algae.

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JM: Because you definitely need to have some EPA. Even though DHA is probably the more important one, it's like anything in life, if you don't get the right balance, you're going to be running into complications. You do need EPA too.

JD: Yeah. Absolutely.

JM: [inaudible 50:13] trade DHA. That's a mistake. I think there was a company – Martek. I think they had cornered the market on supplementing infant formula with their DHA. It was just DHA. There was no EPA. And there still may be. I haven't looked at it for a number of years now, but it's a big issue for infant formula.

JD: Yeah. I'd like to think of EPA as the circulating omega-3 in the bloodstream more, whereas DHA is more stored in the heart and the brain. That's kind of like how I'd like to view both of them. Reducing inflammation out in the systemic circulation is more EPA's role in the red blood cells and things like that. DHA is more brain-heart.

If your cells aren't saturated with DHA, especially in the cardiomyocyte, you're going to have a significant problem, because the anti-platelet effects of omega-3s are extremely important. EPA serves different roles. The DHA, like you said, is a little bit more beneficial in regards to lipids. That's what lowers triglycerides and creates a more buoyant LDL. EPA does have some of those effects, but not nearly as much as DHA. But it's important to give both. They both serve different roles.

JM: What's always amazed me when I was in practice – and I would see it frequently – is people come in with elevated fasting triglycerides, I mean 500, 1,000 or 2,000. You could tell because when you spin the blood down, the serum, which is normally clear, translucent yellow, was cloudy. It looked like milk.

What most of those people needed was just to restrict carbohydrates, because they were absolutely insulin-resistant. When you remove carbohydrates from their diet, it will improve the insulin sensitivity, and the triglycerides will improve dramatically. That's a really important risk factor for heart disease, these high triglycerides.

JD: Right. Because it really is just a marker of insulin resistance, to be honest.

JM: Yeah. Alright. Any other highlights from the book that you want to review?

JD: I think we did a good job.

JM: Alright. Yeah. It is a good book. The book comes out on November 13th. If you found this conversation interesting, please get the book. Because there are a lot more details in it. It will really help you more carefully and clearly understand your choice of these really vital, important nutrients, and make sure you're choosing the right ones and avoiding the ones that are going to cause harm and damage.

Alright. Thank you for joining us. We look forward to you picking up the book and exploring it even further.

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