The Carnivore Code: A Special Interview With Dr. Paul Saladino

By Dr. Joseph Mercola

JM: Dr. Joseph Mercola
PS: Dr. Paul Saladino

JM: Well, welcome everyone. This is Dr. Mercola helping you take control of your health. And we are in for a real treat today, at least from my perspective, because we're going to be busting some long-held nutritional myths. And my guest today is Paul Saladino, who is a physician. And really, I view him as one of the leading experts in the importance of having animal food in your diet. And he takes it to an extreme and is really a strong advocate of the carnivore diet to the exclusion of all vegetables or plants materials and that's certainly debatable. But he has some pretty compelling evidence. And I don't know anyone personally who has reviewed the literature more carefully and can put together a coherent argument. It kind of reminds me in the past of the low fat theory that Paul ... predated Paul a bit. I mean, he was part of it, but I mean, most of us that were growing up in the 60s and 70s, I mean, we just ate this.

JM: Everyone bought it hook, line and sinker. And I feel [inaudible 00:01:04] a similar way we've adopted this plants are beneficial for us. So, he's going to really challenge your beliefs and I want to apologize beforehand. This is not meant to offend anyone, if someone believes that they should, for ethical reasons, and that's certainly their choice. That they should not have animal material or animals in their diet. You can certainly choose to do that, and I'm not chastising you for that. It's just that you just need to be aware of the biological consequences of that choice. So, well, welcome and I'm so excited that we're here to discuss your new book which is-

PS: Thanks for ... Yeah. Thanks for having me on.

JM: [crosstalk 00:01:45]. It's the Carnivore Diet is the name of the book?

PS: The Carnivore Code.

JM: Carnivore Code. Okay. Sorry.

PS: The Carnivore Code. Yeah. Thanks for having me on Dr. Mercola. It's great to be here.

JM: Yeah. Yeah. Well, boy, man, it's just so incredible. There's so much information to pack. It's going to be a little bit longer one, because I just want to talk about so much. So, you really ... you state in the book that you're not talking about ... You're going to really bust some nutritional dogma. And I think you do a really good job of that. Let's state [inaudible 00:02:15] precedent, and I want you to give your brief history, because I don't want to go into too much details. There's so much content in the book. But, you really got into this because you were eating a vegan diet and you were a vegetarian and a vegan. And you had pretty strong autoimmune challenges which totally resolved with carnivore. So you have this prejudice and bias towards it, admittedly.

JM: And so why don't you talk about your personal history and then we'll go into the details in the book.

PS: Yeah. So I've always been interested in health and nutrition. I grew up with a dad who is a doctor and a mom who is a nurse practitioner. The dinner table conversations were things like atrial fibrillation and Warfarin. Going with my dad to the hospital growing up, I always wanted to know, "Dad, what is causing people to be sick? And what's going on here?" And so it was so interesting throughout my life to think about what was at the root of our illnesses. And then when I was growing up, I had asthma and eczema and I never really knew what was going on with those. And I got the traditional therapies. I got Theo-Dur which is theophylline. I got inhalers. I got all the traditional stuff for my asthma and eczema and every once in a while I would take a corticosteroid cream and put it on.

PS: It never got any better. We just kind of put a bandaid on the symptoms and hoped that they would go away to come back another day. And as I progressed in my medical career, eventually I went to PA school and was a PA in cardiology for four years. But as we talked about in the first interview that we did, I think I wasn't satisfied there. I knew that I was going to do ... I wanted to change the paradigm. I really wanted to treat the root cause of illness, and so much of what I was doing there was working with some great physicians but I was giving medications to ameliorate symptoms much like I had experienced throughout my whole life. And I thought, "You know what? I want to get to the root of these things." And during that time, I continued to have eczema and I continued to have flares of varying severities.

PS: It was about that time when I was a PA that I was a raw vegan for seven months. And experienced what didn't seem to be a good reaction in my body at all. I lost maybe 25 pounds of muscle. Got to be very, very sort of skinny. My eczema only got worse and I didn't feel that great. I, at that time, learned about the paleo diet. Heard Dr. Jeff Bland speak. Learned about functional medicine and heard about this concept of a book of life in our genetics and what's written in our book of life. How are we meant to eat? How are we programmed to eat based on our human history? That's really where my book, The Carnivore Code starts and we can talk about it. But I think that that story of where we've come from and how that might inform the choices we make dietarily was so interesting to me then. It spurred me to add meat back to my diet and I became sort of a paleo diet eater.

PS: And the eczema seemed to get a little better, but it never went away and it continued. And at times in medical school, which I went to shortly after that, it got to be very bad. At times, so bad that one time I got septic and had to go to the hospital for IV antibiotics. After I got a streptococcal infection on my skin. And I was doing a lot of jujitsu at the time, so it was constant eczema on my elbows and my knees. It was really bad. And throughout the whole time, I kept thinking, "Gosh. There's something wrong. I still can't fix this." And I'm so grateful for everything I went through in my childhood and seeing my dad and just becoming ... I've just been obsessed with understanding the root cause for my whole life, because I think that if I weren't so tenacious about that, I probably would have just been putting steroid cream [inaudible 00:05:51] my whole life and never thought, "Why is this recurring?"

PS: But because it continued, I knew that there was something going on with my food and there was something I was reacting to. So I then tried autoimmune paleo and learned more and more in the functional medicine, integrative medicine sphere. Is it oxalates? Is it lectins? We'll talk about all these probably a little bit today. What is causing this eczema for me? And then I finished medical school. I still was eating a paleo diet. Went to residency in Seattle at the University of Washington, and had eczema there too! And I thought, "Man. This isn't even a warm place." I went to medical school at Tucson at the University of Arizona, there.

PS: And even in a cool place like Seattle, I had eczema so bad on my lower back that it just was handicapping. And a couple of times I had complete [inaudible 00:06:37] reactions where I broke out in eczema throughout my whole body. And it was some time along that path, maybe two years ago or so, that I heard someone talking about the carnivore diet and I heard about how ... This was Jordan Peterson I heard talking about the carnivore diet and how he'd used it to improve his autoimmune symptoms. How his daughter's autoimmune symptoms had improved. And my first thought was, "That's crazy." We've always been told that the plants are good for us. And throughout my functional medicine education, I was steeped in the knowledge that ... or I was steeped in the teaching that that plants were beneficial in some way and that we needed these and we couldn't be without them or we shouldn't be without them.

PS: But the more I thought about it and dug into it, I started to realize, maybe there's something to this. I'm at least going to try it. And so the first time I tried it, within a few days, my mood changed, and my outlook on life got to be significantly better and more positive. And I thought, "There's something to this." And then a few weeks later, the eczema had completely resolved and hasn't come back since. I've been eating a carnivore diet for the last year and a half. But there really was this sort of personal quest throughout to find out what the triggering food was and it was just so striking for me to see the eczema go away when I cut out all plants, and then the added benefit. Sort of mental clarity, psychological benefits were surprising. And I think that kind of hooked me, and I thought, "Okay. I need to just pour myself into this and understand this because this is going to help a lot of people, or it potentially could."

JM: Yeah. Thank you for that frame and background because I think it really helps the viewers understand your perspective. And I'd just like to add one more piece of information that might even elucidate [inaudible 00:08:16] further in that, unlike most people who go to medical school which is typically in their early 20s ... I mean, there's certainly people who enter school at a later age, like you did. You're kind of the exception. And you went into it ... essentially, having gone to medical school once, because the basic medical science you take as a PA is almost identical, if not identical to the one you take in medical school. So you went to the basic science twice. And the second time around, you had a very specific goal. You weren't just going through the motions to get your degree. You wanted to understand it at a foundational level. And it's that commitment to learning and the discipline you acquired that I think has given you the unusual skillset to really dig deep and go to places which really haven't been explored before.

JM: But you got those skills in your training. So I commend you for that.

PS: I was lucky enough to go to medical school twice.

JM: Yeah. Yeah. That's [crosstalk 00:09:11].

PS: Not everybody gets to do that.

JM: Yeah. It's a great thing. So what I want to jump into now is The Carnivore Code and what was really interesting to me because it seems there's a lot of confusion on this. There's many of the vegetarians or vegans promote the anthropological justification for their choice. And you walk through that. I mean, going back six million, three million years ago, and this evolution or this ... I hesitate to use the word evolution because many people [inaudible 00:09:41] be distressed with that word. But the transition might be better. Into modern day hominids. And the timeframe suggests that there's really something seriously going on here that can really, by itself, almost justify the choice of using animals as your primary food source. So, why don't you walk us through that?

PS: Yeah. This is an interesting story. I love this detective work. I'm not a paleoanthropologist but I kind of wish I'd studied it more in college. I've really become an acolyte of this discipline and studied with people like Mickie and friends and had a gentleman named Bill Von Hippel on my podcast. And just really been thinking about this ancestral [inaudible 00:10:25] transitional story. And it kind of starts with our brain as humans. And where we've come from. And the idea here, if we go back and we look at fossils, we can see the size of the brain based on the size of the cranial vault. And what we discover is that our primate ancestors who were around for maybe 60 million years, the size of their brain was essentially consistent throughout all of their prehuman evolution, right?

PS: They were just eating mostly leaves, probably. There is some good evidence that many primates do eat some animals. And they hunt some. So we probably even did not evolve from herbivores. We didn't transition from herbivores. We are descendants from probably omnivores directly. But the majority of our predecessors diet was probably plant foods. And with that in mind, it's interesting to note that the size of their brain never changed for 60 million years. And then about six million years ago, something happened in Africa. Probably due to tectonic plates shifting where the East African Rift Valley rose up and the forests became dry grassland savanna. In that forest, the population of our primate ancestors who were probably chimpanzee-like people or chimpanzee-like organisms, lifeforms, to move across the open plains rather than being sheltered in the trees.

PS: And the prevailing theory is that that caused us to eat different foods. And it was about that time that we start to see a gradual increase in the size of our brain. So between six million and 2.5 million years ago, there was a gradual increase in the size of our brain, from maybe 500 cc to about 600 cc. It's not a whole lot of change in the size of our brain, but a little bit. And connected with that, we see some really interesting changes in the skeleton. We were walking more upright. Our feet looked different. There were all sorts of adaptations to sort of moving through the savanna grasslands. And there's a famous fossil from 3.5 million years ago called Lucy which is Australopithecus afarensis.

PS: And that one is something that we've all maybe seen on Smithsonian or National Geographic. And Lucy, Australopithecus, didn't really look like a human. She looked like something in between

us and she wasn't really walking fully upright. And as we'll talk about soon, the size of her gut was still pretty big. She was somewhere in between what we would consider to be a human and a primate ancestor like a chimpanzee or a bonobo. And if we look at the size of the human brain, I've got a graph here from my book that I want to screen share in a moment, but if we look at the size of the human brain based on these fossilized cranial vaults, we see something really, really interesting happen 2.5 million years ago.

PS: So, here. I'll do the share, and show everybody this graph. So, when we're looking at this graph, you can see what I was talking about here in the past, that primate ancestors are back here, and then Australopithecus afarensis is-

JM: Otherwise know as Lucy.

PS: Lucy. 3.5 million years ago. Size of the brain. And then about two, 2.5 million years ago, there's this inflection point where the size of the brain suddenly starts to have this logarithmic curve and go up and that's about ... that's when Homo habilis arose. So this is a really interesting point. And as you can see on this graphic, what I've noted here, this is one of the graphics from my book, there were two things that were co-occurring at this time which suggests what might have caused this. The first of these was we find stone tools, which are called Acheulean tools. They're bifacial tools that look like they've been ... they've clearly been fashioned into a sharp edge by a lifeform. By our ancestors. And if you saw these on the ground, you would think, "That's been formed by a human." Or, "That's a tool. That's been made into something." You would know, because it's a bifacial tool. And so the stone tools arrived right about that same time.

PS: And then we also see cut marks on animals. We see cut marks in the bones of fossilized animals that are two to two and a half million years ago suggesting that our ancestors were butchering these animals and that they were using these Acheulean bifacial stone tools to cut the meat away from the bones. So super interesting stuff happening there. And there are all sorts of other suggestions that the hunting practices began at that time. There are large conglomerates of animals that were killed all at once. They were sort of herded into ravines and slaughtered all at once. So there are large graveyards of animals.

PS: There are stone tools that appear to have been moved from where they were created. So we were making the stone tools, appreciating the value of the stone tools, and carrying them with us. But there are all of these adaptations suggesting that we were hunting and that it was the practice of hunting that began 2.5 million years ago that probably created or allowed for this sudden shift in the size of the human brain. And that was really what made us human. And so you can see beyond that, the brain size just keeps going. Homo erectus comes in about 1.8 million years ago. And then off of Homo erectus, there's another species called Homo heidelbergensis, which isn't on the graph. And then Homo heidelbergensis evolves into Homo sapiens.

PS: And an interesting thing to note here, there are two other things I'd like to note about this graph that are remarkable. The oldest evidence we have for fire is about a million years ago. And so some people have said ... There's a book by Richard Wrangham who is an anthropologist. It's called Catching Fire. That it was fire that made our brains grow. Fire that made us human. But I don't think so, because fire wasn't around for 1.5 million years after the beginning of our brain size

growth. Certainly, fire played a role, but fire is a relatively recent adaptation in our evolution. So I don't think it was fire. I think it was hunting.

PS: And then we can see that the brain size gets really big, up to about 15,000 years ago. We can talk about what happened then. And it's been shrinking a-

JM: [crosstalk 00:16:28].

PS: What happened then?

JM: Yeah.

PS: That was the advent of the Neolithic Revolution, most likely and the fact that humans became pastoralists, agrarians and farmers, rather than hunters. So we'll talk about that. But there's one other point I want to make about the human brain size and that sort of 2.5 to maybe ... that 2.5 million year period to the present. Other advocates, and I think that within the vegan and vegetarian circles or plant-based circles, people sometimes say, "It was tubers that caused our brain to grow." And I think there's really good evidence that it was not tubers. And this is coming from the amylase gene duplication, which is such a fascinating concept. So I'll try to make sure everybody follows this.

PS: But as we saw on that brain size graph, there were multiple ancestors of us kind of preceding us. There was Homo erectus and then Homo heidelbergensis. And it's believed that Homo heidelbergensis that some of those people left Africa 600,000 years ago and became Neanderthals in Europe and Denisovans in Asian. And then some Homo heidelbergensis lifeforms or ancestors stayed in Africa and became our species which is Homo sapiens. And then about 80,000 years ago, Homo sapiens left Africa, going to Northern Europe and remet Neanderthals. That's why we have Neanderthal genes. We probably mixed with them at that point. And some people in Asia have Denisovan genes. But there's these two lineages of humans that separated and came back together about 60,000 years ago. And there are two really interesting things about that.

PS: If we look at fossils of those people from 60,000 years ago, and we look at the collagen in those fossils, the bones, we can look at these stable isotopes and get a sense of what we were eating, both Homo sapiens and Neanderthals. And the stable isotopes, these are the d15 Nitrogen and d13 carbon, can suggest where we were getting our protein from. And the studies that I'd mentioned in the book that you probably read about suggests is that we were getting the majority of our protein from high level trophic animals. And we actually were eating more animals than other known carnivores at the time, like hyenas.

PS: So the levels of nitrogen and carbon in our fossilized remains from 60,000 years ago, are greater than hyenas. And you can see this sort of trophic accumulation of the stable isotopes, suggesting that we were eating bigger and more animals than known carnivores. So this is evidence that we were eating mostly animals 60,000 years ago. The salivary amylase mutation is fascinating because it doesn't occur in Neanderthals and Denisovans. But it does occur in Homo sapiens. And this suggests that the branch point from us for them, from us, was about 600,000 years ago. So up until about 600,000 years ago, we did not have a salivary amylase gene mutation. So it's very

unlikely that we were eating any significant amount of starch, because if we had been, we probably would have developed a selective pressure to get this salivary amylase mutation. This, at least, duplication. What we see now is that all living people on the Earth have a salivary amylase duplication because we're all descended from a Homo sapiens group that left Africa 80,000 years ago that appears to have had an amylase duplication. So they were eating more tubers 80,000 years ago.

PS: But up until that point, there's no evidence for an amylase duplication, arguing strongly again the notion that we've been using tubers for any significant amount of nutrition. So, does that kind of make sense? It's pretty interesting.

JM: That makes sense.

PS: Cool how it fits together.

JM: As you go in later in the book, we'll understand that tubers may be a very poor choice for a wide variety of reasons, even though there is a lot of confusion on this, and you kind of dispel some of that. So, why don't we go to the next point which let me just see here. Oh, Weston Price. Unless you want to say anything more about the human brain evolution because I think that's a really interesting preface to the details of addressing some of the conventionally held views on the benefits of vegetables or plants.

PS: Yeah. I think that it was hunting that made us human is the takeaway. It was hunting and animals that made us human. Not eating tubers. Not eating vegetables. And that animals have always been a big part of our diet. But yeah. We can definitely talk about Weston Price's observations.

JM: Yeah. So as many people know, he's a pioneering dentist from a hundred years ago. And really traveled around the world to document these indigenous cultures and the foods they were eating. [inaudible 00:21:14] correlated the foods they ate to their health. And well, to me, the big take home point was that he never found one culture that was thriving on plant foods alone.

PS: There was no single culture that was even eating plant foods alone, because yeah. That didn't exist. And then the other point that I highlight in the book is that there were some instances where he could directly compare in Africa sort of tribes that were more plant heavy and tribes that were more animal heavy. And the tribes that ate more animals were stronger, taller, and had better health than the tribes that ate more plants. So he had a direct comparison looking at the overall health, strength, virility of people's in Africa in the 1930s and 1940s, and he saw that people that favored animals foods were doing much better than the people that favored plant foods. So that juxtaposition was quite revealing to me as well.

JM: Yes. So, from there, we go into the issue of phytonutrients. Fido meaning plants and plant-based. It's from Latin or maybe Greek. But anyway, that's a pretty pervasive belief. And one that I held up until recently. In fact, I'm writing this treatise that will probably turn out to be a few thousand pages. It's like over a thousand pages now. Maybe 1,500. And the initial focus of that [inaudible 00:22:42] a few hundred pages, was all about phytonutrients from seeds. And I was

under the understanding and belief that it was these incredible phytonutrients that were largely responsible for activating these profoundly powerful pathways for longevity. And you kind of turned my world upside down in reevaluating that with this perspective. So, it's ... Why don't you take off from there? Because I mean, phytonutrients which you call ... which is [inaudible 00:23:08] precise term phytoalexins are these plant defense compounds that may be causing more harm than good, even though we're led to believe. And it's a widely held belief. And I would suggest probably more than three-fourths, maybe 90% of people believe this. Maybe 95%.

PS: It's pervasive. I think this is one of the more controversial things, controversial hypotheses that I have advanced is ... and we can talk about xenohormesis as a corollary to this. But if we just back up one moment and think about it from our ancestral perspective as well, from an evolutionary perspective, it starts to make more sense similarly. Plants and animals have been coevolving for 450 million years. The Earth is really old. It's like six billion years old. But plants and animals, the best data I could find was that plants have been on land for 500, 450 million years. And animals and insects have been around for a similar amount of time. And so if we think about it in that perspective, this all kind of makes a lot of sense. And I think that so often today we're losing these perspectives and so many of these ideas are accepted without a real examination if they make sense from an ancestral evolutionary perspective.

PS: But for plants and animals to have coexisted for 450 million years, there's a real discrepancy between the plant and the animal. And the biggest difference here is that plants are stuck in the ground. I provide this sort of thought experiment in the beginning of the book or in the beginning [inaudible 00:24:48] chapter three, where I ask the reader to imagine that they are going to be buried in the sand like they were as a child at the beach. But they're buried in the sand so tightly, that they can't move. And only their head is above the sand. And then I'm just going to sneak up and paint their face like a soccer ball, and then what do you know? What do you know? This trove of 20 irascible six year olds from the soccer team shows up and you're buried up to your neck in the sand with your face painted like a soccer ball with a bunch of cranky six year olds hanging around. How do you feel?

PS: I think you'd feel vulnerable. And I imagine that's how many plants feel. If we can anthropomorphize a little bit, that's how plants are. They're stuck in the ground. And so, out of necessity, in order to keep the ecosystems balanced for 450 million years, they've had to evolve. They've had this selective pressure to evolve plant defense chemicals. And I don't think anyone debates the presence of phytoalexins. This is widely accepted within botanical literature, scientific zoology. No one ... I don't think anyone debates that plants make defense chemicals. And we humans, we're familiar with some of these. I just think that we're not familiar how many these are. How pervasive they are, and how many of the plants we eat contain thousands and thousands of them. But you know, it's almost the holidays now. I guess when this podcast comes out it's going to be after the holidays. And around the holidays, I always remember growing up, my mom said, "Don't eat the poinsettia. Don't let any kids near the poinsettia plant to eat the poinsettia." These bright red plants that are frankly toxic to humans.

PS: Similarly, rhubarb leaves. You can't eat the leaf of rhubarb. You can only eat the stem.

JM: [inaudible 00:26:32].

PS: Yeah.

JM: It's not a good idea.

PS: You could get really, really sick because of the oxalates in rhubarb. And we'll talk about those maybe a little bit, too. But we're aware that some plants are so toxic that they're frankly poisonous. That we could die, right? And it's the same idea that basically every plant that we observe in nature is part of a delicate balance. A delicate exchange system with other animals. And it's had to develop plant defense chemicals which are phytoalexins. So, I think the part of this that is so radical for people and challenges so many of our long-held beliefs, like you're suggesting perhaps 95% of the population or more is going to be challenged by this notion is that so many of the chemicals that we imagine to be phytonutrients or to be hormetics in plants are actually phytoalexins. They're plant defense chemicals.

PS: And there's definitely some nuance here. As you know, I did a podcast with David Sinclair where I sort of challenged him, friendly, on this notion of xenohormesis and we can get into that. But-

JM: Yeah. And let me just insert. That was an excellent podcast. One of the best interviews with Sinclair I've ever seen. And I called you up the day after [inaudible 00:27:44], because you really, you really hit him with some really good molecular biology review. So if anyone wants to review that, we'll probably put a link to it. It was just an outstanding interview.

PS: So if we think about the plant compounds that are thought of as phytonutrients, right? Because when we're thinking about a carnivore diet, if I'm going to suggest a carnivore diet, or anyone's going to suggest a carnivore diet, one of the things that people often question is what about all the nutrients in plants that I'm missing? And there's a chapter in the book where I talk about the actual vitamins and minerals. That's chapter eight where I go into detail and say, "Hey, look. In terms of vitamins and minerals, you can get everything from animals. Animals are a better source of all the vitamins and minerals than plants." But then people say, "What about all the polyphenols and these phytonutrients?" They're kind of magical, right? And this is where we get into the realm of phytoalexins. The plant defense chemicals.

PS: And so this is the concept that I think is one of the more challenging ones in the book for the mainstream consciousness that so many of these chemicals that people think of as beneficial are plant defense chemicals. The majority of polyphenols are plant defense chemicals. And the interview with David Sinclair is a fantastic example. David Sinclair, I think was one of the first people to really isolate the molecule resveratrol.

JM: No, no. He wasn't. There was researchers that preceded him. His claim to fame was that he found that there was a connection [inaudible 00:29:10] resveratrol activating SIRT1.

PS: Right. Right. So, but this resveratrol molecule is found in grapes and peanuts and it's also found in some berries. But it's a defense molecule. And what it does is it's produced in response to the botrytis fungus. So whether a grape is getting fungus on it or a peanut is getting fungus on it,

it'll produce resveratrol. And resveratrol is an oxidative stressor to the fungus organism and does other things negatively for the fungus. It's a plant defense molecule. So resveratrol is a plant defense molecule. And when we look at the literature regarding resveratrol, which I talked about a little bit with David Sinclair in that podcast, what we find is, it's interesting because it definitely does activate SIRT1, which appears to be a good thing, but it has other negative effects in the human body. Specifically, there's a good amount of research on resveratrol suggesting that it affects hormonal metabolism negatively.

PS: It decreases androgen precursor, specifically DHEA, leading to lower levels of DHEA and testosterone and other androgens when you're using resveratrol. And many molecules that are polyphenols do this in the flavonoid class of molecules. So, when we're talking about polyphenols, we're looking at molecules with these aromatic ring structures. And we can go into detail on other ones in that class, as well. Curcumin is another one. And I'll clarify this briefly just so people understand my position. It's not that I'm saying these molecules have no value in humans. It's my urging, my suggestion when we're thinking about these molecules is that we think about them like pharmaceuticals, because they really are.

PS: And pharmaceuticals are really powerful and can be life saving molecules. But if I'm going to prescribe or recommend ibuprofen or metoprolol or a psychiatric drug to a patient, I'm always going to have a conversation about the potential side effects. But what we've forgotten about with these plant molecules is that they too are molecules and that they too are pharmaceuticals, in a way, in many ways, and they too have side effects. And those side effects are what I'm calling attention to with our discussions in The Carnivore Diet. I think that for some people, plant molecules can have a medicinal value. But when we're using them as food, every day, my concern is that we can be getting too much of a medicine and the side effects start to outweigh the benefits. And that is where I think the elimination of them becomes valuable for people and the cutting out of all the plants can be really a game changer in terms of inflammation and autoimmunity.

PS: But the takeaway with regard to these molecules is that they are pharmaceuticals. And if we just touch briefly on curcumin, that will illustrate it as well. Curcumin has a lot of good data about being an anti-inflammatory molecule. There was some conflicting data and I know that there are some randomized trials that are lacking. But I think that curcumin, we can pretty safely say curcumin is anti-inflammatory. But so is ibuprofen and so is naproxen. And we know that ibuprofen and naproxen have negative side effects. In the case of both of those, nonsteroidal anti-inflammatory drugs, they can affect the production of mucous lining of the stomach. And by inhibiting the production of prostaglandins can negatively affect kidney function.

PS: In the case of curcumin, and I talk about this in the book and I provide all the references, there are a number of studies which show that at a biochemical level, it can also do some negative things in our body. It can affect the enzymes that wind and unwind DNA called topoisomerases. It can affect ... it can potentially affect oncogenes like p53. It appears to affect a potassium channel called the hERG channel. And it's been shown to damage both [inaudible 00:32:56] and cancer cells in cell cultures. So, my suggestion for people or the hypothesis that I'm advancing in the book with regard to many of these polyphenol-like molecules, is that we need to remember that they're pharmaceuticals and that they have side effects and we can't ignore the side effects. Whether it's

resveratrol side effects on androgen precursors, or curcumin side effects in other places in the body. DNA winding and unwinding, et cetera.

PS: The final thought there is why are we using these molecules and can we achieve health without these molecules, right? So if we're using curcumin as an anti-inflammatory, why not just try and address the actual root of the inflammation rather than taking a pharmaceutical that has side effects? It's the same model that we see in Western medicine, right? [inaudible 00:33:42] I shouldn't give somebody ibuprofen for a headache. I should try and figure out what's causing the headache.

JM: Yeah. And it becomes ... Let me just comment on the curcumin, because it is ... I couldn't agree with you more in the pharmaceutical applications of these phytonutrients. And curcumin is a very good example, because it's profoundly beneficial in almost every single cancer. It's a part of the regiment. And I would be reluctant to not recommend that anyone who has cancer, because I think it's a useful strategy. But it's a drug, like you say. And then the other component is many times, there's other ways to reduce ... well, in curcumin's case, to reduce inflammation. But a more challenging one [inaudible 00:34:21] is the longevity proteins when you're activating them. What are you going to do to activate them? Well, there are some number of things that you can do.

PS: Exactly.

JM: Like exercise and time restricted eating.

PS: Ketosis.

JM: Ketosis.

PS: Exactly.

JM: And then you get the same benefits and more with none of the downsides. It's crazy. But it's true.

PS: That's exactly what I talk about in the book. And I call it the radical life. I have these little shticks. And so I think of it the same way. It's like, "Hey, look. We can ... " And I talked about this with David Sinclair. And we can change the NAD to NADH ratio in the cytoplasm of our cells by being in ketosis. And during that podcast, he and I discussed multiple published studies, both in animals-

PS: He and I discussed multiple published studies, both in animals and in humans. And it's very well documented that beta-hydroxybutyrate metabolism in the cytosol is going to bypass glycolysis and therefore changes the NAD to NADH ratio. We don't have to go into the detailed biochemistry, but when you increase NAD to NADH in the cytosol, we know that the sirtuins get turned on. So you can affect longevity mechanisms by going into ketosis from time to time. Whether you want to do it long-term remains a question, but you can cycle it, and like you said, you can do time-restricted eating. So, you can do fasting. And with regard to antioxidant defense mechanisms, we didn't even talk about sulforaphane and the isothiocyanates, but-

JM: Well, before you get there, I mean when you activate [inaudible 00:35:42], you're also activating FOXO3.

PS: Exactly.

JM: Which activates your endogenous antioxidants. That along with NRF2, I mean you're off to the races.

PS: And MT2-Metallothioneine-2, yeah. And the sirtuins are these deacylase enzymes that turn on a whole bunch of other great genes. So you can turn on your sirtuins without resveratrol. And that's my message to people is, hey, we probably don't need these plant molecules necessarily. In certain applications, I think they definitely have profound value. But let's look for the root cause and let's think about how we can do this with less side effects.

JM: Yeah. Now before we leave that and go on to the other ones, you had a really terrific analogy that I want you to share with respect to these phytoalexins. And that they serve great roles in plants and that's why they produce them. Not only is plant defenses but for about their mechanisms. But it's like the difference between two operating systems, that one for the PC and one for the Mac and they don't, aren't the same. And if is to try to mix them up, you can run into complications. So why don't you share that? Because I thought it was a great analogy and really help people understand what's going on here.

PS: Yeah, it's funny. I don't remember when I came up with that analogy, but it's been a fun one to kind of play with. I think I probably came up with it on Ben Greenfield's podcast. But the idea is it's totally true. Plants and animals diverged hundreds of millions of years ago. And it's just kind of human evolution. Just like homo heidelbergensis, the Neanderthals and Denisovans branched off 600,000 years ago and they went in different directions. Life on the planet between plants and animals diverged hundreds of millions of years ago. And our parallel evolutions have yielded vastly different biochemistries. This is just our internal mechanics, our internal engines and our internal computer programs.

PS: And when you look at the way a plant does their biochemistry and the molecules that plants use for their biochemistry, they're just not the same as ours. We have different operating systems. We have our own system of antioxidants. We have our own defense system. It's called the immune system. And there's innate and adaptive immunity. Plants don't really have that. Plants make molecules to defend against invaders. We have an immune system with cells and plants use a different chemicals in a way, they use chemicals differently than we would. And so there is a real deeply seated misconception within the broader populace that molecules from plants can be "antioxidants in humans" and this is something that David Sinclair talks about in his book. And I really appreciate that he also fought against this notion. Plant molecules do not act as direct free radical scavengers in our body. Plant molecules are not directly in antioxidants. They can trigger our antioxidant response system, which is hormesis and we can talk about that. We sort of already have.

JM: No. Only need to expand out because it's a really important concept.

PS: Yeah, we'll expand on that too. But the plant molecules do not act as antioxidants in the human body. We have glutathione, we have the enzyme, superoxide dismutase, we have uric acid, we have vitamin E. We have molecules that do the free radical scavenging the human body. What we're talking about here is the movement of electrons. When people hear the term free radical, that's a molecule that's had one of the electrons in orbit pulled out and they have an unpaired electron. An unpaired electron is kind of like those irascible children that are going to kick your face when it's painted like a soccer ball, they don't behave well.

PS: They run around the body. If it's unpaired electrons, these are free radicals. They run around the body and they try, and pull electrons off other molecules. But if they pull an electron off a lipid, they're going to make a lipid peroxide. And if they pull an electron off a protein, they're going to oxidize a protein and change the shape of the protein. And that's how we get oxidized LDL. So free radicals go around kind of making everybody else have a bad day by pulling electrons out of their orbitals. But we have our cellular police force at glutathione to go and say, "Hey, I'm going to give you an electron so you can calm down." And that's what glutathione does. And that's our antioxidant system. Plants don't do that. Plant molecules do not come into us and donate electrons. They're the reverse. Because they're plant defense molecules, they're pro-oxidants. And we'll talk about this in a moment.

PS: But plants and animals have different operating systems and the molecules don't act in the same ways. The same is really true of the vitamins and minerals in plants versus the vitamins and minerals in animals. And we can talk about that as well. But let's just talk about this concept of hormesis, because I think it's really interesting. And I think that we kind of talked about it, but let's just go into detail here.

PS: So David Sinclair and others have advanced this concept of a xenohormesis, which means molecules that are outside of us are good for us because there are a little bit a poison. And that can sort of be true, but my problem with that theory has to do with the side effects, right? And so it is true. Sulforaphane maybe a good molecule to illustrate this. Sulforaphane is an isothiocyanate. Sulforphane comes from a glucosinolate called glucoraphanin. When an enzyme called myrosinase degrades glucoraphanin it becomes sulforaphane. And I'll just describe-

JM: It just by the way that's in broccoli, the primary glucosinolate in broccoli.

PS: Yes. That's the primary glucosinolate in broccoli. But isn't that interesting because that's a plant booby trap. Then myrosinase and the glucoraphanin don't get combined unless an animal's chewing it, right? This is how we know that it's a plant defense molecule. This is a plant booby trap. Sulforaphane is a plant booby trap. People might've heard goonies, booty traps. Sulforaphane is a plant booby trap and it's a pro oxidant molecule. If Sulforaphane were circulating around broccoli or kale or cabbage or cauliflower, any of these brassicas, it would act as a pro oxidant.

PS: It would be a bad thing for them. It would run around stealing electrons from their molecules. They don't want that to happen. They keep Sulforaphane in a tight little package. They keep the booby trap unsprung as glucoraphanin and then they add myrosinase. It's kind of like super glue. You combine the two types of glue and it has a chemical reaction. The enzyme acts on

glucoraphanin, boom, booby trap is sprung. You get sulforaphane. Now, sulforaphane comes into our body and acts as a pro oxidant. It acts as a pro oxidant not an antioxidant, a pro-oxidant. And by acting as a pro oxidant, it triggers the antioxidant response system in the liver, which as you suggested is NRF2. It's the NRF2 pathway in the liver.

JM: It's the DNA. So, I'm not sure just liver. It's systemic, I think.

PS: Yes, it's systemic. So the NRF2 gets activated probably throughout our body, but especially in the liver.

JM: Right.

PS: And when NRF2 gets activated. NRF2 is transcription factor, kind of like some of these other transcription factors we talked about earlier. And it can control the turning on and off of other genes. And it's going to turn on genes like glutathione peroxidase, glutathione transferees other genes involved in the antioxidant system. But when NRF2 goes on, glutathione goes up, which is a good thing in the short term. And people see that and they say, "Okay, great." And there are studies for sulforaphane showing that glutathione goes up and DNA damage goes down in the short term. You can say, "See it's a hormetic molecule." But what they're forgetting, what they're missing here is what we talked about before. The side effects, right? The side effects of this molecule are what people are missing.

PS: These are what I've called collateral damage and the notion that we advanced earlier is still relevant. We don't need sulforaphane to protect our DNA. We don't need sulforaphane to have optimal antioxidant status. We can do things like heat exposure and cold exposure and exercise, which can also turn on the antioxidant response system and increase our supply of glutathione, protect our DNA. And as you suggested, don't have any side effects, right? They don't have any side effects. But sulforaphane has side effects. It's a molecular pharmaceutical, it's not-

JM: Well, actually, it does have side effects. I was mistaken, but they are beneficial side effects.

PS: Yes.

JM: I feel deleterious.

PS: Yes, that's true. It's a six pack and biceps and things like this. So sulforaphane when it circulates in our body can oxidize membranes of cells and create 4-HNE which is 4-Hydroxynonenal, Acrolein which are products of oxidation. These are lipid peroxides, which can be very damaging. And it also interferes with the absorption of iodine and competes with iodine the level of the thyroid. So people may be familiar from like these national geographic programs.

PS: They may see people with these big necks, these goiters that occurs throughout the world. And people who are eating isothiocyanates in areas where there's not enough iodine and they get goiter. They get reactive hypothyroidism and the thyroid gland swells because they're not getting enough iodine. So you don't see people in the US getting these goiters when they eat broccoli. But there are many cases now of people having pretty significant thyroid issues from over consumption of

brassica vegetables. It's not really doing us any favors and we just need to remember they're springing booby traps on us. You can get by and we can talk about how to detoxify sulforaphane and isothiocyanates and glucosinolates and that's actually a pretty interesting story as well.

PS: But it's not doing us any favors and it's not a good molecule for us, right? We don't need sulforaphane to have optimally antioxidant status. So xenohormesis, for me, the concept falls apart because of the side effects. We don't need these things. There are no examples of plant molecules that I have seen. Again, we're all learning, but I am not convinced the plant molecules provide any net benefit. They don't let us do anything we can't otherwise do by living a radical life and they have all the side effects which kind of drag us down. That's where I think xenohormesis goes wrong. But it's an intriguing concept based on a model which does work, which is environmental hormesis and those are all the things you are describing. Exercise, sunlight, heat and cold.

PS: These are not molecular hormetics. It's the xenohormetic, the molecular hormetic mechanisms where I think that the pattern, it's incorrectly applied, right? Hormesis applies to environmental exposures, not to molecules. Because the molecules from plants, from a different operating system, they always run around and do bad stuff on us because they because side effects.

JM: That is such an important distinction. Really, a groundbreaking shift in thinking. So I thank you for bringing that to everyone's attention. Another environmental hormetic that's not been proven but I believe is true would be EMF exposure. In my belief, and I hope so because the exposures we're going to have in the very near future are going to be pretty radical as you would put it. Radically increase for sure almost exponentially and they're already very high. But if you can get into a safe environment where you're a safe sanctuary and get this regeneration repair going on, that when you go out in the environment, you get exposure, then you get hormesis. You go back in at night, you recover, repair and it actually makes you stronger. But environmental exposure is not a plant toxin.

PS: It's not a toxin. It's not a molecular hormetic. It doesn't break down, yeah. And I think that you're right about EMF. I definitely think it probably could be an environment hormetic. We know that we've always been exposed to some. But as you have pointed out so well, the levels are so much higher now than they used to be. As people will know, hormesis is a U-shaped curve. If you get too much of a toxin, it gets really toxic. I mean, I have friends who are radiologists and radiologists circles, people really, for the most part, accepted the notion that gamma rays and the radiation that we get from just being on the face of the earth is a little bit hormetic.

PS: But we know that if we're exposed to too much in a CT scanner, that can increase our risk of cancer. So there's a sweet spot and we've always been exposed to some EMF, but now it's abundant and we're going to need to find a way to let our body regenerate. So we don't get broken down by it.

JM: Yes, indeed. Now in the book you talk about the amount of exposure that we have to toxins. Most of us, certainly people who view our site are concerned about pesticides, the agricultural pesticides, they're being user pervasive, especially glyphosate. But when you look at the amounts and you compare them to the environmental, not the environment but the plant toxins, it's like 10,000 times different. And I believe you got the data from Bruce Ames who I've interviewed

previously. He was like, we get one and a half grams of these plant toxins versus 10,000 of that for the synthetic pesticide residues.

PS: Yeah, it's pretty striking. That's a paper called Dietary pesticides (99.99% all natural) and it's a great paper. The references in that paper are gold. And in the paper he has a chart which he lists 42 naturally occurring pesticides in cabbage naturally occurring pesticides, not pesticides that are sprayed on carriage pesticides the cabbage produces. Because for the last 400 million years its ancestors have been trying to get rid of bugs and say, "Stop eating me."

PS: And as Bruce Ames notes astutely in that paper, only a fraction of these compounds and plants have even been tested for mutagenicity in humans. But there are a couple of papers that I referenced in the book and until I read that paper from Bruce Ames, I'd never seen these studies, but they're fascinating. And one of them is on 951 substances and what they're looking at is clastogenicity. It's in cell culture, which is the best we can do. But they can expose mammalian cells, they can expose human cells to many of these plant compounds in cell culture and look for DNA breaks. And that's called clastogenesis. And it's a fascinating paper that I reference in the book. And you can look, many of these polyphenolic compounds in cabbage and other plants do break TNA in cell culture.

PS: It's quite concerning. And so you're thinking, "Wow, I don't know. Do I want these in my diet? I don't think, maybe I don't." Curcumin is one of those. It's shown to break TNA. So it's like, "Risk benefit." Right? Allyl isothiocyanate which is a different isothiocyanates and sulforaphane has also been found to break chromosomes very strongly as have many of those 42 compounds in cabbage, some of which are glucosinolates and otherwise. But yeah, these plant toxins, we just haven't studied them. And you're right, we get about a gram and a half per day of them. If we're eating a plant, a diet that contains significant amount of plants, we'll get a lot of these. And it pales in comparison to the amount of glyphosate in our diet. I think glyphosate is a big problem and we should-

JM: [inaudible 00:50:28] the negatives of glyphosate.

PS: No, not at all.

JM: Because you could avoid it at all costs. There's no question.

PS: Yeah, absolutely.

JM: So shifting gears so that we've been discussing the adverse potential problems from consuming plants. And then the counter point to that is the benefits of consuming animal foods. Before we talk about the three big Cs, which I really want to dive deep on, would be the ... Had you mentioned B12 in there. Because I mean no one will disagree, certainly anyone who's-

PS: I do.

JM: ... vegetarian, that B12 is a big issue. Maybe one of the most important, certainly from a neurological perspective. Why was-

PS: Absolutely.

JM: Then we start with B12.

PS: Yeah. So the first part of the book is about the evolution. The second part of the book is about the plant toxins. And the third part of the book, or at least chapter eight is about all of the nutrients that we can get in animals that we can't get in plants. And like you're saying, this is a real ... I wanted to juxtapose these and say, okay, plants are probably not that great for humans. But similarly today a lot of animal foods are vilified. And I really wanted to stand up for animal foods and show how uniquely healthy they are for humans because that's the other side of the equation. And so chapter eight is all about these unique nutrients in animal foods that you can't get in plants and how they affect us as humans profoundly.

PS: So B12 is a great example. There's one study I cite in that chapter, which is probably my favorite study. And I think it was epidemiology, but it looked at B12 levels in relation to the size of the brain. And what they found was that the people who had the lowest B12 levels had the smallest brain. And it's scary, right? This is a really a scary thing. And you think already you get [inaudible 00:52:13]

JM: That's not evolutionary. That's epigenetic.

PS: That's epigenetic. But, if we go back to that original graph that I showed looking at the human brain size. We know the brain size has been declining over the last 15,000 years. We talked about the neolithic revolution. Certainly when humans stopped hunting, started farming more. Their B12 and many other nutrients went way down. And that's a compelling hypothesis for this decline in brain size. We see it in observational studies as well. There's a very strong correlation between brain size and B12 and there's unpublished data from study. I don't know if you know about this.

PS: This is actually really interesting and I'm trying my best to get ahold of the researcher who did that study, who's now in England. But there's unpublished data from that study looking at which people were vegetarian and vegan, and there's a strong correlation between low levels of B12 and vegetarianism, veganism, more of the veganism and get this. The largest vegan brain was smaller than the smallest omnivore brain, right? So that's a pretty scary thing to think about. You can see the headlines now, plant based diets will shrink your brain. It's a scary thought. And it's really not that much of an extrapolation, but that's unpublished data from that study that I don't know why they didn't put it in the paper.

PS: But I really want to get that researcher to share that data with me so that I can formally share it in the book. But the levels of B12 correlate with brain size. And we know that this is true and the only place to get B12 is animals. People will say, "Oh, you can get an allergy." That's baloney. I don't think it really is the same type of B12. And then furthermore, we have to think about the bioavailability of B12. And so this molecule is critically important in the folate cycle. It's needed to convert homocysteine to methionine and it's needed to make succinyl-CoA for the Krebs cycle. It's needed for all growth in our neurons. It's really important.

JM: Yeah. And so it's a significant cause of blindness in that community too. If you're not getting it, that is a clear side effect. And there's been a number of reported cases of that.

PS: Yeah, it's scary.

JM: Yeah, so let's progress now to some of the other nutrients.

PS: Okay.

JM: The three Cs. Talk-

PS: Yeah, let's talk about Creatine. Can we talk about creatine first?

JM: Absolutely. Go for it.

PS: Creatine is amazing. People may be familiar with creatine from bodybuilders, but creatine is also part of what's called the phosphagen system in our muscles. It stores a phosphate item as creatine phosphate and donates that phosphate to ATP when it gets used up in our muscles during intense exercise. And it's also part of energy metabolism everywhere, not just in our muscles, in our brain too. When our brain uses ATP, creatine donates a phosphate to the ATP and it runs the machine.

PS: So basically ATP and by proxy creatine, our molecular gasoline. These are what we run on. And there are some incredibly striking studies that I talk about in the book where vegetarians and vegans were supplemented with five grams of creatine per day, which is the amount of creatine in one pound of meat. And invariably they had improvements in working memory, intelligence, decision-making tasks.

PS: It was just so striking to see that you could get intelligence, memory and recall improvements in plant-based dieters when you give them back creatine. There's no in plant foods. We can make a small amount through methylation. In fact, the majority of our methylation goes to make creatine. So when we do the methyl cycle, we make homocysteine to methionine become SAM-e. A lot of that SAM-e goes to make Creatine, but if we really cannot make enough on our own and when we get it in our diet, we are allowing our brains to function at full capacity and our muscles. And in plant based dieters, if we give them back creatine, they get smarter. That is not an indictment against the plant based diet, I don't know what is. It's like, wow, okay. And as you suggested at the beginning of this video, I have nothing against people who choose to do plant based diets intentionally for their health.

PS: I just want to warn people about the potential problems with that so that they can supplement and monitor their health. If there are people listening to this there are vegans, vegetarians and they're thriving, I'm so happy for them, more power to them. I personally think that animal foods are going to be better sources of nutrients, but I just want people to lead full and happy lives. And if they're doing that, eating that way, that's great. They just need to be aware of the fact that, that could have some serious health consequences like this one and B12. Without enough creatine, we're not as smart or strong as fast as quick on the draw as we could be. It's pretty striking.

JM: Yeah. So let's go to the next C, choline.

PS: Choline is so important for the membranes of every cell in our body. Every cell membrane is made from phosphatidylcholine, in the head of a phospholipid molecule that forms the lipid bi-layer. And so we are made of choline. The reason we are together as humans, the reason we exist is because we have cells that are in wrapped by a lipid bi-layer. And that lipid bi-layer is made from choline. And choline is found primarily in animal foods. You essentially cannot get choline in any particular, in any significant amount in plant foods.

PS: You'd have to eat like three pounds of broccoli a day to get your RDA for choline. And you can get your RDA for choline in a few egg yolks or a few ounces of liver. It's much more bioavailable in animal foods as well. So if we want to have enough choline to make neurotransmitters acetylcholine and to make membranes of ourselves, we must eat animal foods or supplements. The other problem with choline is that if we don't get enough, it's very well known that we will develop nonalcoholic fatty liver disease. Because one of the ways that choline is using the body is to package triglycerides into VLDL molecules in the liver for export to the peripheral tissues.

PS: But if we don't have enough choline, we can't make enough VLDL because VLDL is a single lipid layer, a single phospholipid layer, particle in the blood. And we may talk about lipids later. But if we can't make VLDL because we don't have enough choline, we're going to get nonalcoholic fatty liver. The only other thing I'll add about choline, and this may wrap into a later discussion about TMAO is that people who are worried about TMAO will say, "Don't eat foods with choline because you don't want TMAO." And this is really badly misguided. Maybe later we'll talk about TMAO. But we need choline for healthy membranes, healthy neurons, healthy neuronal transmission to make critical neurotransmitters in our body. Only found in animal foods, egg yolks, liver are probably the best sources.

JM: Well, they are the best sources and-

PS: Yeah.

JM: ... the point here is don't avoid choline because you're concerned about TMAO which is essentially fake news folks.

PS: Fake news, yeah.

JM: Yeah, that is not true that TMAO is a nonissue as we'll talk about later. So choline is good. I personally have at least four egg yolks a day. Because the average person is seriously deficient in this. And I think men need about 550 milligrams and I think four egg yolks gets you that. And I still have liver too.

PS: It can depend on polymorphisms and some of the methylation genes. If people have MTHFR polymorphisms or polymorphisms in the enzymes that make choline then, which is PEAMT,

phosphoethanolamine methyltransferase. But people can look at that. But generally speaking, I think it's better to get more. You definitely want to be on the positive side of choline.

JM: That's for sure. Crazy not to. So the other C, that I'm become recently passionate about after attending the Academy of Comprehensive Integrative Medicine a few weeks ago is carnosine. So why don't you talk about it and I'll talk about my new insights on it.

PS: Yeah, I'm excited to hear, what you've learned about this one too. So carnosine, I mean carnosine and carnitine are both brothers. They're a little different, but they both have carn, C-A-R-N in them. And that's connected with meat, right? So they're only found in meat. The story is the same for all of these molecules. Our body can make a little bit, but it's not enough. And as I talk about in this chapter in the book, vegetarians and vegans are universally, not universally, I can't say that, but they are essentially usually deficient in all of these nutrients because they're not getting it in their food. So carnosine is thought to be very important because of its antioxidant roles directly and the way it controls the formation of advanced glycation end products. And the events glycation end products are the attachment of sugar molecules to protein and lipids.

PS: And they seem to correlate to aging in humans. Many of the listeners may have heard of AGES. But what's so interesting here is that we can get AGES in the diet, but the majority of AGES are probably formed endogenous honestly. And we don't want to eat a bunch of AGE rich foods in our diet. These would be things like deep fried foods, bacon that's cooked in its own grease is delicious and crispy, but it's probably the highest AGE food that I'm aware of. People might be surprised this guy is a carnivore.

JM: I don't think bacon is an AGE. I think it's an ALE.

PS: Oh, interesting.

JM: Advanced lipo oxidation end products.

PS: Yes.

JM: Because definite a distinction, the advanced glycation end products really are for more for the glucose and protein products.

PS: Right. When these are measured, maybe the ALEs are also found in butter. So butter and bacon are kind of high, but you can also find them in cooked plant foods. Anything that's deep fried, anything that's, has that like crispy shell is AGEs. And it's hard. I don't think we know how much dietary AGE contributes versus endogenous, but I think that the major problem is endogenous AGE, the ones that are formed in our body. There are a number of pathways that produce them, a methyl glyoxal and CML, other sort of pathways that produce the AGEs. But carnosine is an important nutrient in preventing the formation of those. And guess what, not present in plant foods at all. Only present in animal foods, vegetarians woefully deficient. And as I mentioned in the book, there are plenty of studies that show that vegetarians have higher levels of AGE formation in their bodies.

JM: Well that's a good primer. And I want to expand on that from the information I've learned just recently. Is that the value of carnosine, there's probably dozens of clinical conditions where it's useful for. From heart disease to brain disease, Alzheimer's seizures. It's amazing. So what is carnosine is it's really a di-peptide. Simple two amino acids plugged together, Beta-Alanine, which really isn't used too much to make proteins. It's called a non proteogenomic amino acid and then Histidine, which can serve as a precursor for histamine.

JM: But together, a lot of people are saying it's an antioxidant, but it really isn't a traditional antioxidant. In other words, it's not scab, it's not adding electrons. It does it indirectly through this other component. I'm not sure if you've heard of the RCSs, the radical carbonyl species and carbonyls are essentially a carbon with a double bond to oxygen on it. And then the links on the other side chains to two other structures. So these carbonyl, they're highly reactive. And here's the thing that I just learned this morning, I was reading a study on them is that they last like maybe a billion times longer than reactive oxygen species.

JM: So the big focus is on reducing oxidative damage, but a very few people, and I'm certainly one of them, really appreciated the impact of these AGEs that you're talking about. And the ALEs, they're both important and I think the ALEs are probably more dangerous because the lipids last a lot longer than these other things, although certainly some of these agents can last for months too. But what the way carnosine works is it acts at the reactive carbonyl species. So it mitigates the damage there.

JM: It stops those the RCSs from forming AGEs and ALEs and that's how probably one of the biggest mechanisms of it. And it works at that level. So it's just an amazing. And it works for mitochondrial dysfunction, which is really attracted to me and may be one of the most important nutrients that we have to slow down the aging process. And I've just seen so many case reports of it radically improving people's health and it's so many levels

PS: And it's only found in meat.

JM: Yes, you're right. There you go. You're getting pretty healthy levels, especially in the amount of meat that you're eating, but-

PS: Yeah. And we can talk about that. I don't know if we mentioned this on the first podcast that we did. It's an observational study, but the only food that has been correlated with longer telomere length is red meat.

JM: That's right.

PS: Yeah.

JM: [inaudible 01:05:40].

PS: Yeah. And it's-

JM: I'm not convinced that telomere length, even though it's [inaudible 01:05:44]-

PS: Exactly.

JM: ... but the conventional way telemere measure I think is factually incorrect because they're just measuring, I think red blood cells or it's platelets because I can't remember what, it's not your tissue. It's these temporary cells that are circling around and it may not be the best indicator of what your true telomere length is, at your tissue level.

PS: I talk to David Sinclair, I want to get my cells tested for the methylation clock. I want to have-

JM: Oh, yeah [inaudible 01:06:14].

PS: I want to have them take a look at the [inaudible 01:06:15] clock on like carnivores cells. I hopefully, will arrange it. And it'll be interesting to see because I kind of challenged him, and when I met him in person the first time. I was like, "Let's just compare mine versus yours. Let's see who's younger."

JM: Yeah, he's pretty healthy though.

PS: Yeah, he is.

JM: I think he could, most like most of us could improve what we're doing. Hopefully you are able to connect with him later on and form a good relationship and really kind of mentor him some of the things that he doesn't aware of. Because he's really, as you mentioned to me he's very narrow but really deep, a mile deep and very narrow. I think, I forget what [inaudible 01:06:47], but it was a really accurate description is not-

PS: Brilliant guy.

JM: Brilliant is an understatement, that's for sure.

PS: Yeah.

JM: Yeah. All right, so let's go on with some of these other nutrients. Let's hit beta carotene/vitamin A and the misconceptions there.

PS: This is an interesting one as well. Many people may know that there are common polymorphisms in an enzyme called BCMO and BCMO is what we use to convert beta carotene into the retinol form of vitamin A. Retinol vitamin A is essentially two retinol vitamin A molecules from beta carotene. But in a lot of people, they can't break it down. They can't make retinol form of vitamin A. And so when you think about it's quite intriguing, the biological value of beta carotene is significantly lower than vitamin A. I forget the number off the top of my head. Maybe you remember, because I was just working on a chapter a few weeks.

PS: I think it's something like 1 to 20. It takes 20 molecules of beta carotene to equal the biological value of one molecule of vitamin A. And so when we think about it in that way, in order to get a

reasonable amount of vitamin A from plants, we would have to eat pounds or very large amounts of the richest beta carotene containing sources of food. Beta carotene is the only form. It's the vitamin A precursor, found in plants. Again, this is the operating systems concept. Retinol vitamin A is not found in plants but is exclusively found in animals, egg yolks, liver, very rich sources. Kidney is another good source of vitamin A, but beta carotene is all that is in plants.

PS: And so we have to eat 20 times more beta carotene to get the equivalent biological value of a molecule of retinol. And I talk about this in the book. In order to get the right amount of vitamin A, you have to eat something like close to a pound of sweet potatoes a day. And if you ate three pounds of broccoli earlier today to get your choline requirement, I don't know how you're going to eat another pound of sweet potatoes. And there's nothing else to even eat four pounds of vegetables, I mean like maybe-

JM: Let alone all the oxalate.

PS: I know all the oxalate. All the isothiocyanates you are going to get from all the broccoli and then all the oxalate, I mean a pound of sweet potato is going to have a ton of oxalates. We didn't really talk about oxalates. I know you've done some great podcasts on oxalates.

JM: That was Sally Norton, who is an amazing repository of knowledge on this topic.

PS: Yeah. But sweet potatoes are very high in oxalates and so it's very misleading when people say that you can get all the vitamin A you need from beta carotene. It's pretty hard to actually. You'd have to eat a lot of beta carotene and especially if you have a polymorphism in the BCMO enzyme. In that case, I am dubious that anyone eating a plant based diet can get an adequate amount of vitamin A. And so yeah, we, again, we have to get it from animal foods less we just accumulate tons of toxins and have pounds and pounds of vegetables to eat a day.

JM: Yeah. So the next one is vitamin K and the differentiation being K1 in plants and K2 in humans, which seems to have most of the benefits.

PS: Oh, it's so interesting. So K2 is menaquinone and this gets a little complicated for people because there are multiple menaquinones. There's MK-4, MK-7, MK-11 we don't really need to dig into that.

PS: Menaquinones. There's MK-4, MK-7, MK-11. We don't really need to dig into that. It has to do with the length of the side chain on the menaquinone molecule. You can get vitamin K2 in plant foods, but it's rare. It's only in fermented plant foods like natto, but generally speaking-

JM: Which is pretty high. It's one of the highest sources of-

PS: Oh, it's very high in natto. Yes, although I think natto is MK-4.

JM: Yes, it is MK-4.

PS: And probably-

JM: It's actually a derivative bacteria. I mean it's not the plants make it. It's the bacteria that make it.

PS: Exactly, yeah. It's the bacteria that make it. Humans probably need MK-7, MK-10, MK-11 all the forms of vitamin K2, and I don't think it's known if we can inter convert them. So I would argue that animal forms of K2 are necessary even if someone is eating natto. But generally speaking, K2 is not found in any plant foods except those that we talked about that are fermented from the bacteria. And the plant form of vitamin K1 is phylloquinone. Again, different operating systems.

PS: What's so interesting here is that in studies, and there are multiple studies which show this, the Rotterdam study is the study I talk about and there's another study I talk about in the book that showed the exact same thing. These are observational epidemiology studies, but the associations are quite strong. There's an inverse correlation between the amount of K2 consumed and the incidence of cardiovascular disease and calcific aortic sclerosis across thousands of patients. One study was 14,000 patients. I believe it's the Prospect-EPIC cohort, and then the Rotterdam study was 4,900 patients. And the more vitamin K2 people consumed, the upper tertile was 39 micrograms I believe per day, which is not even that high, but if you are above 30-

JM: It's not even the RDA.

PS: Yeah, it's not even the RDA. So above 39 micrograms. I wish they'd done further divisions and looked at higher levels, but they divided into three groups, and there was a clear association, a clear decrease in cardiovascular disease as you get more vitamin K2. It's such a clear correlation. And then the other one is with calcific aortic sclerosis, exactly the same thing, the more vitamin K2 the less of these cardiovascular diseases. But guess what? There was no correlation with vitamin K1. It doesn't matter how much K1 you get. No protective benefit because I think most of us are probably pretty bad at converting K1 to K2, but I think the reverse is not true. I'm not convinced there is any unique biological role for K1 in the human body.

PS: Some medical textbooks will say clotting, but I don't have any K1 in my diet and I clot just fine. So either my body's able to retroconvert K2 into K1 or the body can use both menaquinone or phylloquinone for clotting. But vitamin K supplements are often phylloquinone, which is so-

JM: I just want to bring up the point that you're a physician and you're really aware of the medical laboratories and you've done a few podcasts on this. And you've extensively done studies on yourself and others who are just eating animal foods exclusively and you've had nothing but good results. And it had no adverse consequences on extensive, really extensive testing.

PS: Yeah, we've looked at everything. I mean we've looked at a lot of stuff. There's individual variation. But I was just looking at one of my clients yesterday who was on a carnivore diet for months. Her hsCRP is 0.3. The F2-isoprostanes were very low, which is a marker of oxidative stress. There's no evidence for DNA damage with 8-hydroxy-2'-deoxyguanosine, et cetera, et cetera, right? We can look at all this stuff. They're incredibly insulin sensitive. There's no damage to the kidneys. BUN is usually normal if people are getting adequate sodium. Anyway, we can talk

about that too. But yes, I've looked at clotting is fine. There's no impairment in clotting. And so my impression is that humans can use K1 to do the clotting factor synthesis, but that it's not going to serve a very good role in directing calcium partitioning like the menaquinones do. And then in order to prevent cardiovascular disease, we need to partition calcium properly. We need to move it in and out of bones. We need to not put it in arteries. And that's the role of vitamin K2.

PS: Vitamin K2 is also involved in the synthesis of hormones, sex hormones, all these important things. And K1 can't do that. And again, this is almost exclusively found in animal foods. What's so interesting is that I went on a TV show, you and I talked about this a little bit.

JM: Doctors.

PS: I went on The Doctors, so I went on The Doctors last week, and part of their criticism was that there's no vitamin K in animal foods and I almost fell out of my chair. I was like, "How uneducated? You guys are doctors, are you sure?" But this is the problem is that if you go onto a lot of nutrition calculators, I don't know if Cronometer makes the same mistake, but nutritiondata.self.com makes this mistake. If you look at liver, it will tell you there's no vitamin K in liver. And I'm like, that's-

JM: K1.

PS: They're only looking at K1. And that's the problem that so many people are making. Any nutritionists or anyone that tells you there's not enough vitamin K in animal foods is 100% wrong, and in fact they're so far wrong, they're on the other side of wrong because there's more of the good type of vitamin K that we actually need in animal foods that we can't get other places.

JM: Well, thank you for pointing that out there. That is a common misconception and you were attempted to be humiliated on national TV, and actually you were like sabotage where there was like six people but one. That was some crazy odds that then they'd never even give you a fair chance to speak. I mean probably only had like a few minutes to talk and they probably consumed the majority of the time just berating you.

PS: It was pretty crazy. Yeah, it was pretty crazy. They just wanted to rest on their credentials and claim that I was hurting people and questioned my morals. It was pretty ridiculous. I'm sure it'll be the first of many, and I'll get back on there and set them straight, but I didn't have a chance to actually have conversations like this and explain why xenohormesis isn't really the way we think it is and why polyphenols may not be so beneficial. They were just throwing their hands up and going, "There are thousands of studies that show the polyphenols are beneficial for humans." And I'm going. "Hold on." And then they would just berate me even more and move on to the next topic. Yeah.

JM: So another misconception and one that I held and I think many other peoples don't hold is the benefit of fiber. In fact, you may probably don't know this. But when I was in med school in the late seventies, my nickname was Dr Fiber because I was a strong advocate of... Focusing on nutritionists [inaudible 01:16:28] at that time fiber was thought to be a really important and valuable element of a healthy diet, and I primarily got my information from Burkett, Dennis

Burkett. He's a Michigander MD who you discussed in the book. But you dispel these myths. And not only that, the sort of the side effect of the influence on constipation and cancer is also the microbiome and that you need these plant fibers to create a healthy, diverse microbiome. So why don't you hit that because that's a big one that many people don't understand.

PS: Yeah. In the book I talk about the non-utility. There's no benefit for fiber in terms of cancer. And I talk about many studies which show that. And then if we just briefly, we'll just gloss over the literature. I talk about it in the book in detail. There's no benefit in terms of constipation for fiber either, which may be confusing for people. And there's actually studies that I referenced in the book and quote in the book, which showed that removal of fiber actually improves, completely resolves idiopathic constipation in one moderately sized interventional randomized trials. So that's really striking. But the microbiome I think is the last bastion of hope for fiber advocates. And I'm going to... I mean I don't know, It's so interesting to think about the reason people think fiber is necessary for the microbiome is probably twofold. The first is because people will say you need plant fiber to have a diverse microbiome and we hear this word diversity thrown around a lot.

PS: What people are referring to is the alpha diversity of the microbiome, which is alpha diversity is an ecological term. It's a zoological term, which means how many species are in a specific area. So it does appear there is some evidence to suggest that a diverse microbiome is a good thing, but not always. And it's more complicated than that because you can have a diverse microbiome that's full of a bunch of criminals like proteobacteria or gram negative aerobic organisms, which are not good, but your diversity is still high. So you can have a neighborhood with lots of different people, but a lot of them are criminals, or you can have a neighborhood with lots of different people from all over the world who are all really good people and all work together and have beautiful potlucks and everything is good. So that's the first problem with using alpha diversity as a measure.

PS: The second problem with this premise or this assertion by people who will advocate for plant-based fiber is that it's just not true. They're looking at epidemiology studies and they're looking at a series of associational studies that show that people who live in rural parts of the world have a higher microbiota diversity than people who live in urban parts of the world, and then they're correlating that with the number of foods they eat. Again, this is not an interventional study. This is just observational epidemiology and it happens over and over, but there can be many confounders here. And what's so interesting to me, and I cite these studies in the book, is that when we actually do the interventional studies, which are much higher value evidence than epidemiology, adding fiber to the diet does not change alpha diversity and removing fiber from the diet does not decrease alpha diversity.

PS: So there's actually... This is amazing. I'm so happy that Harvard did this. I don't think they intended it to be used for this purpose, but there was a week-long study done at Harvard where they put people on a carnivore diet, like a pure animal-based diet, and another group on a pure plant-based diet. And at the end of a week they looked at the gut flora and there was no difference in the diversity between the two groups. So a carnivore diet did not decrease alpha diversity in these groups at all. In fact, the carnivore diet increased the beta diversity, which is another measure of diversity. So diversity actually increased.

PS: And if you look at the interventional studies with fiber, there's no increase in alpha diversity when we eat more plant fiber. But this is just like the Xenohormesis concept. I think 98% of the world believes that the way to get a healthy gut microbiome is to eat more types of plants and that is just false. It's never been proven in an interventional setting. It's based on epidemiology, and like we're suggesting here, the interventional studies do not support it.

PS: I think that the real way to get a diverse microbiome is something that we're just missing here. And it's our environment. It's probably the fact that we're swimming in lakes and rivers, we're outside breathing fresh air, we're in the woods, we're touching dirt, we're touching our pets. That's how we get a diverse gut microbiome. And then the other thing that people are often overlooking is, are there two arrows of causality here and which direction are they going? And by that, what I mean is many times people assume that it's a gut microbiome that's creating health or disease in the host, but the arrow of causality goes the other way as well. It's possible that someone could be sick and that could create a negative diversity or a decrease in diversity in their gut microbiome.

PS: So we can see correlations between diabetes and metabolic syndrome and low diversity in the gut. But again, we don't know what direction the arrow of causality goes. It probably goes in both directions. Certainly, I think the gut microbiome affects the host, but the host also affects the gut microbiome. And if we are sick, that can negatively affect our microbiome. So I think a lot of times we're seeing low diversity. It could be something that's completely separate from the gut. It could be inflammation from somewhere else. I mean a lot of inflammation probably starts in the gut, but it's this crosstalk going back and forth, and it's being radically oversimplified to always assume that it's the microbiome that's affecting the host exclusively. There's got to be this stuff going both ways for sure. But the idea that we need plant fiber and a diversity of plants to have a diverse microbiome has been disproven time and time again.

PS: I've done my Onegevity and I'm in the 90th percentile for gut diversity. And many other carnivore dieters have done this as well. And I've seen some of the highest diversity scores in people not eating any plants. So that completely like... We need to publish a case series and just blow this concept out of the water. And then I believe studies had been done in the Hadza as well. And the Hadza have an incredibly high alpha diversity. But they only eat three or four foods at any one time because there's only three or four foods in season. Over the course of a year, they might shift their foods and they certainly eat some plant foods now because they've been marginalized and they can't hunt animals as much as they would like. But the Hadza are not out there right now eating a hundred types of plants. There's just not that many plants in that region of Africa. But they have a high alpha diversity.

PS: So this notion, I think it's one of my missions in the next few months to continue to have conversations about this, and I'm definitely going to have more conversations with people on my podcast and challenge this notion that we need plant fiber for high alpha diversity. That's not true at all. And then at a clinical level, I'll just add this, the amount of improvement symptomatically that people get from a carnivore diet is strongly suggestive that the removal of fiber is not hurting the gut because basically what I see is people completely resolving the symptoms of small intestinal bacterial overgrowth, gas, bloating, constipation among others. There are published case reports of resolution of Crohn's, ulcerative colitis and many other bowel issues. If the removal of fiber were so catastrophic for the gut, we should all have Crohn's or inflammatory bowel disease

or we should all have major gut issues when we're not eating plants and we see the complete opposite in the community.

JM: Yes indeed. Well that was just terrific. Absolutely terrific. So it was a great summary in the whole topic. Your insights on the backward connection between a person's health and their gut flora, which I've never heard anyone address is, is probably on target and it makes perfect sense. I bet it's a novel insight. So thank you for sharing that. So on the tail of what you said about the autoimmune disease, I want to jump into that too because this is sort of an artifact of some of these molecules and phytoalexins that are in the plants -, and the absence of them when you stop eating the plants, it seems like almost every single autoimmune disease improves. And that was initially how you yourself went into this not only for your personal experiences, but on the tail end of Mikhaila Peterson is really I think maybe one of the leading people who've catalyzed it in the modern era as the carnivore diet is Mikhaila. And she's an unbelievable story. And if you really want to go deep in this, listen to her two to three hour podcasts with Joe Rogan few years ago.

PS: I did a podcast with her on mine too. Yeah.

JM: I didn't know you did. Yours is probably better, but amazing story and this woman, my heart goes out to her. She went through so much misery, but she got better on it as did you. So why don't you talk about the impact of carnivore on autoimmune because I think it's a fascinating concept?

PS: Yeah.

JM: In my view, I think it's almost medically and criminally reprehensible malpractice if a condition does not put a autoimmune patient on a carnivore diet.

PS: I would agree with you. And that's my goal is to help people understand that it's a safe intervention that we're doing elimination diets now. I was on another podcast recently and I said something that every once in a while you say something and you're like, "Oh man, that came out good. I said it good." Not all the time, but I said it good. And when I sat on that podcast, I've kind of echoed this sentiment in the past, but I really can't wait for the day that modern mainstream Western medicine just realizes that food is the biggest lever in health and disease, and that it doesn't matter if somebody wants to do a vegan diet or a carnivore diet or a paleo diet.

PS: The most important thing for me is that they make an intentional choice with their diet and that they try and that they use diet as a lever. I personally believe strongly that a carnivore diet or something like a carnivore diet that gives attention to plant toxicity is going to be the most efficacious intervention with food. But I am just excited about Western medicine, realizing that food is the biggest lever and that often the elimination of food is what we need to do to leverage or to move autoimmune disease.

JM: Let me just butt in here for a bit and just mention that, it's the point I wanted to make earlier, and it's not just the food is a biggest lever but also the time that you eat the food because you have to eat the carnivore diet. But if you're eating it for 18 hours a day, I can guarantee you you're going to run into problems. So it's not so much the food you eat too, but it's when you eat that food. So

it's the combination that forms this incredible magic that just just moves your whole biology towards health.

PS: I couldn't agree with you more. I totally agree with you and thank you for highlighting that. Yeah, and people should just know that I definitely think about time restricted eating as well. Yeah. And I'll talk about that in the book also. But we make an intentional choice for diet. We do an elimination and that's how we affect autoimmune disease. And some of the things I've seen are striking. I can't even imagine. I would never have imagined I'd be here two years ago, honestly. But I got an email this morning from a guy who said, "Hi Paul. I sent you a message on Instagram. You replied with some messages and I tried the carnivore diet and my psoriatic arthritis has gone and I've been off Humira for a few months."

JM: That's hard. It's a hard one to treat.

PS: Right? I can't even tell you how many cases of psoriatic arthritis I've seen go away from it though. There are multiple stories on my Instagram. I've posted lots of testimonials from people who had bad plaque, psoriasis, fibromyalgia, eczema, asthma, I mean lupus. On Instagram, I'm quite good friends with some people called the Strong Sistas, and it's spelled S-I-S-T-A-S. So if people want to look them up on Instagram, I think it's strong.sistas on Instagram. They're an amazing set of women who both were diagnosed with lupus. They both had positive ANAs. I believe Sarah's ANA was 1 to 640 and Ashley's was about the same in terms of the titer. So they had very positive ANAs, were diagnosed with undifferentiated mixed connective tissue disease. And now, I mean, we haven't repeated Sarah's labs, but neither of them has any symptoms anymore on a carnivore diet. And Ashley's titers are normal. Her ANA titers are normal. They're not even elevated anymore. She doesn't have anti-nuclear antibodies anymore. So it's like, "Wait a minute, this is really cool."

PS: Now I must say that I can't claim that the carnivore diet cures 100% of people, but it's pretty darn effective. It's a really powerful intervention. I think for some people, there's other things going on, GI dysbiosis or gut infections or heavy metal toxicity. Who knows? But of all the things that I've seen-

JM: Vitamin D, emotional traumas.

PS: Yes, yes. Many things. Yeah, but it's a pretty darn effective intervention. And generally, this is what the book is about. Hey, look, plants have toxins. Eating animal foods is safe. Don't fear them. If you're sick, if you're not kicking as much bud as you want to, then try the carnivore diet especially if you have an autoimmune disease. It's incredible. I mean it totally resolved my autoimmune disease and I've seen it happen for people over and over and over. It's pretty cool. I think it's going to change medicine. Just today on my Instagram stories, I posted a bunch of stuff. People who have been writing to me about SIBO and other GI conditions and other autoimmune conditions, RA, rheumatoid arthritis, and even if the carnivore diet only helps 60% of people, there's nothing else that we will ever have that's that efficacious without side effects, right? This is far and away the most effective intervention I've ever seen them.

JM: Yeah. I developed somewhat of an expertise in treating RA in the late eighties when I was using a derivative of Thomas McPherson Brown's protocol, he's since passed many years ago, but it was using minocycline to treat a mycoplasma infection, and it was fairly effective. But then I eventually evolved the protocol to stop using that and just using these lifestyle adjustments. And at the time, things like vitamin D and emotional issues and just optimizing diet, insulin resistance. But I'd never really fully appreciated the importance of limiting any plant material. Even that without the carnivore I was able to get 70% to 80% of people better. It would've probably been 95% with the carnivore.

PS: I don't doubt it.

JM: Yeah. So let's go to another surprising point in the book, which is really nothing less than shocking from my perspective because the Blue Zones is a commonly used defense the plant-based approach because it's going to increase your longevity, and boy do you decimate that myth. So I'll let you have at it, man.

PS: This was one of my favorite parts of the book to write. So if people are not familiar, the Blue Zones are five regions of the world that were sort of proposed by Dan Buettner and some of his colleagues in I believe the 90s in National Geographic. And they are a Ikaria in Greece, Sardinia in Italy or Sardinia, yeah, Loma Linda, which we can talk about in detail because that one's quite interesting one, Okinawa, the Nicoya region of Costa Rica. So there's five. And what's so interesting about this is that I guess this is just what we do as humans. I mean people are probably familiar with the work of Ancel keys and the Seven Countries Study, which was where the diet heart hypothesis regarding saturated fat and cholesterol was born.

PS: What we know about Ancel Keys is that he did a pretty good job at cherry picking seven studies that fit his data, and unfortunately Dan Buettner has done that as well, but not even a very good job of it. Not only did he choose, he just cherry picked five places in the world that have exceptional longevity. He didn't actually really look at what they were eating. He just claimed to think he knew what they were eating. The first part of that criticism is that there are many regions of the world where people have exceptional longevity that were left out. Hong Kong has the largest life expectancy in the world right now or 85.6 years.

JM: I think he quote 84.3 in the book with eating a pound and a half of meat. I actually fell on the beach because I was walking when I read it. I couldn't believe it. A pound and a half of meat a day.

PS: They have the second or third, maybe the highest consumption of meat in the world or the third highest consumption of meat per capita in the world. And they have the longest life expectancy. And life expectancies are different whether you're looking at countries or provinces. Hong Kong isn't always included because of idiosyncrasies and how they're defining it. But there are many regions of the world where people have exceptional longevity and they eat lots of meat that he just left out conveniently. But let's just go through some of the ones that he did include.

PS: So the first one is the Nicoya region of Costa Rica. Well then acquire region of Costa Rica is only a region of longevity for males. I don't know why. It probably has to do with genetics and we talk about that later, but it's only males that have exceptional longevity in the Nicoya region of

Costa Rica. But if anyone has ever been in the Nicoya region of Costa Rica, they know that both men and women treasure meat and they eat more meat in the Nicoya region of Costa Rica than the average person in Costa Rica and they use more animal fats to cook it in.

PS: So they don't use the vegetable oils as much because I guess they're more expensive and they're just going to use the animal fats, which many people would say, "Oh you don't want to use lard." But they're just using natural lard to cook things in and they're eating more meat than the regular people in Costa Rica. And then the males are living longer. That's a region of longevity for males. So I don't know why that one got included in the thing.

PS: And then in Sardinia in Italy, they treasure meat. I mean if you look up Sardinia, it's just out of this world. They have whole feasts of meat and there's something in Sardinia called Sarda pig. They have a delicacy named after their conflict named after where they live because they treasure meat so much.

PS: The other one that I think is really interesting, let me think. We had Ikaria, Sardinia, Nicoya region of Costa Rica. Okinawa is a very interesting one as well. So Okinawans it's only since the 1930s and 1940s and colonialism in Okinawa that Okinawans have been eating more vegetables. They generally ate pork for their... That's a pork-loving country and there is a lot of animal food eaten in Okinawa even still to this day.

PS: And then probably the most interesting one is Loma Linda. So Loma Linda is in Southern California. There's a big Seventh Day Adventist population there. Within the Seventh Day Adventist's population, there are a number of things that are advocated for. They suggest avoidance of smoking and excessive, any drinking and then they don't believe in eating meat. People may have heard the previous podcast that I did with Gary Fettke. I talked about this. As part of the Seventh Day Adventist's religion, they believe that meat creates carnal desires in humans, which is probably true because it allows us to have healthy hormone levels, right?

PS: And so that's a fascinating story. People should listen to the podcast I did with Gary Fettke and the story of Kellogg's. So Kellogg's, the cereal company Kellogg's was founded with the hope of quelling carnal desires in humans. And so it's a very effective way to completely destroy your hormonal balance by eating processed plant foods. That's how you do it. So if you want to have a chemical castration and you don't want to have hormones or libido, you can do that by eating plant foods. That's why it sounds like hyperbole. It sounds like I'm making this stuff up. But that was the original intention of the Kellogg's cereal company founded in Battle Creek, Michigan. And it was funded by the Seventh Day Adventists and the Seventh Day Adventists continued to believe.

PS: I think from a place of genuineness, I don't think it's malicious. I think it's a sort of religious belief that humans are too carnal. Well, we all have hormones. This is just how we are as humans. But they believe that if we eat a vegetarian diet, it will control our carnal desires, which it probably will because our hormones will tank in a negative way. The Seventh Day Adventist region of California is a zone of longevity. They live about 7.3 years longer than the average Californian. But what's so interesting, this actually came up on The Doctors, is that the Mormons, California Mormons also live seven-ish years longer than the general population, but they don't shun me to all.

PS: And so it's probably not the shunning of meat in Loma Linda or in any of these other blue zones that's leading to longevity. In the case of Loma Linda and the Mormons, what they have in common is that they don't smoke and they don't drink and they have a tight community. And that will be our takeaway from the blue zones eventually.

PS: But the most interesting thing about Loma Linda for me is that there's a very striking study looking at the sperm quality. I don't know why they did this study, but I'm so glad they did. They looked at sperm quality of people in Loma Linda. And the ones who are vegan, vegetarian had abysmal sperm quality. They measure this in terms of numbers and motility. There was a clear differentiation that the less animal foods in Loma Linda ate that the worse the motility and the numbers of their sperm war. And so in the book I make this kind of glib comment like it makes you wonder what part of Loma Linda is actually blue. So we'll just leave it at that. But it's just crazy to me that these are the blue zones, Dan Buettner? This is the region they have sperm that's completely impaired. They're clearly not getting the nutrients they need. That's a blue zone? Really? That's crazy.

JM: So the blue zones is what many plant-based people will use to justify their choice with respect to longevity and health. But the other ones out there are loads of studies purporting that plant-based diets are far healthier than ones that consume animals. So I'd like you to discuss that because I think it's a really important concept and the issues such as healthy user bias, these are our epidemiological studies that that are essentially correlation. They're not... What is it? The interventional trials.

PS: Yes.

JM: That Are more definitive and you can make causal causal conclusions from which, but you can't from these correlation studies. So why don't you discuss that? Because I think this is really, not a strategy, but a story and a line of reasoning that many plant-based people use to justify their choice.

PS: This is probably one of the most important things I talk about in the book, and I think this will clear up things for people in general. And this is probably going to be a large part of my crusade moving forward is to just educate people about types of studies and what they are in medicine. The majority, in fact I think that like almost every single study that is used by the plant-based movement to support their cause is epidemiology. And as you suggest, epidemiology is not interventional. It's observational. It's not an experiment. It's an observation. They take a group of people, and they observe them for 10 or five years, and they give them a survey at the end of that time, or they give people a survey and they look back on their health. But it's observational. It can only tell us correlation. It can't tell us causation.

PS: It's valuable science. But there's a great website that I referenced in the book. I didn't actually show you the graphics because they're not in there yet, but it's called spuriouscorrelations.com. I would encourage people to go to that website. On the website this kind of funny guy has, there's actual real correlation between the per capita consumption of cheese and the number of people who die by getting twisted in the bed sheets at night from 1995 to 2000 in Michigan, and the

correlation is like 0.93. It's a very strong correlation between cheese consumption and death by getting tangled in the bed sheets and things like this. The number of movies that Nicholas Cage has appeared in in any given year and the number of deaths by homicide in Illinois or something. It's just so silly. You can make correlations between anything that don't have a causal relationship.

PS: But what we see here is that, as you're suggesting, there are studies epidemiology, observational studies that show a correlation between fruit and vegetable consumption and improved health outcomes. But does that mean that it's the fruit and vegetable consumption that's causing the health outcomes? We can't tell. It could be or it might not be. The problem is that in nutritional research, it can generate a hypothesis, but very seldom is that hypothesis tested with interventional research. It has been sometimes, and we'll talk about that, but the preview of that is that when we actually do interventional studies with vegetables, they don't look very good. And we do interventional studies with meat. It doesn't look inflammatory or damaging for humans at all. So we'll get to those.

PS: But in terms of epidemiology, what's going on here is this. Just think about what we've been told about meat and animal foods and fat for the last 70 years. Well, probably because of good old Ancel Keys in the Seven Country Study in the 1960s, we've been told that these foods are bad for us. And so when humans hear that something is bad for us, there's two groups. There's two reactions. One group of people says, "That's bad for me. I'm not going to eat it." And those are the people who are doing all the other healthy things in their life, right? Because those are the people who listened to health advice. They're also exercising. They're walking. They're playing tennis. They tend to be more affluent. They go to the doctor more. They have more access to healthcare. They smoke and drink less, and they spend more time with family. This is what we know about people who listen to health advice regardless of what the health advice is, they have a different lifestyle.

PS: There's another group of people who are like the James Dean rebels, right? They hear health advice and they go, "I don't care. A hamburger tastes really good. I'm going to keep eating it." And those are the people who are much less likely to exercise and do all those other healthy things, right? So basically we have this real dichotomy between people who listen to health advice, people who don't listen to health advice. And what we know is that people who don't listen to health advice don't do anything else that's healthy or they're much less likely to do that. They're more likely to be obese, to ride motorcycles, to do other dangerous behaviors, to smoke, to drink, to have less family ties, to be less affluent and to be less healthy overall. And then the people who listen are going to be healthy. And so healthy user bias is a bias that cannot be controlled for in these epidemiology studies.

PS: What we're probably seeing here is that people who eat more fruits and vegetables are also doing other healthy behaviors, right? They're exercising. And this is the takeaway from the Blue Zones too. Yeah. People in Loma Linda live longer because they don't smoke or drink. The Mormons live longer because they don't smoke or drink. The people who can do the exercise, be in the sun, those are healthy behaviors as well. That's going to create longevity. But when it comes to diet, we really can't tell because that's a very complicated thing.

PS: And there's one study in the book that I mentioned that really, really drives this point home. It's called the UK Shoppers Study. People Google UK Shoppers Study, they'll probably find it. This was done in Britain, and they compared the the death rates, the standard mortality ratio of vegetarians to the general population. And the vegetarians live longer. They had a lower standard mortality ratio to the general population, right? But then they compare the death rate of vegetarians to other people in the population who ate meat, they were omnivores, but these people did healthy behaviors. So they were actually able to compare two groups of people who listen to health advice and do healthy behaviors and they had equivalent death rates equivalent.

PS: So it's not the exclusion of meat probably that's causing these health outcomes to look good. It's the other things they do. This is healthy user bias. But when people are hearing this in the media, if they say people who eat fruits and vegetables live longer, dah, dah, dah, dah, dah, it's often healthy user bias. So what do we do? We generate hypothesis. We go back and we test the hypothesis. It's just very hard to test that hypothesis because how do you do a study long enough where you're giving some people more fruits and vegetables? So what has been done is a series of about five studies that I talk about in the book where fruits and vegetables were removed from the diet. So this is the interventional stuff.

PS: Fruits and vegetables were removed from the diet. So this is an interventional study. It's not observational and so in these studies they range in length from four weeks to 11 weeks and they had about 30 to 60 participants per study. So we're looking at over 200 people total and what they did was they removed fruits and vegetables completely from one group and let the other group eat like a pound and a half of vegetables a day. So fruit, vegetables, they ate a lot. These were not like weanie vegetables. This wasn't just like iceberg lettuce. This was like a broccoli, cauliflower, Jerusalem artichokes, carrots, apples. They were eating vegetables that people would consider to be quite valuable vegetables and then the other group had no vegetables. They completely got rid of their vegetables and what do they see? At the end of four weeks they looked at a number of things.

PS: They looked at oxidative stress, they looked at inflammatory markers and they looked at markers of immune activation and what do they see? They were completely the same between the two groups. Meaning that when we remove fruit and vegetables, these are fruit and vegetable depletion studies. When we removed fruit and vegetables, there is no detriment. There's no change. There's no benefit to having them in there. There's no change when we take them out in terms of oxidative stress, [F2i suprastin 01:46:15] , DNA damage, 8-Hydroxy-2-deoxyguaonsine, immune activation, markers of immune populations.

PS: So just when we remove vegetables, we don't see any change in the population. So how can vegetables be so beneficial if we take them out and we don't see any, there's no difference. We didn't get more inflammation or more oxidative stress or any of these things. So we need to do more of those interventional studies and the flip side is that a lot of these plant-based advocates will deride meat or they'll vilify meat saying that meat is bad for us and they'll point to similar epidemiology, which has the unhealthy user bias.

PS: Again, this is surveying people, this is showing people who are the James Dean types, who are doing bad things with their diet and also not listening or doing any of the other good health

behaviors. So in those people they can show that the people who eat more meat do worse. But again, it's unhealthy user bias. It's probably that those people who are eating more meat are you getting more meat with milkshakes, processed foods, bread, sugar and all these other thing, soda, because they not listening to health advice. It's all about the narrative in the culture and how that affects us as humans.

PS: What I'll say with regards to that is that when we do interventional studies with meat, there's a great study in the book, probably one of my favorite studies in the whole book. They had people replace carbohydrates in the diet, which would be plants, with half a pound of meat per day. So they had people add eight ounces of meat per day and they have replaced plants with that. At the end of the study period, which I believe was for six weeks, inflammatory markers went down. When we do interventional studies with red meat, we clearly see meat is not inflammatory. End of story, end of story.

PS: The last thing I'll mentioned with epidemiology is that if we look at countries where there hasn't been the same narrative around meat, where we have not been told that meat is bad for us, this would be like Asia. In Asia, the narrative around meat is that people who eat more meat are affluent. Meat is a sign of affluence and in those countries when we do the epidemiology, plant-based dieters never tell you about this. The people who eat the most meat have the lowest rates of heart disease. The men have the lowest rates of heart disease and the women have the lowest rates of cancer. So the epidemiology is often sold to us as half of the story and really it's more about the healthy behaviors and the epidemiology than the actual foods. We have to go to the interventional studies to look at the foods. When we do that, we see that vegetables don't have a benefit when we remove them and that we add meat, it's not inflammatory. So hopefully that made sense. It's such an important concept because so many of this, this narrative is so misleading.

JM: So another point that you didn't mention in there, but I think leads into the next and last issue I'd like to discuss with you is the connection between meat eating and high cholesterol levels and an acute risk of heart disease and the actual high cholesterol, hypercholesterolemia, and you bring up a fascinating point and discuss it a number of times in different podcasts that high cholesterol levels are a nonissue if you have insulin resistance, which relates to some of the other epidemiological observations, just as long as there is a person's eating meat, it may be connected more to the fact that they're eating highly processed foods and grains that are causing insulin resistance, which by the way, as you accurately mentioned in the book, is essentially present in about 85% of the population.

JM: So it's that that is the more important variable than the impact on a cholesterol. But why don't you talk about that because I think it really is another fascinating point that many are confused on.

PS: The cholesterol story is so interesting. You summarize it very well. The takeaway with regard to this, and I'll unpack it here in a moment, but the takeaway is that LDL cannot be interpreted in a vacuum, nor should total cholesterol be interpreted in a vacuum. But usually we're looking at LDL now because we can fractionated easily, but we cannot interpret LDL in a vacuum and we must interpret LDL in the context of insulin sensitivity. If we really look at the data, I break this down, this chapter, that's a long chapter. I break that down in detail that in the setting of insulin sensitivity, LDL is not an issue at all and probably more LDL is protective. So let's just set the

frame here. Many people have only heard of LDL as "bad cholesterol," but what would evolution have created something in our body that is uniquely bad for us? No.

PS: Clearly everyone can.

JM: It doesn't make any sense.

PS: It doesn't make any sense. Like why would we just have this horrible thing in our body? It's bad for us exclusively.

JM: Well, because need to have drug companies tell us.

PS: Exactly. Right. What we know is that LDL has very many valuable roles, but nobody ever talks about this, but I extrapolate on it. I explain it in the book. LDL has an immunologic role as in addition, it's a nutrient delivery system. I talk about it as buses and I described the whole liver protein system, but LDL delivers cholesterol and triglycerides to ourselves.

PS: Cholesterol is used in every single membrane of every single cell in our body. It's a precursor for steroid hormones. So if we want to be healthy men and women with libido or glowing skin or muscles, if we want testosterone, if you want to obviate and have babies, we need cholesterol.

PS: I'll just mention for people just so they know, I should have said this in the beginning, cholesterol specifically refers to a steroid backbone molecule, the sort of broad definition of cholesterol in the blood, is a summary of all of the cholesterol moving in lipoproteins. So cholesterol is not the same as LDL.

PS: LDL is low density lipoprotein. It's a bus that moves out of the liver. It comes out of the liver as VLDL and then IDL and then LDL and LDL is circulating in the body, but there's so much hubbub now about high levels of LDL being bad for us, and this is really false and the first thing that I was describing there is that LDL is a valuable particle. It's also in addition to delivering nutrients throughout the body, which is its main role, it's also involved in the immunologic response and that series of studies that I talked about in the book was so striking when I found it. In rats, if they infuse more LDL into rats, they survive much longer when they're given endotoxin, which is LPS, it's a very inflammatory component of a gram negative bacterial cell wall. They can even infuse the rats with MRSA or staph aureus toxin, which is quite toxic and the rats live longer when they have more LDL.

PS: LDL is also known to negatively affect quorum sensing between bacteria. This is sort of the bacterial cellular network, so that when bacteria is trying to invade our body, they talk on their cell phones, they're texting on their cell phones and the LDL severs that so that bacteria can't communicate and decide when they should invade us and divide, right?

PS: So LDL has clear immunologic rules and there are, there's other experiments in mice where they take out the LDL and those mice die fast. Those mice die really fast when you give them endotoxin or staph aureus toxin.

PS: I talk about this in the book as well. There's actually a genetic condition in humans, Smith-Lemli-Opitz Syndrome, I believe, where there's a congenital deficiency of LDL and those patients, human patients are very susceptible to infectious illness that can be rescued by giving them sort of infusions of LDL. So there's actually a condition where we're giving people more LDL and it's helping with their immune function.

PS: So LDL is a valuable molecule. So let's just pause there for a moment and think about this. Why would a molecule that's clearly indispensable in humans and valuable also be killing us? That doesn't make any sense. But let's just move on and keep talking about it.

PS: The main hypothesis advanced by, again, 98% of the people out there today is called the Response to Retention Hypothesis. This is the idea that LDL moves in the blood and then gets deposited or moves into the subendothelial space, which is just below the endothelium of the blood vessel, it's the intima and then gets stuck there and that forms a plaque.

PS: Now parts of this theory may be true. We're still trying to figure it all out and there are experiments in mice where they get rid of a part of the apoB molecule on the LDL. So LDLs lipoprotein particle that's identified by apoB-100 and the apoB-100 protein in the LDL particle, again, this is a single lipid membrane particle that is encasing cholesterol and triglycerides, that apo B-100 protein stuck in the membrane, binds to proteoglycans in the intimal space. When they get rid of that, they can reverse atherosclerosis in mouse models. So it does appear that LDL is binding to the proteoglycans in the intimal layer.

PS: But one of the things that I talk about in the book that I think is so interesting is that it's not about how much LDL is in our body, it's about how much LDL gets stuck in the sub endothelial space and what determines how sticky LDL is and how sticky our subendothelial space is, insulin resistance, insulin sensitivity. So there are a number of studies that I talk about in that book where we can see that when we are insulin resistant, there are different proteoglycans laid down in the subintimal space and the LDL accumulates a molecule called APOC3 and becomes more sticky.

PS: So it's almost like, I talk about it in the book, throwing a bunch of tennis balls against the wall. They're not going to stick, right? LDL normally is meant to move in and out of the sub endothelial space. This is the normal action of LDL. It delivers nutrients to the intima because the intima doesn't have a direct blood supply and needs to get nutrients and the cells in the intima need cholesterol and triglycerides as well. LDL moves in, it's like a bus. It goes into a little neighborhood, it comes out of the neighborhood, but if the LDL is sticky and the intima is sticky, then it gets stuck there and insulin resistance can cause it to get sticky or damage to the endothelial layer.

PS: Damage to the blood vessel can cause that to get sticky and it pulls in more LDL. Insulin resistance can probably also cause damage to the endothelium, but it has to be sticky. If I coat the tennis balls in Velcro and I coat the wall and Velcro and I throw the tennis ball against the wall, it's going to get stuck, right? That's how it works here.

PS: So I want to share one other set of studies here, which I think really illustrate this well. This is really fascinating. So what we're looking at here, can you see this okay?

JM: Sure. Yeah.

PS: So let's look at this one. In the book I talk about this. The Framingham Study is one of these studies that shows, hey, look, the more LDL you have, the higher your increased risk of coronary artery disease. This is across the entire Framingham cohort. This is about a 5,000 person epidemiology study from New England, but what's so interesting about the Framingham-

JM: The longest epidemiological studies.

PS: Yeah. Well you'll see here that you know LDL at a hundred you've got an increased risk, 2.5 relative risk and and you go up to an LDL of 220 you've got a relative risk of eight. So they're saying, "Hey look, this guy is wrong. Doctor Sal, this guy doesn't know what he's talking about."

PS: But look at this graph. So this is that same data from the Framingham Study stratified by HDL and you see two very distinct patterns regarding LDL and risk of coronary artery disease. What we know about HDL is that the HDL number is a very good proxy for insulin sensitivity and that low HDL, sometimes, the majority of the time, low HDL indicates insulin resistance and a high HDL indicates insulin sensitivity. One of the things that I use in my clients to get a good sense of insulin sensitivity is triglycerides to HDL ratio. I think if you looked at that, you would see the same pattern. But this is the exact same data that I showed in the first graph and what we see here is that in people who have high levels of HDL, meaning that they're insulin sensitive, there is essentially no correlation between the levels of LDL and the relative risk of coronary artery disease.

PS: But conversely, people who have low HDL insulin resistance, there's a very strong correlation between levels of LDL and levels of heart disease. So the takeaway here is that it's not about how much LDL is in your body, it's about how much LDL is getting stuck because these people are insulin sensitive. They can have tons of LDL, 220 milligrams per deciliter. It's not getting stuck in the endothelium it's not causing heart disease. When we stratify the data, and I think that if we did this with all of the studies that had been done, all of the epidemiology studies that have been done correlating LDL with cardiovascular disease, if they stratified them by insulin resistance, we would see a totally different pattern emerge. Yeah.

JM: Yes, indeed. So thank you for sharing that and just to make a point of distinction, the LDL is not the total cholesterol. So that's significantly lower in the 220 you quoted might correlate with a total cholesterol of over 300.

PS: Yeah, it could.

JM: Well, thank you for all that information and I think the concluding components should be your tips and recommendations on how to implement this, which is the nose to tail strategy that you recommend. Also, just to reinforce the timing on this, because in our initial discussion, I was concerned about the mTOR activation and you helped me understand that it was insulin activates mTOR far more than that. But certainly the leucine content of meat will activate it somewhat and if you're doing a chronically, it's a problem. But when you restricted it isn't.

JM: The other thing is is you are not insulin resistant if you're not eating carbohydrates. It just is not going to happen and you have really high ketones and your [APK 02:00:11] is elevated and you're excreting your certain ones and all these powerful metabolic pathways are being active. So how do you implement this?

PS: So yeah, I go into detail about this in the book. It's, I think about it the way that our ancestors would have eaten meat, the way that indigenous people eat animals I should say. They eat the whole animal. They eat nose to tail and this means eating animal meat but it also means eating some animal fat from good animals. I only recommend, or I strongly advocate eating animals that are grass fed, grass finished ,the best quality we can get, supporting regenerative agriculture farms like White Oak Pastures for sure.

JM: I've been to Will Harris' farm.

PS: I'm going in December. I'm super excited.

JM: Actually comment too on the dangers of plant oils, not necessarily highly refined processed oils, but like coconut oil would be one or MCT oil that can be used and would seem to be synergistic with a carnivore approach, but technically certainly plants.

PS: We can talk about that too. What I talk about with in terms of how to implement it, I recommend that people think about eating some organ meat and this may be foreign to some people or if people are from other countries, they may have already embraced organ meats. These are things like liver and kidney are probably the first two to start with and those in addition to animal meat and animal fat will really change the nutritional composition of the diet.

JM: Let me give a little tip here. I'm going to start with interrupting. But liver, as liverwurst, which liverwurst is about 50% liver is a much more palatable cooked and it's, that's the way to do it. That's what I do. I've ordered like 25 pounds of that stuff and so they have it pretty much every day.

PS: It's good. Liverwurst is a great way to get liver, but you know a carnivore diet, I guess we haven't really defined that. It's just animal foods, so no plant foods, but depending on people's tolerance and what they like, I think it's reasonable. You're not going to find toxins like you do in plants and animals because animals can run away. They don't make the same plant toxins and so you can include seafood, you can include eggs, you can include chicken, Turkey, pork, beef. I think the red ruminant meat is probably the best thing to have in your diet, but eggs are a valuable component.

PS: People could include dairy if they're not sensitive to it. You might want to think about organic dairy. You might want to think about A2 dairy versus A1. Casey and I talk about that in the book. That would be things like goat and sheep milk or specific A2 dairy from cows rather than A1 dairy.

PS: But you can include a lot of foods that are not just steaks and the more foods, especially things like eggs, liver, kidney, organ meats, on the last podcast we also talked about ancestral

supplements. These great adjunct because they make freeze dried components of these organs that you can get the organ meat if you're not used to eating it. But I would favor the real organ first. So that's how you do it.

PS: In terms of basic macros, people can go to my website which is carnivoremd.com. I've got a carnivore diet pyramid there which has a lot of this laid out in it that you can download and in terms of macros, what I recommend for people is thinking about how much protein you want to get in a day. I think something, we may differ a little bit on these recommendations. I generally recommend 0.8 to one gram of protein per pound of lean body weight and then a reasonable amount of fat to go with that.

PS: Then some organ meats and eggs and a lot of salt. I think that it's important to get a lot of good salt since the electrolytes don't get out of whack. I go into detail about all of that in the book, in chapter 12 and chapter 13 and I've got meal plans and everything in there.

PS: So the last piece that you mentioned there is the the vegetable oils and I recommend that people avoid these when they're doing a carnivore diet because of something called oleosins. Even in lipids, even in oil, we can find proteins which can be immunogenic and people could reintroduce these just like they would an elimination diet later. But if we really want to see how our body does with no plant compounds and you want to get rid of the all the plant oils because of oleosins. Oleosins are proteins in lipid droplets, in plant oils, meaning that there could be, there are proteins in olive oil, there are proteins in coconut oil that can still trigger an immunologic reaction.

PS: I really believe that animal fat is more nutritious in general for people.

JM: Certainly with micronutrients and the other concern about olive oil, which is generally recognizes, I mean very few people would dispute that it's not one of the healthiest oils out there, but what they failed to consider, at least from my perspective, is that you have to have it in very small doses because it's loaded with Omega-6. It's healthy, Omega-6 but it's still Omega-6. You have to go have an eight nine tablespoons of olive oil and your 6:3 ratio is going to be 20:1.

PS: Yeah. Yeah. I think that if you look at what happens when you eat animal fat from pastured animals generally, I mean I just saw somebody yesterday who had an Omega-3 to Omega-6 to three ratio that was like 3.5 and they were just eating animal foods. So you can obtain really good 6:3 ratios.

JM: I think that goes back to what you were talking about the K1 versus K2 analysis and nutrition tables and I think a lot of that data is still flawed because they are not measuring levels from pastured animals.

PS: Exactly.

JM: It's a total different fatty acid profile.

PS: Yes.

JM: Their levels are probably better than that. That would be the worst they could be.

PS: Yeah. Yeah. The other thing I'll mention, two more things before we close out here. We've done an epic here, Doctor Mercola, we talked about everything. This is pretty comprehensive.

JM: I think this is important because this is some groundbreaking information and one of the other things I enjoy about your take is that you are an innovator. You're thinking out of the box. Now you're not the first person to do carnivore, but you really explored it at a profoundly deep level.

JM: All the ins and outs. You aren't parroting someone else's information. In many cases, just bringing this to the table and you're the first one who's really uncovered this fat.

PS: Yeah. So what I'll say about animal fat is that it's very rich and vitamin E. One of the criticisms of the carnivore diet has been, where are you going to get your vitamin E? I think it's just like you're saying, it's just not measured properly in animal fat because when I've checked my vitamin E levels in the serum and the vitamin E levels of all of the people I work with, they are astronomically high. I mean they're not like dangerously high, but we have robust levels of vitamins E in our blood and it's coming from animal fat and so we're not measuring it properly.

PS: Then I just wanted to touch on vitamin C before we go because a lot of people wonder about vitamin C on an animal based diet. Where are you getting it? Are you getting enough? There's a couple of things. This really mirrors the K1 and K2 discussion, again. It just recapitulates what we were talking about. For the first thing to know note is that there is plenty of vitamin C in animal foods or I should say there is vitamin C and animal foods and then I'll qualify what I believe is plenty of vitamin C. But there's vitamin C in animal foods. It's just not been measured by the USDA. But there are papers that I cite in the book. It's very clear, meat is known to be an antiscorbutic.

PS: Fresh animal meat cures scurvy. There is vitamin C in animal meat. It's about 15 milligrams per pound. It's more in grass fed meat and then there's more vitamin C than that in the animal organs. So liver and kidney have about 30 milligrams of vitamin C in three ounces and so in a day without supplementation, I probably can easily get, I would say 60 to 80 milligrams of vitamin C in my diet, which is almost even the RDA for vitamin C from animal-based sources.

PS: I think that the more we learn about vitamin C, well we will learn that animal and plant sources of vitamin C are very different and affect the body differently. But the two takeaways of vitamin C are this.

PS: In order to prevent scurvy, we can eat as low a dose of 10 milligrams of vitamin C. There are studies on conscientious objectors in the 1930s in 1940s, which show that 10 milligrams of vitamin C reverses all of the clinical symptoms of scurvy. Scurvy is when we don't have enough vitamin C to do collagen synthesis properly. We can't make the triple stranded, the triple helix of collagen, and we can't hydroxylate the single strand collagen well, and then all of our tissues break down. But there's never been a documented case of scurvy in the carnivore community, in anyone that was eating a non processed meat.

PS: Now I will say that processing of meat will destroy the vitamin C. So if we're eating fresh meat, even if we cook it, but cooking of meat does not affect the vitamin C content the way it does in plants. The vitamin C in plants is much more heat labile than it is in animal foods. So it's much more resistant to degradation. So we can even cook meat and get vitamin C and be fine.

PS: Then the most important point here is that I think that vitamin C research really needs to move forward because in interventional studies with vitamin C, we do not see a benefit in common cold longevity, mortality, all cause or mortality from cardiovascular disease. So when we give people, 100, 500 milligrams of vitamin C, interventional studies, we don't see any benefit.

JM: You know what that is?

PS: Why do you think so?

JM: Because that's not how vitamin C should be used. It's like you said earlier, it's a drug and when you are sick, then taking intravenous vitamin C in high doses, 50, 100, 200 grams, or you can use liposomal like five grams every hour, that is converted to hydrogen peroxide.

PS: Right.

JM: That's probably also converted oxalates too, which might be an issue, but it's converted to hydrogen peroxide and that will address the infection.

PS: Yes,

JM: But it's only short term. You're not talking, you take this every day. This like a short term intervention.

PS: That's the way the vitamin C has been used in sepsis and cancer is intravenous. That's a completely different use of vitamin C than taking it orally.

JM: That's a pharmacological use.

PS: That's a pharmacological use of vitamin C, but when we're talking about vitamin C and the diet, I think that the literature would point to the fact that we don't need as much vitamin C as we are being told we need in order to achieve optimal antioxidant status.

PS: So vitamin C is known to recycle glutathione in conjunction with vitamin E and the membrane in ourselves, but nobody really knows how much we need to do that well. What I have seen in my own data and other people's data, and hopefully we'll get some actual experimental data about the soon, is that markers of oxidative stress do not go up with vitamin C and takes a 50 or 60 milligrams a day. [F2i suprastin 00:25:51] don't elevate, hsCRP doesn't go up and 8-hydroxy-2-deoxyguanosine.

PS: So vitamin C has just become entrenched in our consciousness and everyone listening to this is going, what do you mean I need 500 or a thousand milligrams of vitamin C? Again, I think that's

just going to turn into oxalates and be a problem. I think that's too much vitamin C and my assertion is at least clinically and scientifically, based on reviewers literature, there is really no evidence that excess vitamin C other than what we get in like fresh food is what we need for humans. I do not think, I do not believe that we need vitamin C from plants to get optimal or adequate amounts of vitamin C any way, shape or form. I've never seen evidence of increased oxidative stress and I'm eating 50, 60 milligrams a day.

JM: Well that's great. So just an interesting factoid before we close, is that my favorite dipeptide is carnosine and did you know where 99% of it is located in the animal?

PS: Tell me.

JM: Skeletal muscle, not the organs. His skeletal muscle.

PS: Exactly.

JM: That's all loaded. It's in the muscle. I mean a lot of people when they implement carnivore and I would agree with you. I think it's a flawed limitation just to eat rib-eyes and anything like some people do. It's better to eat nose to tail because the collagen is so crucial. But you're going to get a lot of carnosine if you do that.

PS: Yeah, you will. You will. Well, I mean, like I said, I think that if you're eating a 0.8 to one gram of protein per pound of body weight, you're going to get a lot of muscle meat. I'll eat a pound and a half or two pounds of muscle meat a day.

JM: That's Hong Kong style man, Hong Kong.

PS: That is Hong Kong style. It's all grass fed. It's from White Oak Pastures. It's the best I can find. But yeah, I definitely get a lot of protein and I think you get some micronutrients in the organs and then you get all the carnosine and the muscle. Yeah.

JM: Yeah. So it's great. So you've done a magnificent job. I really applaud your work. You're making valuable contributions; contributions to the whole science of what we're starting to understand what it takes to really optimize your health at a foundational level and you're really challenging some long-held tenants of health. So in dogma, so, and I know you're going to get a lot of, you have gotten a flak, and you're going to get a lot more, so I'm just delighted that you're doing the work that you're doing and really providing this information so that we can learn more.

PS: Thank you for that. I mean it means the world to me to be able to do it. I'm just grateful to be able to contribute. It's so much fun to be able to have a career now where I can think creatively and think outside of the box. I never wanted to just be a doctor. I would not have been satisfied just treating symptoms. I think that there are many valiant, intelligent, compassionate doctors who are in that system and that wasn't where I wanted to practice and so it's really great to be able to be sharing ideas and I'm glad that they can be valuable for people.

JM: All right. Well you keep up the good work and actually before we close, of course we'll have it on the article that we're doing, but for those who are just watching the video, your website is carnivore-

PS: Md.com.

JM: The book is Carnivore Code.

PS: The Carnivore Code. The subtitle is Unlocking the Secrets to Optimal Health by Returning to our Ancestral Diet.

JM: No, you needed a longer subtitle.

PS: So The Carnivore Code is the book. People can go to thecarnivorecodebook.com which is a landing page. Depending when this podcast comes out, you can pre-order it. It'll be available on Amazon for pre-order. It comes out February.

JM: When does it come out?

PS: February 2020.

JM: That's when my book comes out too.

PS: Yeah.

JM: I'm pretty confident we will certainly be able to have this before your launch, so probably the week before or the week of.

PS: That'll be amazing.

JM: It's going to be on Amazon?

PS: It's going to be on Amazon and it's going to be an audio book. It's going to everywhere.

JM: Are you doing the audio?

PS: I'm not. I'm not reading the audio. No, no. Maybe I'll do a little bit of like the intro or something, but I'm not reading. I'm not reading the audio. I've got too many other things cooking right now and then I'll leave it to professional voice actors. Maybe the next one, maybe the next one, but yeah, Carnivore MD is the website and my podcast is Fundamental Health. People should check that out. Lots of valuable information there.

JM: Yeah, it is. I pretty much, it's in my queue and I look at every episode, so don't watch necessarily every single one, but it's the valuable insights and I actually, you know what? Instead of watching like a Netflix videos or something at night before I go to bed, I'm watching podcasts and you're one of one. I've got your current one queued up today with a Chris Masterjohn.

PS: Yeah, that was a fun discussion. Yeah, absolutely.

JM: All right, so well thanks again and hope that you have good success with your book and your information gets out there and people understand some of the basics about the benefits of animal foods.

PS: Oh man, I'm so grateful. Thanks for the opportunity. It's always good to talk to you, my friend.

JM: Okay. All right. Take care.