The Human Upgrade: Episode 1145

[00:00:00] **Dave:** You're listening to The Human Upgrade with Dave Asprey. Today, we're going to talk about dogs, specifically how to hack your dog to live a lot longer. Okay. Maybe not quite, but maybe. Our guest is Matt Kaeberlein, who's working on exactly that, radically extending the lifespan of dogs, and other pets as well, but starting with dogs. Because dogs are better than cats, in case you were wondering.

[00:00:29] Now, what you're going to hear here is a really fun conversation that mixes human longevity because Matt is the CEO of Optispan, where they're extending human life, or at least healthspan, as much as they can. By the way, he's one of those guys who still says healthspan because he hasn't quite bought in on the fact that we are extending human life, and that's the goal, but I'm going to bring him over on this one. So that'll be fun.

[00:00:51] Oh, hey, Matt. You didn't hear that. And then we're going to also talk about hacking your dog, which is what I already mentioned. And we're going to talk a lot about longevity in your pets because there's such a commonality of humans and dogs. So listen to the whole episode.

[00:01:06] You'll learn something about how to think about longevity for you and how to translate it over to your pet. Matt's in town because it's South by Southwest here in Austin, which means it's a madhouse. How bad was the airport coming in?

[00:01:19] **Matt:** It was pretty crazy, but I made it. So that's totally counts.

[00:01:24] **Dave:** Right. My son is coming in. He's flying by himself, and he's probably never seen an airport at that level of chaos, so I'm looking forward to his eyes rolling back in his head as he just sees what true traffic chaos is like. Now, I'm giving a couple of talks at South by this year. You're also giving a talk on your work doing with pets.

[00:01:45] **Matt:** Right. Yeah. So there's a panel discussion on, I think it's called something like, could dogs be the key to unraveling human longevity or something like that.

[00:01:55] **Dave:** But it's neat. So you're doing the dog aging project.

[00:01:57] **Matt:** Right.

[00:01:58] **Dave:** And dogs have this really nice space between-- in longevity research that a lot of my books are based on, it all starts with yeast and roundworms. And then maybe goes to mice.

And then maybe we have some other data somewhere or another, and then we start using it with humans using aging clocks.

[00:02:20] **Matt:** That's what we do today. You're right.

[00:02:21] **Dave:** Yeah. And aging clocks are pretty useful because before that we couldn't do anything. There's still some Luddite skeptic people.

[00:02:28] **Matt:** Like me?

[00:02:28] **Dave:** Well, no, like Peter Attia, who says aging clocks aren't scientific. I'm like, I want to see Steve Horvath in a wrestling match with Peter Attia.

[00:02:37] **Matt:** Well, I know both of those guys, and I'm pretty sure I know who would win the wrestling event.

[00:02:40] **Dave:** Well, Peter would win, but that's because he's a meathead, not a longevity scientist, is my point here. So sorry, Peter, you need to revisit what is scientific, and maybe less time in the gym, more time in the labs, just saying.

[00:02:52] **Matt:** So I actually want to push back on that a little bit. Peter is a friend of mine, but also one of the people--

[00:02:58] **Dave:** I've been on stage with him. He's not a bad guy. He's not a believer in longevity.

[00:03:03] **Matt:** No, I would disagree with that. So look, I think Peter has strongly held opinions, like all of us do.

[00:03:08] **Dave:** That are wrong.

[00:03:10] **Matt:** Most of the time he's right. And I would say among--

[00:03:12] **Dave:** Oh, on statins and vaccines?

[00:03:14] **Matt:** Among a lot of the influencers in this space, I think Peter does a good job of doing his homework. And I think there is some nuance here. I am certainly not going to try to speak for Peter, but here's the way I would frame the aging clocks. I think they are very useful

research tools. I think that the utility of the aging clocks in the consumer space is pretty questionable in my mind.

[00:03:39] And part of that is because we really don't have much data on how precise or reproducible these clocks are when sold by companies to consumers. And so I actually have a lot of concern that these clocks are being misused and abused in the consumer space.

[00:03:56] **Dave:** Interesting.

[00:03:56] **Matt:** I also think that it's important to recognize that all of the clocks that are currently in use are really only measuring one, sometimes two small aspects of biological aging. We don't really know to what extent are they capturing the entire biological aging process.

[00:04:15] **Dave:** Nothing does right now. And we do know that the aging clocks and Steve Horvath's interpretation of it is the first one, but there's about a dozen others. They can predict when you're going to die within about 10 years pretty reliably.

[00:04:29] **Matt:** I think it depends on what you mean by pretty reliably. I can look at somebody and predict pretty reliably whether they're going to die in 10 years.

[00:04:35] **Dave:** That's a good point. We'd have to look at-- I don't know if they compared it.

[00:04:39] **Matt:** I've had this conversation with Steve, so I know how the math plays out. They're better than just looking at somebody. They're honestly not that great.

[00:04:49] **Dave:** They're better than telomeres.

[00:04:51] **Matt:** Yes, that's true, but the question is, is that because we don't have the tools yet to precisely measure individual telomere lengths? I actually think this is a super interesting question. It's way off on a tangent, but I think it is really something we don't know at this point, whether the epigenetics, which is what Steve's clock is based on, those are nice because we have huge dimensionality. There are tens of thousands of epigenetic marks we can measure, and we can do it very precisely.

[00:05:20] That's very different than the telomeres where we only have 46 or so things we can measure, and we can't do it very precisely. So maybe telomeres actually are a pretty good clock if we could measure them. I think we just don't know that. I actually don't believe that. I'm just

saying we don't know the answer at this point. It's true that we're discovering a lot, and we know more now than we did.

[00:05:39] **Matt:** Nobody can argue with that.

[00:05:41] **Dave:** Every 72 days, the amount of info we have about biology doubles, right?

[00:05:46] **Matt:** Yeah.

[00:05:46] **Dave:** So it's this golden age of longevity research. And I've seen telomere numbers swing by 20 years in one week. That's either because of lab errors or because your blood is a terrible place to get telomeres.

[00:05:59] **Matt:** Well, that's I think that's a good point. Yeah, absolutely. The tissue type that you're measuring and blood, of course, is a complex set of cell types that you're actually measuring. So yeah, there's that. But this goes back to my point earlier, which is that I think that while these things can be quite useful in a research setting, because of a lot of those uncertainties about what we're actually measuring, their utility in the real world, for now, I think, is unclear. But I'm super enthusiastic that we're going to get there in probably the not too distant future.

[00:06:27] **Dave:** I would say that aging clocks are extremely scientific in early days.

[00:06:34] **Matt:** Fair.

[00:06:35] **Dave:** And so the idea that, well, there's no way we're going to extend human life, and there's no way to measure how old you are biologically, which is a summation of Peter's work, which is why I'm like, let's go to the A4M. Let's go to the people who are actually looking at extending life with something besides exercise and statins. Because, well, I think those have been tried for a while, and no one's living longer. That's why I have a bone to pick there.

[00:07:03] **Matt:** You can tell by looking at it.

[00:07:03] **Dave:** Twenty minutes a week of exercise is all I do. And I do it with AI at Upgrade Labs. I believe that most people way over exercise, including Peter. Because it turns out it's not the area under the curve. It's the slope of the curve up, and most importantly, the slope of the curve down. And then the nutrient and protein availability.

[00:07:21] **Matt:** So I'm going to push back a little bit and suggest--

[00:07:25] **Dave:** But wait, Peter could kick my ass, just to be really clear.

[00:07:27] **Matt:** Most people don't over exercise. Maybe people in the optimization world over exercise.

[00:07:33] **Dave:** People who exercise every day usually over exercise, and almost everyone doesn't move enough. So there's a lot going on there. And it's funny because we're here to talk about dogs. We're talking about humans because I think dogs are the bridge between the research from mice and yeast and the little creatures.

[00:07:54] And hey, if they can live longer, there's pretty good evidence that some of that stuff will help us, but it doesn't always. Well, what's in the middle? Well, dogs are a lot closer to people than they are yeast, but they're still pretty far away.

[00:08:07] **Matt:** Yeah, yeah. And so first of all, I love the way you frame that because my career path has followed that trajectory. I started in yeast as a graduate student, then worked in nematode worms as a postdoc.

[00:08:20] **Dave:** There you go.

[00:08:20] **Matt:** And when I started my own lab, we started doing stuff in mice. Because I believed that there were aspects of aging biology that were in fact shared across this broad evolutionary distance. Turns out that's right. And then about 10 years ago, it occurred to me that, as you suggested, dogs are this really powerful bridge between the laboratory studies and humans.

[00:08:42] Not only because they're closer to humans in a biological sense, but there's this huge environmental component that we can't or choose not to study in the laboratory that's really important for human aging. And because dogs share so much of the human environment, at least companion dogs or pet dogs do, we can capture that environmental component and really understand not only what are the most important genetic factors that influence longevity, but what are the most important environmental factors.

[00:09:10] And then there's this huge other piece, which I'm most interested in, is we can actually test whether interventions affect lifespan and healthspan metrics in dogs, which would

be great for giving us confidence about people, but I'm a dog person. So what I really care about is making my dog and other people's dogs live longer, healthier lives.

[00:09:27] The number of people in the longevity enthusiast field, whether you work in the field or you're just someone like me who's spent decades-- I guess I do work in the field, come to think of it, but I didn't used to. So the number of us who have pets who are not on longevity regimens is very low.

[00:09:44] All of us know, huh, the things that work for me, like red lights, and we know what it does with mitochondria, and cell folding, and all that stuff, maybe I should shine it at the dog when the dog hurts, and then the dog stops hurting, and it probably affects longevity, and maybe not feeding them the pet equivalent of chicken nuggets will also affect longevity, right?

[00:10:03] **Matt:** Right.

[00:10:03] **Dave:** So we all do that, whereas it's still very common people go to the store, and they buy big things of pet food that are made out of euthanized dogs. Maybe your dog shouldn't eat that. There's some really bad stuff happening.

[00:10:19] **Matt:** There's some really interesting and, in many ways, unfortunate parallels between what most human beings eat, or at least the quality of the diet that most human beings eat and the quality of the diet that most human beings give to their companion animals. And there's complicated reasons for that, obviously, but I think you're right. I think the people who are on this path towards improving their own healthspan trajectory often recognize that the same thing is true for their pets and take a healthier approach to their pets.

[00:10:52] **Dave:** So you're a PhD.

[00:10:54] **Matt:** Yeah.

[00:10:54] **Dave:** And actually a very well-studied one because you, well, I guess published 250 studies.

[00:10:59] **Matt:** Something like that.

[00:11:00] **Dave:** You founded research centers, and you're in the American Aging Association. And so you talk like the best PhD ever, but I think what I heard you say, and that was that humans and dogs shouldn't eat kibble. Did I get that right?

[00:11:13] **Matt:** Certainly, I think humans probably should not eat kibble. I think, again, the quality of the food itself in the general food supply is really problematic both for dogs and for people.

[00:11:28] **Dave:** Yeah. So we've got to fix food, and for me, food is mostly not a longevity issue. It's a health span issue.

[00:11:38] **Matt:** I don't know. I understand that perspective. I would say I'm a little bit less certain. I certainly believe to some extent that the reason quality diet, appropriate amount of exercise, whatever that is-- I think we're still figuring that out-- exercise, sleep quality, I think the reason those things impact multiple age-related diseases simultaneously is precisely because they are impacting the biology of aging.

[00:12:03] The way I would frame it is you can only get so far by modifying those lifestyle factors. And so you're not going to see 150, 160-year-old people in good health simply by modifying lifestyle.

[00:12:17] **Dave:** Exactly. We're not going to extend life very much. You can reduce toxins. You can probably hit your maximum lifespan.

[00:12:26] **Matt:** Genetic potential without additional intervention.

[00:12:29] **Dave:** Yeah. And that's lifestyle and all that.

[00:12:31] **Matt:** Right.

[00:12:32] **Dave:** But extending it, that's stuff like the gene therapy that I just did.

[00:12:36] **Matt:** Maybe. We'll see.

[00:12:40] **Dave:** We'll see, of course. And for all of history, it's always been a we'll see for all the things we're doing on things like that. We just have better tools now to do it. And hey, the guy who said man will never fly a year before the Wright brothers flew, maybe he was right. I'm going to bet on us extending human lifespan because we are.

[00:13:03] **Matt:** I have no doubt.

[00:13:04] **Dave:** You see all the research being done.

[00:13:05] **Matt:** I have no doubt that it's possible. Of course. The questions in my mind more revolve around how long is it going to take before we can actually demonstrate that these things work.

[00:13:18] And this is where, going back to the aging clocks, I think once the aging clocks evolve to the point where there's confidence that they are in fact predicting future mortality, future disease risk with high precision, then you have a real opportunity. It's still not going to be proof until you actually see somebody live that long, but you can be pretty darn confident.

[00:13:38] Yeah, these things are having the effect that you wanted them to have. Then the last question is, is there some long-term consequence that we don't know about that's going to offset that? Because it only takes one mortality event to keep you from living a long time. So that's an unknown, and we're not going to know until we get there.

[00:13:53] **Dave:** One of my big longevity strategies is to drive a heavy vehicle.

[00:13:58] **Matt:** Avoid dying, I think, is rule number one of longevity, yeah.

[00:14:03] **Dave:** Use physics.

[00:14:03] **Matt:** Necessary, but not sufficient.

[00:14:07] **Dave:** Yeah, sorry. If you're in the Prius, I want to be in the Suburban. And it's not to say that I think I'm more important than you. I'm just allocating my resources in a way where physics protects me. And it's something I think is not looked at enough in the field of longevity, is just don't fall down and break yourself. Don't blow out your spine hitting a new PR when you're 60. It's probably not worth it. Right?

[00:14:31] **Matt:** Yeah, absolutely. We think about four movements at Optispan as we're trying to communicate some of these ideas to people, and one of them is staying alive. You got to avoid risky behaviors, and there's a lot of luck. There's a lot of probability in life, but you can change the probabilities in your favor, like you're saying, by taking steps to reduce your likelihood of dying.

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[00:14:57] **Dave:** If you had to pick one thing, not exercising or eating just the worst possible fast food diet for the rest of your life, which one would you pick?

[00:15:05] **Matt:** So I have to do one of those two things? Oh God.

[00:15:09] **Dave:** If you want to exercise, you have to --

[00:15:10] **Matt:** I don't think I could eat the worst fast food diet. I'd have to choose not exercising. That would suck for me, but I could-- yeah. Again, as somebody who ate a pretty crappy diet like many people for a big chunk of my life--

[00:15:22] **Dave:** Yeah, I think we're about the same age.

[00:15:23] **Matt:** Now that I'm out of that world, it just is repulsive to me to think about going back to it.

[00:15:29] **Dave:** I will never go back to eating food that makes me feel crappy, and I am willing to die on that. And this is what the regulators who think we're going to live on crickets and grain-- I can't live on that. And I won't. And I'll eat a politician before I'll eat a cricket. And I'm just okay with that. They're made out of meat. It's not my ideal source of meat.

[00:15:46] **Matt:** I actually think this is pretty interesting because I've talked to a fair number of other people who share that, not so much about eating politicians, but the revulsion at the idea of going back to this standard American diet.

[00:16:01] And it's hard sometimes for people who aren't already in this place to understand that, but I think it's just worth emphasizing. It's really about building habits. And if you can get out of the habits that you're in, that society and culture pushes you towards, and get to a better place in terms of the quality of your diet, I can almost guarantee you will agree that there's just no way you want to go back to that once you get out of it.

[00:16:29] **Dave:** And also it's not the same for everyone.

[00:16:31] **Matt:** This is true. Absolutely.

[00:16:32] **Dave:** There are people who just feel great on chicken. I am a beef guy, and I've tested everything. I would eat gravel if I could, but beef is what works for me. And you try and

take away my beef with a regulatory thing, it's like, no, you've taken away my oxygen. We are not going to be okay with this.

[00:16:47] **Matt:** So here's the way I think about it. I think there are multiple types of high-quality diet. There is not a one size fits all. I completely agree with that. But I also think that there is a high-quality diet that can fit almost everybody. So in other words, I don't believe that there are people who will just thrive, or if there are, they are the way outliers who will just thrive on garbage.

[00:17:15] **Dave:** And there are some. I know people who do at least for a while. And I also know a couple of people who thrive on a vegan diet. It's just their kids and their grandkids are paying for it because it matters. In fact, if you look at the health of vegan offspring and things like that, it's not pretty, the placental health. So you might get away with it, but you're actually paying for it in future generations. It's a big experiment.

[00:17:40] **Matt:** Yeah. I'm less opinionated about vegan diet. I'm not a vegan, but I don't-- I'm certainly not a vegan basher.

[00:17:47] **Dave:** I was a devout vegan for a couple of years, and it trashed my health in a big way. And I'm a food supporter. I absolutely love the vegan philosophy. You want to take care of your health. You want to take care of the planet, and you want to reduce animal suffering and death. The problem is the vegan diet doesn't do any of those three things. And I believed it did.

[00:18:10] **Matt:** Yeah. And I think the way I also think about this, but I struggle a lot with the nutrition literature-- honestly, look, these are good scientists trying to do good science, but it's really hard to do high quality definitive science in human nutrition.

[00:18:28] **Dave:** Oh yeah.

[00:18:28] **Matt:** And so I really struggle with a lot of the epidemiology. And so personally, I'm very slow to come to strong conclusions about what is the optimal diet, what isn't, and what are the long-term consequences to some of these different dietary approaches people take.

[00:18:46] Again, the way I align is there are some things that we can be almost certain are not good to have in your diet, highly processed foods, a lot of simple sugars, low quality fats. So I

think those are the things that most people should agree if they're knowledgeable you want to get out of your diet.

[00:19:06] **Dave:** And all the flavorings, and colorings, and things like that.

[00:19:08] **Matt:** Yeah, right. Yeah.

[00:19:09] **Dave:** And those are bad for dogs. They're bad for people. And from there, what plant toxins can your body tolerate versus mine? 28% of rheumatoid arthritis is caused by nightshades if you have the genetics. But if you don't have the genetics, you can eat them all day long.

[00:19:26] **Matt:** And this really actually ties in in an interesting way to the biology of aging because I think a lot of what's driving that is our own individual immune response. So the reaction of the immune system to molecules that we're getting in our diet and other places in the environment that drive chronic inflammation, some people are just much more prone to that hyper inflammatory state. And that leads to a lot of the conditions that you're talking about.

[00:19:50] **Dave:** I'm certainly one of those being 300 pounds and all the health stuff I've gone through when I was younger. So I recognize I'm coming from behind in my goal to live to at least 180. Well, dude, I trying. I'm fine with that.

[00:20:03] **Matt:** Yeah.

[00:20:03] **Dave:** I just want to do 50% better than my current best.

[00:20:05] **Matt:** But the nice thing is-- this is pretty cool-- one of the things that has emerged from the literature in my academic career is that it doesn't seem like there's a point where it's too late unless you've got a disease or the pathology of the disease has outpaced the biology of aging. So we now know. And we didn't know this when I started, but now we know you can start intervening in middle age and still get pretty big effects on both longevity and healthspan.

[00:20:31] **Dave:** It's funny. If you start before pregnancy, the three months before your mother was pregnant with you and you have a really healthy pregnancy, you get the most leverage on longevity.

[00:20:41] **Matt:** That's all the epigenetic effects, right?

[00:20:43] **Dave:** Yeah. That was when my first book was on fertility. What do you do before you get pregnant? And then you get less and less leverage the longer you wait to do longevity interventions. I'm just luck in my 20s, I learned from people in their 80s, all this longevity stuff in Palo Alto.

[00:20:57] **Matt:** I wonder though, because that's the mindset I think people had before 2009 when the first study on rapamycin in mice was done, where they accidentally started the treatment in a 60-year-old equivalent mouse. But I think what we've learned is that at least with some interventions, you can get most of the benefit, even starting late in life.

[00:21:19] **Dave:** With some of them you can.

[00:21:20] **Matt:** Yeah, and I think as these tools to reset the epigenome evolve, we might actually be able to get closer to that initial epigenetic state that you were talking about.

[00:21:29] **Dave:** It may not even matter at a certain point. In the world that I see coming, you'll be able to edit your mitochondrial DNA so it more closely works with your nuclear DNA. And these are all editable settings in your hardware. The biggest challenge will be if you make too big of a change is just causing systems integration to work between all the differences in your body.

[00:21:51] **Matt:** Yeah. It's interesting. I don't know how far. Again, I agree. Theoretically, that's all possible. I think the challenge that I see, and this is where it'll be interesting to see how effective the evolution of AI tools is at disentangling the complexity of the biology. Look, I'm in this field deep, and I recognize how little we actually understand.

[00:22:13] **Dave:** And half of what we think we know is just wrong. And the AI believes it. That's one of the problems.

[00:22:18] **Matt:** So it'll be interesting to see. But I do agree. All of that's on the table. I just don't know how long it's going to take to get there.

[00:22:24] **Dave:** So I have a pretty good track record of seeing things before they happen. I'm the first guy to sell anything over the internet before e-commerce had a name. It was a t-shirt that said, Caffeine, My Drug of Choice. I sold that in my dorm room. It was in the Miami Herald, the first data center companies.

[00:22:40] I've picked reliably-- I also have a track record of thinking it's going to happen before it does, but now I'm wise because I have some age on me now. So I would have said, oh, it'll be three years before we get there if I was 20. I'm going to say 10 years before we get there, mostly because of advances in AI subject to two things.

[00:22:58] One, that governments don't succeed in basically destroying our access to these things, or to breaking AI. I don't think they can. But they might. And two, a comet doesn't hit the planet. But otherwise, I'm pretty confident on it, because we have a hard time conceptualizing. This is even beyond exponential rates of growth in AI.

[00:23:19] So I think we're going to get there as long as we train the AI to care about it, then it'll do it. But if we train the AI to be really good at killing people, it might not care much about making us live longer. So this is the--

[00:23:29] **Matt:** Yeah, it'd be interesting. So we should sit down and come up with a bet about what do we actually want to bet on? I'm less optimistic, but in part that's because-- and again, this is where the exponential equation can fool you, but I look back at where we were 10 years ago in this field, and I don't see 10 years from now as getting to that point. So we'll see.

[00:23:48] **Dave:** Oh, not everyone will get there. The first couple of people will get there in 10 years.

[00:23:54] **Matt:** There's already people who are editing their genome.

[00:23:56] **Dave:** Sure. I've had the gene therapy, but it wasn't my inheritable genome.

[00:24:01] **Matt:** Which gene therapy?

[00:24:02] **Dave:** I did the Falstad gene therapy with mini circles, just plasmid level stuff. And I'm impressed. And I'll be in the first trial for--

[00:24:11] **Matt:** The biology is real. There's no question about it.

[00:24:14] **Dave:** Yeah. It works. And now the fact that nine years, it comes off your aging clock for that, you can say, well, that doesn't really matter very much. I'm like, yeah, but the bone density increase in muscle and the decrease in fat works.

[00:24:29] **Matt:** Yeah, it's super intriguing. That's one of the spaces I'm watching pretty closely.

[00:24:33] **Dave:** It's funny because in your day job, you're at Optispan. You're working on extending human lifespan for high-end clients, and then in your night job, you're saying, well, let's do the same thing for dogs. So it's cool.

[00:24:43] **Matt:** Yeah, although let me clarify. So it's not for high-end clients only, right?

[00:24:47] **Dave:** Oh, what is Optispan? I don't know.

[00:24:48] **Matt:** So what I would say is our mission at Optispan is really to enable the transition from reactive disease care to proactive health care for as many people as possible.

[00:24:58] **Dave:** I like it.

[00:25:00] **Matt:** And I think there are lots of ways you can do that. There are lots of smart people working on this. But I think we have an opportunity to contribute. And so we're really looking right now at two lanes. One is high-end concierge medicine because that's the place where you can do the most expensive, most sophisticated approaches here.

[00:25:17] But we're also working in the corporate wellness space and trying to say, what can we do in this proactive science-based health care for 1,500, $2,000 a year, something employers might be willing to put towards employee health, recruitment, retention, things like that? So we really are trying to democratize this as much as possible, recognizing that as a for profit company, you also have to be able to make revenue.

[00:25:46] **Dave:** Absolutely. If you run a business that doesn't make any money ever, it's probably not a very good business, unless you're Amazon or Tesla. Oh, wait, sometimes you can have external funding, but I'm with you. I believe that the best way you can change the world is as an entrepreneur or not as a donor.

[00:26:05] And I also have donated meaningful amounts of money to projects I believe in, but end of the day, if the money makes more money to support the cause, now you've got a movement versus you've got a one-time flash. We've talked about human aging a little bit here, and there's a lot we don't know, and we're making great progress. Now, we also know small dogs live longer than big dogs. Does this mean that small humans live longer than big humans?

[00:26:33] **Matt:** Yeah. Actually, there's some evidence to support that. So this is something where people have a lot of confusion. There's this idea that large animals live longer than small

animals, and that's true if you look across species. So if you look across the animal kingdom, larger animals do in fact live longer than small animals.

[00:26:49] **Dave:** Like elephants and whales, right?

[00:26:50] **Matt:** Exactly, yeah. But when you look within a species, in general, at least in mammals, smaller individuals tend to live longer than larger individuals, and that's true in people. It's complicated in people by the fact that there is a social component to being tall, and so you have to take that into consideration. Dogs are interesting because of the body size difference is so much bigger when you look across the dog species that we can really see that difference in aging rate.

[00:27:16] **Dave:** I've had six Dachshunds over the course of my life, including when I was a kid, and all of them lived 15, 16, 17 years old. But the last two were when I knew the most about nutrition and longevity. And I applied those things. A Merlin, who was the last dog that I had, passed away when he was 15 and a half or something.

[00:27:39] But he got a krill oil every day, and he got a little bit of collagen, and he ate mostly raw, grass-fed beef, and some egg yolks on occasion, and very small amounts of vegetable matter. And when he was young I gave him things the BARF diet, bones and raw food. And I don't think dogs are supposed to eat huge amounts of celery and carrots.

[00:27:58] It didn't work so well. But we ended up with a dog that just didn't have pain in his body, didn't have a lot of the debilitating things, no lipomas, and good fur, and it was pretty incredible actually.

[00:28:11] **Matt:** Right. Yeah. I think this goes back a little bit to what we touched on earlier about how a lot of the components of the diet for dogs as well are pro inflammatory. In other words, they're not things that the canine body has evolved to see. So there's an immune response against some of these factors, and that gives you this chronic inflammatory state, which drives a lot of diseases of aging, a lot of the pain that you mentioned.

[00:28:33] One of the things that's challenging in dogs is there's much less data on the long term effects of different diet in dogs. So there's a lot of strong opinions out there, and not a lot of good data. So one of the things we want to do at the Dog Aging Project is actually collect that data so

we can actually start to understand and be able to draw strong conclusions that, yes, in fact, a particular diet does seem to be associated with much longer healthspan, there's that word, and lifespan.

[00:29:04] **Dave:** Well, we all want healthspan. So it's a great goal. It's just not a big goal. I want healthspan twice as long.

[00:29:12] **Matt:** Yeah.

[00:29:12] **Dave:** That's a big goal.

[00:29:13] **Matt:** I agree. So I hear what you're saying. Here's the way I would think about it, and this is easier to talk about in humans than it is in dogs again because we have the data. At an individual level, I agree completely. At a population level, healthspan in the United States sucks. 60% of people have a chronic disease.

[00:29:31] Average lifespan in the United States is 38.1 years. That means that if you define healthspan as the period of life without a chronic disease or disability, healthspan ends for most people before they're 38 years old. Life expectancy is in the mid 70s. So if we could push that healthspan curve out, that's a big deal at the population.

[00:29:52] **Dave:** It's a very easy goal. It's mostly--

[00:29:54] **Matt:** It's easy to talk about, but it's harder to accomplish.

[00:29:57] **Dave:** It's behavior change. We don't need to know more to do that. We just need to do more to do that. You can move it out 20 years with what we know today.

[00:30:03] **Matt:** I think that's true. I think it's more than behavior change, though. I think there's policy factors. There's also a lot of other stuff that goes into that. But I hear what you're saying.

[00:30:11] **Dave:** Yeah. You have to clean out the big food agencies that control the government agencies that tell us what to eat. That might be part of it. And maybe change some tax incentives, but those are all ultimately behavioral things.

[00:30:21] **Matt:** It's doable.

[00:30:21] **Dave:** Yeah. It's not like we have to crack the atom in order to do this.

[00:30:26] **Matt:** This is true.

[00:30:26] **Dave:** And what we're looking to do in the doubling of human lifespan is the equivalent of a Manhattan Project level undertaking.

[00:30:32] I agree. Look, the best that people have been able to do in a mouse beyond genetic intervention through development is about 60% increase in lifespan. So we've still got a long ways to go.

[00:30:46] **Dave:** [Inaudible].

[00:30:47] **Matt:** Right. So you have to be careful about short lived controls. So percent change is numerator over denominator. If your denominator is much smaller than it should be, your percent change gets dramatically over inflated.

[00:31:01] **Dave:** So then you're saying you get mice that are engineered to die in 90 days and make it live 180?

[00:31:06] **Matt:** Or because of poor experimental conditions, they were exposed to toxins, or pathogens, or whatever that shortened their lifespan. So I guess I should reframe that. The largest effect that has been reproducible in a quality experiment is about 60%.

[00:31:22] **Dave:** Okay, there you go. I like that. And it's funny. My 180 number, we have humans who are 120. I just want to do 50%--

[00:31:30] **Matt:** Hey, I think that's a fantastic goal.

[00:31:32] **Dave:** Yeah, that's where 180 comes from for me. And I know I might not make it. But I don't think it's a crazy goal. I think it's a big goal, right?

[00:31:39] **Matt:** I would agree.

[00:31:40] **Dave:** And that's why I keep talking about it. It's so many people like, that's crazy. I'm like, I don't think it is. It might not happen in my life, but I'm going to bet on it. And what else am I going to do with all this time? All right.

[00:31:53] **Matt:** And it's fun to talk about, right?

[00:31:54] **Dave:** It totally is. Okay.

[00:31:56] **Matt:** Back to dogs.

[00:31:57] **Dave:** One of my questions about dogs is, and you try to control what they eat and all this sort of stuff. I have taken my dogs to a dog park. They eat poop. How do you control for poop intake?

[00:32:12] **Matt:** Believe it or not, that is one of the questions on the survey. So the Dog Aging Project longitudinal study actually has a very comprehensive survey tool that we use to collect a lot of data about the dog's environment, health history.

[00:32:27] So indeed, at least one of the questions, there may be more than one, addresses whether or not your dog consumes feces. Also asks about time spent at the dog park, time with other animals. So we really do try to capture that. It's not quantitative. We don't ask how much feces does your dog consume.

[00:32:46] **Dave:** You have to give it a scale before you--

[00:32:47] **Matt:** But believe it or not, there are-- I'm sure you know this-- certain dogs that absolutely love to eat poop from other dogs.

[00:32:57] **Dave:** Yeah.

[00:32:57] **Matt:** And then there are other dogs that won't touch it. They might eat rocks or something. So there's a genetic predisposition here as well.

[00:33:04] **Dave:** But is there a certain breed that's more of a poop eater? Because I don't--

[00:33:06] **Matt:** Probably. I don't know the answer.

[00:33:08] **Dave:** I don't want a poop eater breed.

[00:33:12] **Matt:** Yeah, we had a Kazon or some people call them Keeshonds that was a poop eater, and boy, you had to watch her like a hawk.

[00:33:18] **Dave:** And you're like, don't kiss me.

[00:33:21] **Matt:** And Kazon loves to kiss you as well. Yeah. It's a bad mix.

[00:33:25] **Dave:** The other thing that's a great crossover is that people who own dogs live longer than people who don't. Is that from emotional support, energetic support, or some bacterial thing in the microbiome?

[00:33:37] **Matt:** It's a good question. Yeah. There could certainly be, like you're talking about the microbiome, those kinds of biological interactions. My intuition is that really more comes from the connection that a lot of people get from their dogs. So we all know that human connection is really important for happiness, and that people who have strong human connection also tend to live longer. And I absolutely believe that many people can get at least some of that connection from their companion animals.

[00:34:08] There's also really good data that interacting with a dog, or a cat, believe it or not, can have impacts on the physiology of the person in terms of reducing stress hormones, reducing anxiety. So that certainly could play a role as well.

[00:34:25] **Dave:** So you don't think that the toxoplasmosis from cat owners that causes the crazy cat lady thing is real?

[00:34:31] **Matt:** Again, there's probably a genetic predisposition there.

[00:34:34] **Dave:** That was the best PhD answer ever. That was the worst loaded question I've ever asked a guest, and he dodged it like a pro. So Matt, congratulations. The other difference between dogs and cats is that if you were to pass away unexpectedly, the dog will sit next to you and starve to death guarding you. And the cat will eat your eyeballs, just saying.

[00:34:54] **Matt:** I have no direct experience with that.

[00:34:57] **Dave:** I like cats. If I had a choice, I would pick the dog. So guys, I know that if you have a cat right now--

[00:35:05] **Matt:** You're just really trying hard to go after the cat people today.

[00:35:07] **Dave:** Well, maybe we can do a crossover study with vegans. What do you think of vegan cat food and vegan dog food for longevity?

[00:35:15] **Matt:** Personally, I wouldn't use it.

[00:35:16] **Dave:** As a PhD.

[00:35:16] **Matt:** Yeah, I wouldn't personally feed that to my dog.

[00:35:19] **Dave:** Yeah. What about feeding people food to dogs?

[00:35:24] **Matt:** It depends on the people food. So feeding big Macs and fries to your dog on a regular basis, probably a bad idea.

[00:35:32] **Dave:** So I go back to my childhood. We didn't know much. In fact, we were in the days of get rid of butter, and use squeeze margarine, brown muffins, and it's fine to go get a big Mac. And we would boil hot dogs, just boil them and make hot dogs at home. And we would give the hot dog water to the dogs.

[00:35:51] And they would drink it until they were so bloated, like little stuffed sausages. You could like flick their sides, and they would make weird watery sounds. And I look back, and I'm like, that was the worst thing I ever could have done to my poor dogs. But like, well, they like it, and it's food, and I'm willing to eat it.

[00:36:06] And I was just entirely ignorant of the effect of the food on my biology, which was jazz, and on the dogs. But those dogs still live 17 years. They had a bunch of lipomas, and their final couple years maybe weren't that comfortable compared to later in life. But it seems like food quality in humans from 25 years ago was and now, there's something different that maybe is worse for pets and worse for humans. Do you think there's been a change?

[00:36:30] **Matt:** Well, there's clearly been a change. There's no question that the composition that the typical person is eating in their diet is obesogenic. And that's just using a fancy word to say something we all know, which is that what people are eating now drive us towards obesity.

[00:36:48] And all you have to do is look at pictures from 30, 40 years ago of people, and this is obvious. So exactly what those mechanisms are, I think we're starting to learn. I think there's been a lot of engineering of the food to get people to eat it faster, to get people to eat more. And that's intentional. That is intentional engineering.

[00:37:09] I understand why the food companies did it. But then there's this additional factor there, which I don't know whether it was intentional or not, that it's not satiating. So then people are hungry again. So they eat faster and eat more. And there's this cycle that drives obesity. So absolutely, there have been changes.

[00:37:25] **Dave:** Okay. I think that's a big part of it too. There are some compounds that I've written about a lot of people in the longevity field are paying attention to, like rapamycin. Can you talk about rapamycin? What is it, and should we give it to our dogs?

[00:37:40] **Matt:** Sure. All right. Rapamycin. Right. So rapamycin is a small molecule that was found on Easter Island, which also goes by the name Rapa Nui. That's where the drug gets its name. It's actually produced by bacteria there in the soil. And rapamycin has this super cool backstory. So if anybody's interested, you can Google it and find it.

[00:37:59] **Dave:** Tim Ferriss went to the island to go discover it or something.

[00:38:02] **Matt:** Along with your buddy, Peter Attia.

[00:38:04] **Dave:** Oh, yeah. Yeah.

[00:38:04] **Matt:** And my real buddy, Peter Attia.

[00:38:06] **Dave:** Yeah. No, I've been on stage at A4M with Peter. It's just weird to have someone on stage saying you can't extend human life. I'm like, what is wrong with that?

[00:38:14] **Matt:** This is one of my bucket list items, is to get to Rapa Nui and see where it all started. But regardless, that's where Rapamycin was first discovered. And it was initially attempted to be developed as an antifungal, anti cancer agent, but the way it actually was developed clinically was for organ transplant patients. Because it is a potent anti inflammatory and what people call anti proliferative drug.

[00:38:41] In other words, it slows down the cell cycle. And so in an organ transplant patient who's got a transplanted organ, you want to target the immune system to prevent it from reacting to that organ. So it was clinically approved as an organ transplant drug, and it's called an immunosuppressant, and that's how it's been used clinically.

[00:39:01] So there's a lot of data in human use there, but I think we have to recognize those are in people who there's a reason why they had an organ transplant. They're taking high doses of rapamycin, and they're taking a bunch of other true immunosuppressants. So it does have some side effects in that context. But it's got a bad reputation clinically because of how it was used and developed.

[00:39:20] Now, in parallel, really around the early 2000s, several labs independently discovered that you could treat yeast, worms, flies, like we were talking about, with rapamycin and increased lifespan. And so people started studying what's the mechanism there.

[00:39:39] And it turns out rapamycin is an inhibitor of a protein called mTOR, mechanistic target of rapamycin. And we now know from studies over the last 20 years that mTOR is if not the most potent, one of the most potent knobs that we can turn to regulate the rate of aging in laboratory animals.

[00:39:59] And as we alluded to earlier, there was a study, I think that really changed the game for rapamycin in the longevity field in 2009 from the interventions testing program, where they showed that you could start treating mice at 20 months of age, which is about the mouse equivalent of a 60-year-old person, with rapamycin, still get significant effects on lifespan.

[00:40:19] And then since then, lots of labs have shown not only can you increase lifespan when you start treatment in middle age, but you can actually improve pretty much every metric of aging that people look at in pretty much every tissue and organ where people study it. So it really seems as though rapamycin modifies the biology of aging in a way that can increase both lifespan and healthspan, again, at least in laboratory animals, and you can get the benefits starting in middle age.

[00:40:47] **Dave:** mTOR is an interesting compound because of that research back in 2009 when I first started the biohacking movement and I wrote one of my early posts, was how to triple down on mTOR because it works. You're a PhD. You can say if any of this is wrong, but the picture I have in my head from reading all the papers is that it works like a spring.

[00:41:08] You want to suppress it, but the more you suppress it, the more it's going to spike when you unsuppress it. So the three big things I could find back then that were going to suppress mTOR temporarily were fasting, coffee, and weight-bearing exercise. So that's funny. Drink coffee during the fast. Lift at the end of the fast, and then eat a meal with protein and carbs because carbs raise mTOR 10 times more than protein. Insulogenic carbs.

[00:41:34] **Matt:** Right. So I actually think this is where there's a lot of confusion out there. So most people would say it's the protein.

[00:41:41] **Dave:** I think it's the carbs. Do you think it's the carbs?

[00:41:42] **Matt:** Because I don't know the answer to that. And I think it depends on where you look, is the honest answer. I think it depends on the tissue that you're looking in, but it is absolutely massively oversimplistic to say that it's only amino acids that activate mTOR. But you'll see that all the time.

[00:41:58] **Dave:** It's something that I'll say I probably went 50% too far in that direction in my longevity book. Because I wrote about 0.6 grams of protein per pound of body weight. And there's a bunch of studies that support that. But most of them are because of methionine, and tryptophan, and other things that increase mTOR. But if you want to look good, you need mTOR because that's how muscles build, and you don't want sarcopenia, and you don't want bone loss.

[00:42:24] I have absurdly dense bones and a relatively high amount of skeletal muscle mass when I look at all my quantified stuff. So if I drop my mTOR too much, the only thing I could eat would be fat because carbs raise it, and protein raises it from animals, but I could eat brown rice protein full of arsenic and stuff, but it's biological availability is so low, I need twice as much of it, so I'd be eating industrial processed plant proteins, huge amounts of them, farting all the time, and having all the-- there's no way to live life.

[00:42:55] **Matt:** Yeah, there's a quality of life issue there.

[00:42:57] **Dave:** Yeah, you can't do it. So where did you end up on that protein mTOR?

[00:43:01] **Matt:** So I think those are actually two different questions because I think mTOR, first of all, I'm not convinced that getting mTOR as low as you possibly can is the right answer.

[00:43:11] **Dave:** It's bad. It's bad.

[00:43:12] **Matt:** There's an optimum to everything. But I also think that we need to recognize that mTOR cycles, like many other molecules in our body in terms of activity, and there are natural cycles of mTOR activity that are different in different tissues, in different organs. And the optimal level of mTOR is probably going to be different in different tissues and organs.

[00:43:31] You talked about muscle. We know that in order to build new muscle, you need to activate mTOR. So the optimal level of mTOR in your skeletal muscle may be different than the optimal level of mTOR in your kidney or your brain. And we just don't know. I think this gets

back to how little we know. So here's the way I think about it. I think we know that dietary protein, again, this is challenging because it depends on what else you eat.

[00:43:56] **Dave:** It depends on what kind of dietary protein.

[00:43:58] **Matt:** Yeah, absolutely. But I think if you look at people who are eating a high-quality diet, I think in general, this is my perspective of the literature as it exists now, a higher relative amount of dietary protein in combination with resistance training is a very good strategy for maintaining and growing muscle mass as you get older, and bone density.

[00:44:20] I'm glad you actually mentioned that because a lot of people only think about muscle. But if your bones are crap, it's not going to matter how much muscle you've got. So I think this is actually really really important. And I worry that this-- again, you get some people out there who simplistically think about mTOR, and they're like, well, just don't eat any protein. Low protein is good. I worry we're misleading people.

[00:44:40] **Dave:** It's destructive to be on low protein. So over the last, about three years now, I've tested the 0.6 to 0.8 grams and said, I think it has to be nonsense because of this carbs versus protein thing. So I've been doing reliably one gram per pound of body weight, or a little bit more.

[00:45:03] And I also looked at my aging clock, and the end of my 0.6 to 0.8 kind of things based on my longevity book, I was at 0.69 for my rate of aging. And we said, do those matter, do those not?

[00:45:18] **Matt:** This is the DunedinPACE.

[00:45:19] **Dave:** Yeah, exactly. DunedinPACE. It's part of the TruAge. My audience, most of them heard the episode on the TruAge test. just to figure out, it's like, hey, I need a metric. It's better than telomere. And they have about 20 other metrics for aging. Then I'm tracking. I'm going to do a big post on that that I've done, things like PulseWave and P300D. There's all kinds of things that are correlated with age.

[00:45:42] They don't cause it, but they show is the ultimate output of your system like a young person or an old person. So looking at all that, after three years of absurdly high, 200 grams of animal protein every day, my rate of aging is 0.72, which is the same as Bryan Johnson's on his

non-vegan diet, because he's eating 20 grams of collagen protein every day, which comes from cows.

[00:46:09] **Matt:** I have no idea what Bryan Johnson's doing. I completely ignore that whole thing.

[00:46:13] **Dave:** I interviewed him. He's a very fun person to talk with because he's so unusual.

[00:46:20] **Matt:** Yeah.

[00:46:21] **Dave:** And I don't agree with--

[00:46:22] **Matt:** I'm not trying to bash him. Look, I like the fact that he is making his data available to people. I think that's great.

[00:46:27] **Dave:** I do too.

[00:46:28] **Matt:** There's a lot of noise.

[00:46:30] **Dave:** Well, there's a lot of noise, and also we're all biologically unique. And so for me, I haven't published my list of 150 supplements that I take. And the reason for that is that I don't want people copying me. I have people who worship the ground I walk on. Guys, could you stop?

[00:46:46] I'm a knowledgeable guy, and I'm inspirational, all that stuff. But you didn't use to weigh 300 pounds, have autoimmune conditions and all the weird stuff that I did. And you don't need excessive amounts of B6, and zinc, and biotin genetically the way I do. So if you do what I do, you'll probably at best shit your pants. And at worst, you'll feel like crap for a couple of weeks.

[00:47:06] So we have to customize this to you. And I'm working on tools for you. And you must see this in your Optispan because some people come in, and this person just needs a couple of things. So I don't want to do what a guru does. I want to do the things the guru does for the reasons the guru does them, right?

[00:47:24] **Matt:** Yeah. And one of the things that I like to emphasize is whenever possible-- I think biomarkers are where it's at, for sure. And whenever possible, measure, intervene, and measure again. In other words, you want to know where you're at. You want to know whether

whatever the intervention is that you're doing is working, and you want to get yourself into what we think is the optimal range. And too many people, I think, just start randomly doing stuff. They don't ever measure in the first place.

[00:47:52] **Dave:** Yeah.

[00:47:52] **Matt:** And I think that's something that can be okay, but can also get you in trouble.

[00:47:59] **Dave:** It can. And I'll say, look, if you have very little money, taking vitamin Dake, D, A, K, and E, it's probably better then not doing anything, even if you don't have a blood test.

[00:48:11] **Matt:** Sure. But the blood tests are not that expensive. So again, this is, I think--

[00:48:16] **Dave:** Hold on. Half of households in the US don't have $1,000 to spend on emergency expenses right now. Biden has stripped our currency by just printing a lot of dollars. So for some people, a 50-dollar vitamin D test really is a lot.

[00:48:33] And a lot of people listening are in that situation right now, and they're saying, well, how many supplements versus the quality of my beef? So it's like, you're there. Don't get the test, and do the lowest cost thing. That's likely to move the needle and hope, but I sure want you to get the test. That is it.

[00:48:45] **Matt:** I think that's fair. Look, I think we could go down a whole rabbit hole on healthcare disparities of which there are many, and they are real, and it sucks. That's the reality of the world we live in. Yes. If you absolutely cannot afford the test, you're probably better off being probabilistic and saying there's a good chance I'm deficient. Take the supplement.

[00:49:04] **Dave:** Yeah.

[00:49:05] **Matt:** I think a lot of people who claim they can't afford the test actually don't know how much the test costs and could stop going to Starbucks and getting their 10-dollar coffee every day.

[00:49:12] **Dave:** Yeah, just skip two drinks one night a week, and you'll be fine. I agree with you. People allocate things differently.

[00:49:18] **Matt:** So anyways, we were talking about rapamycin to dogs.

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[00:49:23] **Dave:** You're a broad spectrum, knowledgeable longevity guy, so I'm having fun with you. Talk to me about TRIADs.

[00:49:29] **Matt:** Yeah. So rapamycin, we know from the preclinical literature and laboratory animals, it seems to affect biological aging across the evolutionary spectrum. And it occurred to me, again, this was probably 2013, 2014, that that there were actually several things we knew could increase lifespan and healthspan in mice.

[00:49:50] And those things almost certainly would work in dogs. And I've been a dog person my entire life, and I suddenly realized, holy crap, I could probably extend the lifespan of my dog and other people's dog, but we need a way to actually test it.

[00:50:04] And so that's how TRIAD evolved, was as really an effort to do a rigorous double-blind, placebo-controlled clinical trial in dogs to answer the question, does rapamycin increase lifespan and improve health spend metrics in dogs? And so we designed the trial. It took about five years to get funding to actually do that trial, and then we started enrolling dogs, and it's happening right now.

[00:50:30] **Dave:** Any preliminary data you can share?

[00:50:32] **Matt:** Yeah, so we've done two what we really thought of as safety trials, so these were shorter term. So the first one was 10 weeks, the second one was six months. Again, randomized, double-blind, placebo-controlled. So a few things I can say with certainty.

[00:50:48] So we did not see any significant adverse events in any of the trials to date. So we're pretty confident that rapamycin, at least at the doses we're testing, can be used safely in dogs. We found statistically significant improvements in two measures of age-related heart function, both left ventricular function that was done by echocardiogram in the rapamycin treated dogs compared to placebo.

[00:51:12] And in both trials, there were increases in owner reported activity. So a little bit weaker data because this was owner reported, but again, the owners were blinded, so they didn't know if their dog was getting rapamycin or placebo.

[00:51:25] So I think we, again, can be pretty confident about safety, and there's some evidence for improvements. I don't know anything about the outcomes from the big trial now, from TRIAD, because that trial is still blinded.

[00:51:38] **Dave:** It looks like you guys designed it to detect a 9% change in lifespan.

[00:51:42] **Matt:** Yeah. So it was originally designed to detect a 15% change in lifespan. That was 230 dogs. And then a few years ago, a group of donors came together. Peter Attia actually put this together, along with Tim Ferris and a couple of others, to increase the size of the study to 580 dogs.

[00:52:02] **Dave:** It's worth it.

[00:52:02] Matt: Which gives us the 9%.

[00:52:04] **Dave:** Yeah. So 40 to 110 pounds, you basically want big dogs, at least seven years old. What about guys who like little dogs who think they're big?

[00:52:12] **Matt:** Yeah. So this gets back to the idea that big dogs age faster than small dogs do. So in order to have that statistical power, we needed to have dogs that were aging more rapidly and already in middle age in order to be able to see an effect on lifespan within the three-year window of the trial.

[00:52:34] **Dave:** Okay, cool. All right, and humans taking rapamycin. I've done it on and off. Do you take it?

[00:52:39] **Matt:** I am right now. Yeah.

[00:52:40] **Dave:** How often and the dose?

[00:52:41] **Matt:** So I tend to cycle rapamycin. I've done typically 10 or 12-week cycles, and I'm honestly pretty non-scientific about it. I take it when I feel like it or when things in my life are lining up. But I've taken it I've done six milligrams a week, 10 milligrams a week, eight milligrams a week, so in that range.

[00:53:02] The first time I ever took rapamycin was because I was having some very severe shoulder pain. And after a long drawn out process, got it diagnosed as frozen shoulder or

adhesive capsulitis, so inflammation in the shoulder capsule. The doc really said there wasn't much that he wanted to do.

[00:53:22] **Dave:** There's so much you can do.

[00:53:23] **Matt:** He said, go back to physical therapy, and it might go away in a year. And I was horrified by that because I couldn't sleep, couldn't throw a ball with my kid. And I started thinking, I know about something that is really good at knocking down age-related inflammation.

[00:53:38] So I set up my own N of 1 experiment, six milligrams a week. And I had done it. I planned from the very beginning that I would give it 10 weeks and see what happens. And within a couple of weeks, there was a very significant reduction in pain. And by the end of the 10 weeks, I'd say 95% range of motion, almost no pain hasn't come back. So for me, it was a very important quality of life changing experience.

[00:54:04] **Dave:** It drives me nuts because I was a biohacker when I see that. I'm like, well, have they tried shockwaves? Have they hit it with ozone? Which is very cheap, 150, 200 bucks. And surprising evidence for that. And then have they done hydro dissection?

[00:54:18] Have they done all the stem cell things you could do or just exosomes? And there's all these things that don't take a year that affect quality of life, but all of those, except maybe ozone are more expensive than rapamycin. So if you can knock it down with a pill, I'm all over that.

[00:54:36] I noticed when I went above six that I tended, because it's immunosuppressant, to get more colds and things like that. I typically don't get them very often. I'm very robust now. I used to get them all the time when I was younger. So I guess that there's a dance there that you have to do based on the states of your mast cells whether you have a long coat fit or mold exposure, like I did.

[00:54:58] **Matt:** I think there's a lot of individual variation just in both uptake of rapamycin and the rate at which it is cleared. So we know that rapamycin is metabolized by something called the cytochrome P450 family, which is also involved in a lot of drug metabolism. So some people are just faster metabolizers than others. And this is where, unfortunately, we just don't have much good data on individual responses to off label use of rapamycin.

[00:55:27] **Dave:** I have a question about the dog study that just popped into my head. Are you tracking whether the owner of the dog is a couple versus a man versus a woman?

[00:55:35] **Matt:** Oh, that's a good question. So I think the answer is yes, but indirectly. So we do get information about number of people in the household.

[00:55:47] **Dave:** Okay.

[00:55:48] **Matt:** So we get both number of adults, number of kids, and other animals. I don't think we ask ever about that spousal relationship or anything like that in any of our data.

[00:55:59] **Dave:** It's more what sex are the people I'm thinking about.

[00:56:01] Matt: Oh yeah, we get that. Yeah.

[00:56:02] **Dave:** It'd be interesting to look at whether there's any information in that because you might've seen the studies that show when a woman feeds the lab mice, they get an entirely different stress response than men.

[00:56:15] **Matt:** Yeah, absolutely. There's this pheromone thing going on. Yeah. Super interesting there. Yeah.

[00:56:20] **Dave:** For that matter too, if a woman in the house with a dog is on birth control versus not on birth control, we would know. What's going on with the confounding variables we just don't know about, AI should solve that for us in about three years.

[00:56:34] **Matt:** You're very optimistic.

[00:56:36] **Dave:** Well, it's that my field is computer science. And understanding exponential growth, we still don't think of it well as humans, but this is an exponential. This is an exponential of an exponential right now. So when you go really deep on that stuff, you just realize the world's going to look really different every year going forward.

[00:56:56] Yeah. I'm pretty sure we'll both have jobs, and I'm pretty sure that next year we'll both be sitting at home, and it'll look like we're in an interview right here. The stuff that's coming down is crazy. I am in the very late stages of creating an AI that has every word I've ever spoken, every research paper I've ever used, and all of my books, and this is 1,200 interviews on my show, and probably another 1,000 on other people's shows, along with a bunch of other research.

[00:57:29] So that when people come in and say, I want to do what Dave does, I'm like, no, no, no, do what Dave would recommend based on all of these interviews. What would Dave's guests recommend? And then it's even down to the point of saying, well, how much energy do you have to invest? How much suffering are you willing to endure? Because some things are harder than others, right?

[00:57:49] **Matt:** Sure.

[00:57:49] **Dave:** Are you going to tap into your bone marrow the way I did a couple of times? Probably not. And then how much time, and how much money are you willing to put in, and what is your goal? So that's ultimately where healthspan and life extension end up.

[00:58:03] If your goal is a healthy healthspan, and you want to look a certain way, your recommendations are going to be different than someone who says, I don't care how I look, make me live twice as long. Or I want to be the brain from Pinky and the Brain. Okay. Totally different set of supplements. And I think we're there with our knowledge, but our ability to filter and sort the knowledge, you've said for 20 plus years.

[00:58:30] **Matt:** I don't know. I think you've got more confidence in the quality of the data that's out there than I do. I have done enough scientific research in a lab and seen enough data from other people to be pretty skeptical about the quality of a lot of the data that we've got. And so my worry is that, yes, an AI could come to answers based on those data, but are they going to be the right answers or not?

[00:58:58] **Dave:** So we actually agree violently on that one. Was it the chief of the British Medical Journal just said we've reached a crisis point? You cannot reproduce most of the studies that are out there because of corruption.

[00:59:13] **Matt:** Yeah, I don't know that it's because of corruption. That I would push back on.

[00:59:15] **Dave:** I'm probably adding that part, but he says because of special interests.

[00:59:18] **Matt:** I think there's all sorts of reasons why there's noise in the scientific literature. There's some corruption, no question about it Some of it is just the biology so complicated that-- and the effect sizes are within the margin of error. There's a lot of bad statistics.

[00:59:33] **Dave:** Oh, yeah.

[00:59:33] **Matt:** And there's some just bad training. I see this in my field. You can follow people who were not trained to do rigorous science, who don't do controls, who intentionally misinterpret, or they don't even realize they're intentionally doing it.

[00:59:48] They've been trained to leave out data that doesn't fit the model. So I think there's a lot of reasons. It's not intentional corruption in many cases, but the end result is the same.

[00:59:57] **Dave:** 100%. It's also just human biases. The journal of failure is essentially what we need, and there's a few people working on that who say, here's all those studies that didn't work that wouldn't be published so we could know it doesn't work.

[01:00:09] Matt: Right. That would be extremely valuable, I think, if we could somehow capture all of that data in one place. I would agree with you. Then the computational approaches, the new AI tools could in fact query that data. And I think even if there's a lot of noise, if you've got a large enough data set, these tools can tease out the signal.

[01:00:29] **Dave:** Oh yeah.

[01:00:29] **Matt:** My worry is that at this point, there's so much in there that's just not right. We're going to end up at wrong answers.

[01:00:40] **Dave:** Well, now you said something in there, a little nugget, that's behind-- it's one of the main reasons that I started the biohacking movement. If you have a large enough set of data, you'll get the truth. So the reason I'm happy about AI's ability to do this, it's not that AI is going to believe half the garbage that's out there.

[01:00:57] My AI, I actually only train it with studies that meet my standards. So I'm overweight the ones that actually make sense within all the frameworks we're working in. Understanding I have my own bias, but if you like my bias and my results are good, okay, fine. Maybe my bias is better than someone else's, or not, right?

[01:01:14] **Matt:** Sure.

[01:01:14] **Dave:** So we'll end up in this world where we have competing sets of data. And you have your Optispan set of data and principles and recommendations and your AI. So here's what we would do. And then you get Peter Attia. He doesn't need AI. He's like, just exercise more. That's all you can do.

[01:01:29] **Matt:** Come on, stop bashing Peter.

[01:01:32] **Dave:** It's so fun. Anyway. Yeah, I'm teasing him at this point. Oh, I forgot statins are good too. There you go. These are the most controversial things. But he's going to have his own things. And he writes about it mTOR, and doesn't use testosterone, but recommends it for people.

[01:01:49] I've been on testosterone since I was 26 because my levels are lower than my mom. It seemed like a good idea. So you go through all these recommendations, and you're also going to have these radical vegan doctors.

[01:02:01] Okay. We all have our ability to make recommendations based on our perceptions and our analysis of what science is "real". And then the AIs, though, you can tell them, hey, find all the studies that are in your training data that don't match with each other. And then let's understand the mechanisms behind it.

[01:02:22] And it's already capable of doing that. I have worked with ChatGPT 4, which is not a great AI, to design a new molecule to do something I wanted it to do. This is crazy. I didn't have it manufactured because I have a lot of projects on my plate. But now I know how to do it. And you're going, this is incredible.

[01:02:39] So I'm very hopeful. And my only concern is that we're going to just train them to take money from other people, take attention from other people, and kill people we don't like. That would be wrong. So I'm working on that side of making AI about human flourishing, along with AI flourishing, instead of human power.

[01:02:59] **Matt:** Yeah. That's a fantastic idea, and I align completely with that.

[01:03:03] **Dave:** And I believe it's going to make your job as a PhD researcher and CEO of Optispan easier and better.

[01:03:09] **Matt:** Optispan.

[01:03:11] **Dave:** Jeez. I'm sorry.

[01:03:11] **Matt:** We are not scanning people's eyes.

[01:03:13] **Dave:** Optispan. I got it. Sorry. For some reason, those two words are on my mind. When I say them, I see it.

[01:03:19] **Matt:** Although, retinal scans, I think, are super interesting data type for health span and potentially longevity prediction.

[01:03:25] I think your webcam and your Xbox camera on your eyes, it's an untapped part of biohacking, so we agree on that. Just the ease and regularity of the way your eyes sweep tells you so much about the nervous system.

[01:03:38] **Matt:** Yeah.

[01:03:39] **Dave:** Most people don't know that.

[01:03:39] **Matt:** There's lot of cool stuff there.

[01:03:40] **Dave:** You know all the good stuff. You also know about something called the Million Molecule Challenge, which I'm a big fan of. Tell me what you're doing with that.

[01:03:50] **Matt:** Right. So the goal of the Million Molecule Challenge is very simple. It's to test a million interventions on longevity using the nematode C elegans as our starting point. And the reason for this, I think, first of all, in my mind, it's obvious. We have only explored a tiny fraction of the intervention space, and we are absolutely going to find a whole bunch of extremely cool, large effect size stuff if we screen a million interventions.

[01:04:17] But there's a bigger reason for this, which is that in my view, the field unfortunately has actually narrowed over the last 15 years instead of expanded. And I think the hallmarks of aging, which I like as an artificial construct, have contributed to this.

[01:04:32] So the hallmarks of aging, I know you know, but in case your viewers don't, are a set of originally nine, now 12, aspects of biological aging that a group of people in the field have settled on as being the core mechanistic features of aging. And they include things like your favorite telomere shortening, senescent cells, mitochondrial dysfunction, deregulated nutrient signaling. I could probably get 10 of them. I won't get into all.

[01:05:01] **Dave:** Intracellular junk, cellular stiffening, loss of stem cell volume. Yeah.

[01:05:05] **Matt:** There's the three new ones, dysbiosis. And I don't remember what the other ones are.

[01:05:09] **Dave:** Those didn't exist. When I wrote my book, I had seven, and then nine and 12, and they'll probably be 18 next year.

[01:05:15] **Matt:** Right. So that's the point though, is that I think the adoption of the hallmarks so broadly by the field has sort of forced people to think about their research within that context. And it has become very, very difficult for people to think outside of the hallmarks or maybe more importantly get funded to do research outside of the hallmarks.

[01:05:35] And so the consequence of that is nobody is really doing the large scale discovery science at this point to find what don't we know. That's really the point of the Million Molecule Challenge, is to say, okay, let's not pretend like we completely understand the biology of aging.

[01:05:52] Let's go find out what we don't understand. And in my view, if we're going to do that, the thing you want to do is you want to be able to look at scale, so you need technology that allows you to look for interventions that have a big effect at scale, and we've created that, and you probably want to use lifespan as the end point you're looking for because that is what we're most interested in.

[01:06:15] **Dave:** But I thought you couldn't extend lifespan.

[01:06:16] **Matt:** You can absolutely extend lifespan.

[01:06:17] **Dave:** Don't tell Peter. I'm just messing with you at this point.

[01:06:21] **Matt:** Yeah, no. Look, we've been able to successfully extend lifespan in every animal where we've tried. There's no reason to think humans are any different.

[01:06:28] **Dave:** Now, you created something called the WormBot.

[01:06:31] **Matt:** Yeah.

[01:06:31] **Dave:** Which makes me happy. And listeners might remember the company behind a peptide called OS-01 that apparently reverses cell aging in skin. They looked at something like 600 different potential interventions using skin on a chip, basically, to figure out what worked. So one relatively small company looked at 600 things and found one that worked.

[01:06:55] And you're saying, well, all of the companies here, and you're providing the tool, this WormBot, which is essentially the-- I guess there's some biology from nematodes on a chip? How does the WormBot work?

[01:07:06] **Matt:** No, no. So the WormBot actually doesn't involve a chip at all. So this is using the standard, at least currently, conditions people have always used for aging studies and worms. These are nematode worms. They're about a millimeter in size, so tiny, crawling around on the surface of a plate, and they eat bacteria as their food.

[01:07:23] So in the old days, people had to sit it in a microscope. And in fact, as the worms get older, they slow down. So it's funny. You actually use a wire pick, and you tap them on the head to see if they're still alive. So it's like hitting a person with a telephone pole to see if they're still alive.

[01:07:38] **Dave:** That would take forever too. Oh my God.

[01:07:40] **Matt:** Yeah. So it's extremely time intensive. So we just created a robot that has a camera attached to it, and it takes pictures of the worms over their entire lifespan every 10 minutes. And then we use AI to interpret those pictures and tell us are the worms still moving? If so, how much, how fast? Are they still alive?

[01:07:57] So it's all automated. So it takes us from being able to do a couple dozen experiments in a year to a couple hundred thousand in a year. And it's infinitely scalable. So a million is just a big number. There's no reason you couldn't do five million or 10 million.

[01:08:14] **Dave:** How much does it cost to have a WormBot?

[01:08:16] **Matt:** Yeah, so the WormBot itself probably only takes a few hundred dollars to build in parts. Obviously, there's people required to do it. The AI tools are a little bit more sophisticated, but it's nothing that a computer science major couldn't do.

[01:08:30] **Dave:** So it's like a quarter million bucks?

[01:08:32] **Matt:** To do the Million Molecule Challenge?

[01:08:35] **Dave:** To build out all the-- if I want to run in my living room, I got--

[01:08:37] **Matt:** So if you wanted to build a WormBot factory, which is what-- so we didn't talk about this. Ora Biomedical is the company we spun out of my lab.

[01:08:45] **Dave:** O-R-A.

[01:08:46] **Matt:** O-R-A. It's the Rapa Nui word for health and vitality. So is the company we spun out of my lab to do this at scale. And so the goal there is to build an army of about 40 WormBots and use that army to assess the effect of a million interventions on longevity.

[01:09:02] **Dave:** If you made a t shirt that said WormBot Army, I would pay 50 bucks for it to support your research.

[01:09:07] **Matt:** You got it. In fact, maybe that's what we should do. So if people are interested in learning more, you can go to the Ora Biomedical website. There's a video there where I talk about this in a little bit more detail. You can also actually sponsor an intervention. So we have a leaderboard. I don't know if it's up yet.

[01:09:24] I think it's now up. Where people can sponsor interventions. You can pick your own. Or you can just let the computer randomly pick them for you. And then you get your data back. And so the goal here is actually to do a service for the field, even though it's a for profit company.

[01:09:40] The goal is to create a database of at least tens of thousands of interventions that people have sponsored, to make that open access so that people can use the AI tools to query that database and find new things. So I think there's just so much potential here.

[01:09:55] **Dave:** This is so cool. See, that's a contribution to the field in a big way. Thank you for doing that.

[01:10:00] **Matt:** So believe me, this is a source of immense frustration to me as I tried hard to get this funded on the academic side and just couldn't do it. And finally, it got to a point where I was just so frustrated that I'm like, okay, we got to take this outside of academia or it's never going to happen.

[01:10:15] **Dave:** Well, I'm just thinking I would definitely fund glyphosate. Of course, if it had a huge negative effect, that might be useful data.

[01:10:24] **Matt:** So actually, it's interesting.

[01:10:26] **Dave:** I'll give it to RFA.

[01:10:27] **Matt:** I'll tell you an interesting-- I don't know if it's interesting. It's amusing to us. we've built the WormBot in my lab at the University of Washington, and we first started doing a little bit of screening. And of course we find things that shorten lifespan. We find more things that shorten lifespan than extend.

[01:10:42] One of the first things that we found-- we were looking at some FDA-approved drugs, and this was probably 2021, maybe. So we were still in the COVID-19 pandemic. It might have even been 2020. In fact, it was because it was before the election. And we found this thing that shortened lifespan to half a day. We looked at it, and it was ivermectin. And we were like, whew, I'm glad that didn't extend lifespan.

[01:11:09] **Dave:** Yeah. Well, for nematodes, you would expect it to. That's what it's for.

[01:11:12] **Matt:** That's exactly right. So we're going to own the market on anthelmintic drugs, right? We're going to find all sorts of stuff that shorten lifespan in worms.

[01:11:18] **Dave:** I was laughing because I am a sheep farmer, and we have a bunch of ivermectin. You give it to the sheep because the nematodes make them sick.

[01:11:25] **Matt:** Right, exactly. Yeah, yeah. So it was actually a nice positive control that we had in there.

[01:11:32] **Dave:** And of course you didn't have to enter the land of controversy by saying, oh my God.

[01:11:34] **Matt:** I was just thinking, do I have to publish a paper that says that ivermectin extends lifespan and slows aging? It would have made headlines. I will give it that.

[01:11:42] **Dave:** I do know some people who believe it probably does, most likely by reducing the effect of parasites that we don't know we have.

[01:11:48] **Matt:** Sure, right. And again, that's plausible. I'm certainly not recommending people start taking ivermectin.

[01:11:53] **Dave:** I think fenbendazole is a much better drug for longevity. I take that every three months. I do a course of fenbendazole because before you get to the seven, nine, or 12 hallmarks of aging, you get to the don't die. And if you look at the big four killers, that's what I call them. Others call them four pillars, but it's cancer is one of the big four. And it looks like fenbendazole has a very meaningful reduction in cancer in humans, but it is an anti parasitic for dogs. It's not approved for humans.

[01:12:26] **Matt:** Yeah. I should go back and look. So I personally sponsored a bunch of molecules, and I know fenbendazole was one of them, but I think it was in combination with something else.

[01:12:36] **Dave:** You know all the cool stuff.

[01:12:37] **Matt:** We should have that data. Yeah.

[01:12:39] **Dave:** This is really fun to talk with you because sometimes people will come in, and they're not PhD researchers like you. I'm not a PhD researcher. I'm a researcher, but--

[01:12:47] **Matt:** PhD researchers are overrated. Believe me.

[01:12:49] **Dave:** Oh, it depends on what's your research, but it's cool. You know the little corners where I wouldn't expect most people to do that. And it's because you're working with really three different of the most important markers. You're looking at the early stage, which is nematodes, and then you're looking at dogs, and you're looking at people, and you actually have active projects in each one. So I think you have a different view than almost anyone I've met.

[01:13:11] **Matt:** I've made a dedicated effort to try to be broad, which I think that the flip side of that is I can't be deep on too many things. And it's interesting. And to me this is a source of a little bit of, I don't know, frustration is not the right word, anxiety, maybe, that 10 years ago I felt like I had a pretty good grasp of everything that was happening in the field. I don't feel that way anymore.

[01:13:33] **Dave:** You can't.

[01:13:33] **Matt:** It's grown so much, which is great. I am passionately a believer that we need to do this and that growth in the field is a good thing. But it has made it harder for me to really keep up with a lot of the cutting-edge stuff.

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[01:13:50] This is something that deserves a little conversation here because I think it's affecting you, and it's affecting a lot of listeners. In the early, early days of the internet and the web, back when I'm doing that e-commerce thing, there was a time where I knew every website, every single website, because there was only five, and then there was 10, and then there was 100, and it grew. And I went through this period of crisis.

[01:14:16] And I'd say I was about 23 because I would stay up later and later. I'm like, there's so much new knowledge and information. And I was trying to scale my knowingness of my digital environment with my time. And eventually, I think I was an information addict, what they call email addiction. Now it's just mobile phone addiction.

[01:14:36] And I had a problem with it. And just understanding that there is no way any human, even the very smartest and best of us is going to know everything online right now. And besides, half of what's online is garbage anyway. So the only thing we can do is learn to be comfortable and not stressed with that. Because the stress will kill you.

[01:14:55] **Matt:** And I think recognize your limitations. And that's, I think, where a lot of people fall down, is they don't recognize what they don't know and get themselves in trouble. And I try hard. We all do that. But I try hard to be very humble about my lack of knowledge and recognize that there's way more that I don't know than I do, even about aging.

[01:15:15] **Dave:** Even about aging. And it's also important then. And a big thing that I look at at my job is to curate knowledgeable people who have a good view of the future and have different sets of visibility into practices and all that, so that listeners can come in and say, all right, that feels like a good direction, because the network of all the guests who've been on the show is hundreds of thousands of times more knowledge than I have, even though I'm pretty knowledgeable.

[01:15:43] So it's like, how do we curate eventually to be the best the best experts like you and the best AI tool sets because there will be millions of different AIs that believe different things over the next couple of years.

[01:15:55] **Matt:** It's pretty interesting to think about.

[01:15:57] **Dave:** Yeah. How do we filter that? And it turns out the best filter, that your biology is a filter of reality. It's throwing out a bunch of data before it hits your brain. Well, we need to build systems that are throughout data before it hits our brains. And that's as important longevity as having a dog. Otherwise, you're just like, I'm constantly bombarded. I can't sleep. And then that stress takes away your life.

[01:16:19] **Matt:** Yeah. I think the real challenge right now, as you've alluded to, is the massive amount of information out there and sifting the signal from the noise and particularly the signal that's right for you is really, really tough.

[01:16:32] **Dave:** Nice. I think a lot of listeners are going to be extremely interested in getting their dogs in, and I should mention, my assistant here in Austin, her dog, Dedham, is in--

[01:16:43] **Matt:** An awesome name, by the way.

[01:16:45] **Dave:** It is a really cool name for a dog. It's because she was in the Coast Guard. And she was saying, Dave, the amount of information they were gathering was insane. So kudos for getting all the info you can. Can people listening enroll their dogs to support your project?

[01:16:59] **Matt:** Yeah, so the project website is dogagingproject.org. There's a little button. I think it's up in the upper right-hand corner, that says "Nominate My Dog". You click that, it asks five real simple questions, and then you will get invited to your owner portal, which is where you can, uh, you get access to the comprehensive annual survey. And that's called the health and life experiences survey.

[01:17:21] So I'm sure that's what she was referring to. And it is pretty comprehensive. So it takes people about an hour to two hours, depending on sort of the branching trees that they go down, to complete the survey. Fortunately, it's in 10 modules. Don't have to do it all in one sitting. And then once you've done that, you are part of the Dog Aging Project path, and you are a community science participant contributing to healthy longevity in dogs and healthy longevity in people.

[01:17:46] So it's a super fun project. Some of the dogs will be eligible for what we call sampled cohorts. So 10,000 dogs get their genome sequenced. 1,000 dogs are moved into what we call the

precision cohort, where every year you get sent a sample kit that you take to your veterinarian to collect blood for epigenome, metabolome, fecal microbiome, things like that.

[01:18:09] So we get a comprehensive data set on 1,000 dogs every year. This is a longitudinal study. So we aren't asking, unless you go into the clinical trial, which is TRIAD, which we talked about, that's only 580. Everyone else is in the longitudinal study of aging. We don't ask the owners to do anything different than they normally would, but we collect data, and in a subset of cases, biological samples, really to try to understand, again, what are the most important environmental, genetic factors that influence health outcomes and longevity in dogs.

[01:18:40] **Dave:** Beautiful. Matt, thanks for asking the hard questions for many years. You're the only guest I've had out of almost 1,200 who's done real meaningful work across all of the different models for aging, including the human models. And I think that's unique and interesting. And cat people, I'm sorry, but it seems like cats reproduce so quickly. They don't have to live longer.

[01:19:05] **Matt:** I actually think there should be a cat aging project, but I'm not going to do it.

[01:19:10] **Dave:** We finished the interview, but we were talking before it started that one of the problems is it's really hard to get a cat to take a pill.

[01:19:18] **Matt:** Yes. I often get asked the question, why isn't there a cat aging project specifically in the context of the rapamycin trial? Why aren't you testing rapamycin in cats? And first of all, I'm a dog person. That is the honest answer. But have you ever tried to get a cat to take a pill? Because the owners give the dogs their pill every week. And so it is just much more practical to think about designing this kind of a clinical trial in dogs.

[01:19:43] And here's something for the cat owners. Cats tend to live longer than dogs. So again, thinking about the statistics of how long do you have to do the trial? How many dogs do you need? It would be harder to do that. But I do think it is important to mention that there's no biological reason why rapamycin is going to work any differently in cats that it does in dogs or in people than it does in dogs.

[01:20:04] **Dave:** You reminded me of an episode probably in the first 300. I interviewed a veterinarian who was looking at the effects of MCT oil on health and longevity in dogs and cats.

And I've been suggesting ever since then, add a little MCT to your cat food or your dog food, and watch your dog's food cravings go away. They will eat less poop too.

[01:20:22] **Matt:** Yeah. That's interesting.

[01:20:24] **Dave:** Just because I think their energetics are better. So a lot of things do apply to both, but not everything.

[01:20:30] **Matt:** Sure.

[01:20:31] **Dave:** Awesome. Thank you, Matt.

[01:20:32] **Matt:** Yeah. Thank you. It's been a pleasure.