The Science & Economics Of Infectious Disease And Aging Transcript

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Introduction and Guest Background

– Welcome to another episode of the reversing, implementing summit Body and mind longevity, medicine and I'm your host Dr. Robert Lufkin, we'll be joined today by my co host Dr. Stephen Sideroff today, we get to cover a wide range of topics around the science and economics of infectious disease and aging. Our guest today is Dr. Tim Peterson, who is assistant professor at Washington University School of Medicine.

His focus on longevity has led him to study Metformin and rapamycin and the M tour pathway, arguably the most validated longevity pathway known. We'll also hear about some innovative strategies using the Blockchain to facilitate longevity, drug development. Hey Tim welcome to the program.

– [t] Hey thanks for having me, I appreciate it. – [r] Yeah, so good to have you on the on the on this session today. And there's there's so many exciting things I wanna talk to you about but before we start maybe you could just take a moment and tell us how you became so interested in longevity and kind of how you got to where you are today.

– [t] Yeah so I um I've always been an athlete that's maybe somewhat of a common way for people kind of get interested in their health. Um and then you know through just training in school, I was I was in both M. D. And PhD programs.

I just stuck with the PhD because I was so into being in the lab and then um particularly in the longevity we were studying them tor pathway which is you know one of the kind of more prominent pathways in longevity. So but that was I mean I think it was early 2000 early to mid 2000. So um it really wasn't I don't think we really even knew that we were studying longevity, like the first paper I was part of what we had aging in the title, it was like 2010 I think it's kind of the first time where we were like oh we're actually studying aging here, so um so yeah that was how I got into the space and I think because I was studying M M. Two or um you know the drug rep my son was you know obviously always been key to that space and so I just really got interested in um you know drug development as a result of that and so studied Metformin and and other drugs since that time. And so um leverage that to stay in academia, some assistant professor at Washington University ST Louis School Medicine and um but in the course of work there have been able to launch two companies in this space, one health span technologies and one bio. The former is focusing on RNA therapeutics, we're making vaccines and gene therapies um and then the other ones by was a small molecule company and focusing on um it's kind of we're taking a target target idea approach.

So yeah.

Aging as a Multifactorial Problem

– [r] yeah, before we dive in maybe let's take a moment and tell us a little bit about how you look at aging and longevity and you know why do we age what causes it because everybody seems to have a little bit different view about this definitely.

– [t] So I mean I think the thing what it's really stuck with me is this kind of comforts law was this this thought that you know essentially right aging, aging and death is inevitable and this kind of mathematical formula that the probability of death is exponential. And so the what the reason that was important to me is it comes from really I think how drug development is done these days and how I realized drug development so we can kind of go in with like you know there's this whole rational drug design, the school of thought where you have to like know the target, you know that's and you have to hit the drug with high specific, sorry hit the target with high specific drug.

And that's really dominated a lot of um that's basically that's all farmer right these days. And and so I think that's you know largely not gonna work because you know aging is this, you know, again it's an inevitable thing. It's a exponential curve.

And so exponents involved multiplication. So it's really a kind of fundamental level. We have multiple things basically compounding upon each other to lead to, you know lead to our mortality. And so I think we're you know doing one, we're coming with one agent even if it's M. Two or even if it's you know, you know any D. Or whatever that's I think there's you know there's no chance that's gonna work and we see that and then with a lot of the drugs to come in come in and you know they have a 10 or 20% effect, you know a lot of the cancer drugs and those kind of things um and and diabetes drugs and whatever and you know 10 or 20% is not gonna cut it. Um And so I really think we have to so kind of the prevailing view that and that guides all, you know, my company activity in my economic work is really thinking of how we can come in kind of with a multiple I like to call them kind of multiple tiv therapies where things were you're you're putting multiple things together. Um and they're having a synergistic effect to kind of um you know, do something important. And I think that really this the fundamental example right now that is this reprogramming this Yamanaka um cocktail of factors right, took four factors to do something profound right to to reprogram a fiberglass for an embryonic cell which could then go in and make, you know every cell type.

So I really think you know there are several companies in this space which are doing reprogramming um And the common thing there is they're taking, you know whether it's chemicals or or M. R. S. Or whatever um they're coming in with multiple factors to do something important. And I think that's really the way drug development should be you know progress because like even recognizing the very best case scenario we have right now if you look at all the animal studies it's a 10 or 20% life span extension effect and then of course that will fall apart when it comes to humans.

So animals are all you know the same genetically identical. So your there's no variability there is doesn't exist. And so that's how you can get these 10 or 20% lifespan extension effects.

But in humans right we're diverse and so those gains will be lost. And so um I mean there may be some effects. You know I'm hopeful that um deadly lambing and others that you know like Wisconsin will be you know showing something valuable but I think it's just it's unlikely to be this kind of you know escape velocity that people talk about where we're living you know another 100 years or whatever.

I think we're really gonna need multiple of therapies to do that things that are you know highly synergistic and profoundly affect our physiology. – [r] So after having worked on rapamycin and and that form and you mentioned as well yeah I would assume you feel the same way about Metformin that it's.

– [t] Yeah. Yeah I mean Metformin is Metformin I think is the data doesn't look as strong as you know I think there's obviously, you know, it's a little bit like it's not democrat republican, but it's it's a little bit like where people definitely have their horse.

You know, I think they're both really interesting drugs. Mechanistically very interesting. Science is very cool, you know, both should kind of work. But I mean I think it looks like the data on is kind of clearly better. But again, I think both drugs are, you know, ultimately have a very marginal effect um as you know, as the most drugs. I mean, even like we're still debating whether statins are useful or not. Right?

So, I mean, I think again these kind of single single, you know, um targeted agents, you know, even in the best case scenario are gonna be have some kind of marginal effect, you know, equivocal effect. And so I really think we have to be moving outside that that dogma and you know, the L. S. Drug that just recently came out, it has two components. I'm not sure that date will be much better.

But it's you know, again, I think that's the start of the type of thinking we need to have. – [r] Yeah. Yeah. The idea of taking a single pill for it doesn't work for chronic disease. – [t] Exactly. Exactly. Most chronic disease are, you know, multifactorial right there. You know polygenic, you know, you know, targeting one pathway. It's just the whack a mole thing, right? You plug one hole and then you know a new hole emerges. Right?

So yeah. – [r] Yeah it's interesting. Well the work you're doing on infectious disease and aging is is interesting because it's it's it's an area that is not often covered. So maybe maybe you can just start off a little bit. And for the non scientists in the audience talk about some things like cells in essence see analytics and how those interact with

with pathogens and how how infectious disease is tied to longevity.

– [t] Yeah.

Infectious Disease, Senescence, and Longevity

So maybe I'll just I mean backtracked with a very high level thought that you know really our ability to fight off pathogens is how life expectancy has grown throughout you know throughout human history.

Right? Of course starting with like hygiene efforts and then you know um sanitation and then you know antibiotics and vaccines. I mean really if you want to look at life expectancy gains, it hasn't been through modern medicine, it's been through space even through you know, very simple public health measures um and serendipity in the case of antibiotics that we've been able to live longer. Right?

So and then you see now with covid right, you know come along and actually reduce the life expectancy in the U. S. By a couple of years. You know it's clear that you know that's a major dry and now it's you know number.

Well I think it's slightly behind heart disease but it's you know it's a major you know now new, new cause of mortality. So I think we're just kind of ignoring the influence, there's just been not a really strong um emphasis on infectious disease and it's kind of bad. I mean I think, you know, probably just again can't make money on it. It's not, you know, it's not some new fancy gene that somebody's making a drug to where it's, you know, like the Alzheimers feel, I think it's interesting because there's a lot of data emerging that pathogens um there's like a really cool science translational medicine study that said that salmonella um basically the the amyloid protein is basically trash man, essentially. It's like taking out it's like it's like fending off the pathogens that it's exposed to. So actually the thought that you'd want to be clearing the amyloid protein is like the exact opposite of what you want to be doing.

Like it's actually trying to help. And and so um you know, against against pathogens that are oddly in our brain, it's just been ignored for reasons which are not clear, but of course now with Covid, I think people are um and long Covid, I think people more included into the connections with infectious disease and aging.

And then as far as like the sun essence concept were um you know, over time as our bodies as we age, our bodies accumulate these cells which are seen essence, so they're not no longer dividing um like as opposed to our stem cells which kind of are dividing all the time and producing more healthy cells that help us.

Do you know what our bodies do? The senescent cells are cells essentially have stopped dividing and they instead of being helpful they crank out a lot of cytokines and other other kind of things that are essentially kind of dumping kind of toxic waste on our body.

So it's not totally accurate thumb but there's that's kind of the paradigm. And so um there's many reasons why cells become senescent but again like a very kind of underappreciated reason is that um you know pathogens come along and you know they kind of stress stress the cells they're kind of just another stress just like you know you you be damaged or or you know metabolic stress from you know eating too much and or whatever are um oncogene stress those things are more known they're just you know a big stress on the cell physiology and that can can lead to this this this in essence is kind of a coping strategy to basically I mean it's really an anti tumor mechanism to prevent um you know cells from kind of becoming you know becoming first so but on the other hand yeah they become that you don't get tumors but then you get these senescent cells and so um you know our our work um we did some some genome wide screens and and found some some compounds which seems specifically useful in the context of pathogen induced in essence.

And so that's that's what we're doing at my company by Ohio so and several kind of several initiatives there on those on those classic compounds. – [r] So yeah, it's interesting that the points you make that infectious disease is not just like getting sick with pneumonia and dying. But as you say with with Alzheimer's peach and marvelous or you know, Lyme disease mold all these things that can give you dementia and and essentially clinically identical. Alzheimer's disease is, is fascinating. And and you mentioned all the anchor genes.

It's amazing the number of infectious diseases that can drive cancer that were you know that we're finding now that you know can can be transmitted with these infections and all.

And it's almost like the infectious disease is closely linked with longevity like you say for the for the reasons with the sin essence and all. And it's a wrap of mice and investigator with Joan Mannix work with the Rapa logs and the immune, you know, her classic work back then. I mean just turning down M.

Two or is that going to be you know, to dial down So in essence with rapid something else is going to be the way. – [t] I think it could be. I mean I think it seems like the issue and and and talking I've been in the field for a long time but I'm certainly no expert for like today.

So it's but it's from talking to the other experts, you know Dudley and others. Um it seems like um you know, it's really gonna be about the dose and you know, maybe some people um you know, basically whether or not you're getting kind of immune protection versus immune suppression, I think, you know, could be heavily dependent on the dose, you know, how much how much you're getting and this drug is famously um you know, highly potent, right?

It's binds and very high affinity through FTP 12 to enter and it's not a full inhibitor, but you just don't need much of it. I mean it's really extremely potent.

So so I think hopefully we'll just be kind of micro dozing wrap mice and and then yeah, maybe the rapa logs may work better. Again, I'm not totally up to date with that. But you can imagine of course different catalogs being kind of slightly better on the immune profile, slightly less on the metabolic liabilities. Yeah, I mean, I think it's I mean, I think we're all going to start, I mean I talked with this kind of multiple of therapies were coming with these combination therapies, you know, reprogramming those are of course going to take a while.

So I definitely am hopeful that, you know, various rap rap mice and rats mice and analogs will be kind of the first wave that will achieve kind of broad adoption.

I definitely keen to try it. I mean I'm not taking any of them currently or Metformin currently, but if you know, data came out said, oh, you know, as long as we keep the dose low get kind of all the benefits without you know, low chance of the risk. Yeah. It's hard to say because I mean I think you know, I'm 45 so it's like I'm healthy. I mean I think the question is like, okay, do you start it when you're healthy? You started when you start to kind of you know, for some of your biomarkers start to go off a little bit.

It's hard to know. Yeah, I think it's gonna be hard for us to figure out. – [r] So I mean what do you think the biomarker it will be for a drug like rap a mission. I mean other than obvious, you know, glucose going up that you don't want. But what are the biomarkers for for longevity?

You think that our most promising because that's a big challenge, isn't it? – [t] Taking any of these drugs? Yeah. And like Nir Barzalai right is like he's trying to push the Metformin tame trial and I think, you know, do multiple biomarkers, you know, a lot of them kind of molecular, I'm pretty optimal. You know having having you know have an Apple watch and Apple iphone and the apple health app is really informative.

I mean it has all these gate measurement and has some, you know, heart rate variability and those things definitely. So like over the last year or so I've actually done, um, I really didn't do any strength training and then over last year, so I mean my gate has significantly improved, improved.

And even though I'm not like doing anything gate specific, I'm just doing like, again more kind strength training, but I think that's kind of having some neuro muscular benefits. That's not saying I'm younger, but it is a measure right? I do think, I guess probably more digital things will come out, not just through, I know all the big tech companies are interested in that stuff and of course the other reason those are great is because their consumer products, like you don't have to go to the hospital, you don't do a blood draw.

So I'm pretty bullish on digital biomarkers. You know, whether the FDA will, you know, approve them on some, you

know, timeframe, probably doubtful. But I'm kind of bullish on the thought that a lot of the medical stuff will actually be probably not.

I mean it will first come outside of the hospital system. I know like it seems like apple is really and amazon and stuff like this are really trying to make inroads in ways that probably we can appreciate.

And I do think like the hospital systems will be, may be the last to know, I mean they'll just, they'll be, they'll be a lot of digital things that happen and then I think it'll be interesting to see how it plays out the convergence of the tech and the digital health, the ai stuff and then like the kind of bread and butter, hospital based molecular biomarkers and those types of things.

I think there's going to be somewhat of a battle between those at some point. So that, you know, I mean. – [r] It's more is better with the digital information.

Hopefully the apple watch, the google watch will have the Raman spectroscopy for the glucose. I just saw a Chinese company that has a glucose monitoring watch for $100. – [r] Yeah.

RNA Therapeutics and Longevity Targets

And I know like, yeah, like you can, you know, optometry offices are getting these kind of retinal scanning for, it's not just for diabetic retinopathy, right? The quality of, you know, the visual quality of your retina can indicate all kinds of issues.

I mean, and so people are working on the ai to to do that prediction and of course it's just a snapshot of your eye. Right? And so you don't again, and having optometry office don't anything.

I mean of course you need you need the medical system if there's a real problem. But to do the initial kind of quick diagnosis, I do think a lot of will move outside of the traditional system and. – [R] They're probably mental markers for resilience, aren't there Steve that could play a role in that as well.

– [s] Well, it's interesting as you're talking about infectious disease and how you see that as an important playing an important role. And so from my perspective, stress plays a significant role in immune system functioning.

Have you considered aspect of? – [t] Definitely. I mean, I think it's, I mean, I don't have any specific, like there's not really any science, but certainly, um, I think, you know, insults, not sleeping and poor diets and other environmental stress, poor poor ventilation in your house.

You know, all those things are really, they're pretty basic, but at some level, a lot of people aren't really aware that they need to manage those, like as a scientist you want, you don't want to be anecdote driven.

But like I was, you know, I was triple vax, but then it got got covid force, it was a very mild case, but basically it happened, I really don't know what my family got it and I think I was the one who got it and like even had any symptoms because I hadn't slept like three nights in a row. Like I had huge deadlines for work like that third night, I just was so worn out basically. And then I start to get the covid symptoms and nobody in my family did. So, again, just an anecdote, but I do think the stress, you know, put me over into the symptomatic category.

Um, so, and I do think just in general, those stresses compound. – [s] We certainly see a growing percentage or number of people with chronic illnesses in our country. – [t] Yeah, for sure.

– [r] Just came across a paper this morning. We'll put it in the show notes. But it's about it's a large cohort of patients and they looked at psychological stressors and they calculate biological age and and the psychological stressors had a greater impact on biological age than the smoking history than tobacco use, which was which was interesting. So yeah, we've got a lot to learn.

Yeah. Yeah. – [t] I mean, I think they're just not, you know, those types of things are not considered kind of first

class stressors right there, kind of like, oh, you know, whatever, but I mean, it's, you know, Yeah, I think we do need more emphasis on those kind of things.

Yeah. – [r] Yeah, absolutely. Going from going from diagnosis back to therapy a little bit, could you talk a little bit about RNA therapeutics? What are they? What's the future hold?

Where are we now? – [t] Yeah, there's a lot of amazing developments, you know, of course, the vaccine was an M. RNA based strategy. So just if you're not familiar is the spike protein is a protein in the coronavirus that basically binds to the ace two protein on our cells and that interaction is what's required to get the virus inside ourselves and cause all the damage.

So the vaccine essentially um is an M. RNA to the spike protein. Our bodies make the spike protein. And then our immune system particularly antibodies um that generated can recognize that spike protein and then and then essentially block. So it's just the spike protein, not the virus itself, it's just the spike protein. And so you kind of it's kind of a decoy trigger immune system fight off the coronavirus. So um it's not perfect but it's you know, it's a it is a you know, it is a provides the the filter that would allow us to, you know in many cases to to survive being infected from from coronavirus.

So essentially the RNA approaches are ways to do kind of gene therapy and I know like people get scared if you say it's gene therapy in this case it's like a viral protein is not one of our proteins that were manipulating, but I mean it's ultimately what's so great about it is additional drugs are just are usually like whatever we call small molecule drugs, they're just teeny little things much smaller than the proteins and things that they're trying to bind to. So there's just a lack of specificity there.

Whereas with RNA therapeutics or nuclear guys based therapeutics there um like the the just specificity of the drug is much better. Yeah, the spike protein comes along and the antibodies that get made to it are very selective just for that protein.

They do nothing else in the body. So unlike a lot of the other drugs that we take where they're kind of non specifically you know blocking a lot of stuff.

Um and so that's they they're very kind of side effect prone. So I think, you know the whole idea really came to fruition came to the widespread pub attention with the Covid vaccine. But there's other there's been other efforts over the last several decades which also are really coming to head now.

So Alnylam is a company that has S. I. RNA. Based therapeutics um I think mainly for cancer but some other diseases as well. And and so those strategies often are knocking down a gene. So there's a gene that's bad in some kind of disease context. And um in this case the S. I.

RNA is something that basically recognizes the normal RNA and down regulates that the bad basically the bad protein and and will down regulate that that gene such that it's you know less likely to cause disease. Um There's other approaches um involving CRISPR which probably people have heard of it, which is a gene editing approach.

So you can deliver the CRISPR gene editing um protein as an RNA. You know, get made into a protein by ourselves. And that's another very promising approach to kind of do very very targeted therapy, you know much more targeted than any other therapy that existed. And then the other major component that has come to light is the fact that you have the R. N. A.

And that you know needs to go in ourselves and do something. But it can't just get into ourselves easily. So it has to have basically surrounded by a lipid essentially kind of a lipid particle. Um And that's um there's been a lot of interest in you know how to design those lipids and again that field really both RNA and the lipid fields really came together very nice with the code vaccine.

And other people are realizing that you know just put kind of peanut butter and jelly together and you really can make it really great sandwich that is very specific and can do all kinds of things to help you know in ways that we

haven't previously been able to do.

So kind of. – [r] you know that that's great methodology and great explanation of D. N. A. And new click therapeutics. Now I guess the follow up question to that would be um given that technology, what are the targets for longevity that if you could say knock down this gene or bind to a certain protein, how do you see it playing out for longevity then?

– [t] Yeah so Turn bio is a company that I'm part of this funding collective called beta tau and we can get into that a little bit. We help fund a company called turn bio which is doing M. RNA to the Yamanaka factors too much earlier.

These kind of four factors which can reprogram themselves from fiberglass for an embryonic state. They're doing like what many people are doing, where there's kind of partial repayments to just kind of like turning back the clock partially, not all the way to the embryonic state.

Um so they're they're not only doing these four Yamanaka which like systemic shock for, I mean I actually don't know if those are, you know this is kind of private information but I think many people are kind of focusing on these these four factors which Yamanaka got a Nobel prize for um for Kayla for scenic and camera the other one, sox two or something like this, but there's of course many others now that people have shown to be important and so those are, those are certain class of genes, ones that would kind of reprogram themselves to a younger state.

I do think, you know, going after multiple pathways kind of the canonical pathways and pathways and for pathway, you know that can also be helpful. I think people just have to see, you know, we're yeah, I mean there's just a lot of kind of well known pathways and I think probably your best bet is to, you know go after some of the kind of core targets of those pathways.

So I don't. – [r] rapamycin unlike Metformin is a very clean drug, it's very highly targeted to mentor, I mean what advantage with the, I mean obviously there are a lot of advantages but if it works effectively.

I mean if rapamycin works effectively for em tour um then you want to do a similar thing to like up regulate and pickiness or other other sensing nutrient sensing longevity related proteins. Is that what you're thinking?

– [t] Yeah I think so. I mean I think it's um you know so wrap nice is really interesting because it um again I mentioned earlier kind of partially inhibits immature so it kind of it's it's an analyst erIC inhibitor so it's not plugging the active site of the kind so it's not gonna just like kind of uniformly down regulate the mm pathway.

It kind of has some specificity but which um part of the pathway it hits more so than others. So if you were to download M Tour using an S. I. RNA approach or some kind of nucleic acid approach it would you know it would be it may still function similar optimizing but it may have different kind of kinetics or different kind of target profile. So um you know I'm not totally clear.

I mean it's still the issue with the nucleic acid therapies even though I said like the lipids come along and you know that that's a really nice delivery vehicle.

It's still not as um easy to get the kind of broad distribution.

VitaDAO and Decentralized Science

Not nearly as easy to get the broad like cellular distribution and body distribution as you get with small molecules which are small and can kind of diffuse throughout the body easily. So um I don't mean maybe m is not the best target again because like you just mentioned, there's already a drug for this and you know, it's kind of like everyone's already making good progress there.

But I mean other pathways, I mean the way it's going right now with the new play gas therapist is really going after kind of mono genetic diseases like sickle cell anemia.

There was a crisper trial. Um I guess that wasn't necessarily that wasn't an RNA approach, but nonetheless, people are going after single mono genetic diseases where you know, you fix this one mutation, you you know, you don't regulate this one protein.

You know what it's like has the desired effect. Longevity may not be the first probably is, it's already not the first basically use case because it's yeah, chronic diseases are just harder to fix and you know, so I think it'll be before you figure out the cocktail of things that will work but turns out it's pretty far along.

I mean they're so I mean there and other companies are kind of on their on their heels. So I do think um it will be um it won't be too long. So for the for longevity or more kind of chronic disease applications.

– [r] Well speaking of turn by and you mentioned this a little bit before. We we've got so many things to so many fascinating topics to cover, but I wanted to make sure we talked about via the dow and what what's a decentralized autonomous organization and how is it helping in longevity, drug discovery in other areas?

– [t] Yeah, so I mean, I think what's interesting about, I mean a lot of people, when you hear the word crypto or decentralized Blockchain, all those things, people kind of are skeptical right there. I mean a lot of people are skeptical and I actually think that's what's, there's a lot of people, the longevity field, we feel like we're kind of outsiders as well, right?

I mean, you can actually, you know, I'm an academic, you know, university and you know, you can't really use the word the word aging, right? Like longevity sounds a little bit to do like snake oil, right? Or I mean it's certainly like anti aging you can't use. Right?

So so I did and crypto the same thing, I think it's um you know, this field which is providing an alternative. You know, most people, most of us argue it's like a more fair, you know, money system, right? Where, you know, outside of the government control and anyone can do it right? It's peer to peer.

And so I think the longevity encrypted communities really have a alignment around, you know, we're kind of outsiders and not taken seriously by the mainstream was part of the basis for for for forming the dow and the essentially we're funding collective.

So the DAO decentralized autonomous organization, we're not like registered in Delaware or where, you know, it's it's a it's lives on the ethereum Blockchain.

So it's like, you know, it lives in the metaverse. Again, it's it's a new kind of new kind of concept. And people like the sense of collective where people put their money together and to fund research and it's a nonprofit meaning like any kind of profit that would come out of any drugs that would be just put back into the treasury to fund more research.

So it's not a, you know, it's potentially not trying to be something that would be a for profit company. And um, the, I mean, the other thing too is that we have a token, so a token helps us basically govern the way um to make the decisions on what gets funded.

So, um, it's called the vita vita token, and you can get it on various exchanges. Um, and the idea is that, you know, ownership in that token, um allows you to say to, to kind of chart the course of how, how we, how we spend the money. And so to me that's a really exciting way.

It's a democratized way. It's um, and what's really cool. I'll be compared to, you know, having been in the traditional, You know, academic system and, you know, the traditional way of doing things for many years is really just get people of all different perspectives.

You know, a lot of people in and the kind of crypto world are anonymous and so you don't know who they are, they don't know their credentials are. So it really makes a much more, you know, kind of flat structure and I think you really have to kind of listen to people's arguments. Um, so, so so for example, it's been really interesting. So Fighter

is has has just put has discontinued funds to beat it out recently. And there's been a big announcement on that.

And so we have representatives from Fighter in our organization and they're coming in with their with their traditional approach. And, you know, people like me push back and say, okay, well this is how Fighter does does it, and you know, but we're we're not necessarily operating that way, right? So I think, you know, it's been interesting to see, but they also have value perspective. It's like, okay, you can you can be a dreamer, you can propose, you're gonna, you know, cure these diseases with these crazy strategies.

But there is this kind of, you know, business reality or kind of scientific reality that we need to live in. So it's been fun to kind of have those worlds collide. And I think, um, again, where one is not clearly better than the other one and, and I think that's leading to some better outcomes and better conversation. You know, again, it's not a piratical driven system. – [r] So yeah, yeah, I like the idea you said that VitaDAO is not a registered Delaware c corporation.

It's a non profit, but is it registered as a legal presence at all? So it doesn't exist within our legal framework because it's entirely running on smart contracts and crypto space, which is fascinating.

It's uh, yeah, it's, it's amazing that you've been able to do this. And what are the advantages with this over traditional drug development and what are the challenges? Obviously fighters interested and they're, they're becoming a part of it, which is wonderful.

So how, how is this going to succeed where other drug, other drug projects fail that are driven by, you know, purely for profit for a fighter, let's say.

– [t] Yeah, so, I mean, you know, I think we're, I mean, we're able to do kind of different experiments, right? I mean, I think that's maybe one way, I mean, we'll see, I mean, time will tell, you know, of course.

Um, but I think one thing because of, because we're people are coming in with different ideas, you know, we're able to act in a different way. And so like for example, we funded, you know, we funded a film, which is, you know, hopefully going to be on Netflix soon where just about longevity telling kind of the story of longevity and how people are doing bow hacking and it's really kind of an awareness piece. So kind of definitely media media marketing and stuff like that is, you know, I think it's important our mission.

And so, that's not something Fighter would do, right. They're not fun to film, right? That's not something that, you know, traditional biotech VC would do. So I think we realize the power of is creating awareness around what we're doing. And um, you know, really, ultimately all the big tech companies are all, are all, you know, media companies, they're, they're not only, you know, doing R and D, but they're getting the message out.

So I think awareness is a huge part of what we're doing. We're actually in talks to create a journal and academic journal. And that's, so that's another way we can kind of establish legitimacy, you know, given the, given the intimate connection in publishing and funding, you know, the really the right, the funding drives the publishing and, and then, you know, it kind of feeds back on itself.

So I think we've got to be involved in both those activities, um, and gain legitimacy in those areas. So, yeah, I think it's, I think there's many ways, many things we can do that are kind of outside, you know, it's certainly not a biotech VC firm. I mean, it's a, it's a, it's, we can take on many different things that will help sustain the organization hopefully. – [r] And, and just to be clear, you're, you're actually going to be developing intellectual property that would be licensed as an FTS non fungible tokens and so it can actually profit and, and sustain itself and, and the investors and the patients that participate.

The other thing I wanted to underscore, especially for this audience in this program where people are listening to this because they're interested in longevity for themselves or their families. They want to understand it and VitaDAO is a way to actually participate in it. So any person can go, you know, with a little knowledge, uh, with Cryptocurrency they can purchase invited out tokens and when by purchasing tokens, they become part of divided our community community and can vote on drug development and can participate fully as a member of it.

So it's a, it's really a unique thing that the crypto currency makes possible. The true democratization of research and science and knowledge. It's really fascinating. – [r] And yeah, and I think one of the, several of us in what we're calling the deci community centralized science community.

Several of us are interested in this concept. That token would only be kind of a governance token and voting on things. We would actually be, you know, potentially getting access to medicines.

That doesn't sound right, but essentially be, you know, the token would do more and have much more utility for people give them access to, you know, discounts on supplements things where it was like I said, the simple things like give the token much more utility as to why you'd want to, you know, be part of the community and and deliver a lot more value um for for for both sides for for the people who own the tokens as well as for and other dc organizations.

So I think you can definitely currently it's you know, just voting. That's not maybe that's that's great and we're excited about that. But I think we have many more ideas in the future about how to make much more of an active participation.

So I think that's some, you know, that's really that's really what we're excited about. – [r] And I think VitaDAO is the leading the leading organization of this type in the longevity space. Are there other organizations in other science and health care similar to this is rolling out now, definitely.

– [t] Yeah. So so molecule is an organization based out of Switzerland. That's um this helping launch Daos. So there's Lab Dao, there's side Dao, there's Athena Dao so lab Dao is kind of a general purpose at least currently computational dry lab focused kind of general purpose um infrastructure Dao Lab infrastructure Dao side outs for psychedelic research, Athena Dao's for women's health issues. There's other longevity ones, the nine lives Dao there maybe a few other more fledgling ones that are emerging.

Digital Biomarkers, Social Connection, and Closing Thoughts

Nine lives for I think for cap, you know, the the idea that probably we can get to, we can get to human health if we focus on our pets first. You know, people love their pets, you know, there's um, you know, loyal, the company loyal, which is focusing on dogs, which has gotten a pretty good amount of traction.

So I think it's a great strategy. So I think definitely, I mean, I mean, I think it's definitely the other part about being, you know, a nonprofit and just, I think in general, people don't really preach about crypto. There's definitely more of a, we're all working together vibe and there's actually this kind of acronym wag me, we're all going to make it. So like people just really, it's much more about kind of collaborative environment than, you know, the traditional biotech world. So yeah, – [r] Yeah, that's, that's exciting. Well, we, we just have a little bit of time left, maybe one or two questions I, I'd love to ask you.

Other than the things we've got, we've covered, uh, what, what are one or two of the most exciting areas of longevity right now that we haven't talked about? – [t] That that are not working on. Right?

You said. – [r] Yeah, you mentioned the Yamanaka factors in the partial reprogramming. – [T] Yeah. So that's, that's one, um, you know, I think that that we're not working on, there's, I mean, I think the pets idea is great. I think the pets idea is, you know, focusing on the, I mean if your goal is to just help pets, that's actually great too. But the idea that, you know, you can get to market faster, you can prove things, you know, certainly, you know, I think that's that's a great, great strategy. I do think I kind of said this earlier, the digital health stuff where I mean we have like one project that I'm trying to get off the ground and I just think I just think digital health is, you know, there's this kind of famous you know Mark quote, you know, software is eating the world. I think that, you know, I kind of already said this, but you know, the medical kind of establishment, which I've been long part of all my families and you know, and medicine and doctors and all this stuff. I mean, I just think they're kind of a little bit asleep at the wheel, what's gonna happen with software? I think I think software in general is gonna, you know take over health care just as you know, it takes over every field. So so that's probably the biggest area where I don't have enough of our efforts that I would like to spend more time.

So and I think everyone doing work in that space is, you know, I'm cheering them on. – [r] So yeah, one more, take home thing for people, if everyone listening, if they could do one thing to improve their longevity, what would you like to start with?

– [t] Well, one, I mean actually probably the one thing that maybe people wouldn't say and well maybe a less common answer. I do think the social interactions like I think it's underappreciated how much how human, you know how much basically are kind of psychosocial well being influences our body physiology.

The one like I have a colleague how much time we have like um we need like 10 minutes sometime. – [r] Okay, – [t] so I have a colleague, an old colleague of mine that's at N. Y. U. Bradley Miller who like I'm embarrassed that I didn't know stuff like this. But I mean he showed this video where mice came together. He had he had serotonin um like a real time serotonin reporter and he had a bunch of mice and as soon as they walk together their brains just lit up with serotonin. And never I just never forgot that video because it just shows that you know, we need to be together physically um to be you know, for for our health. And it's just it's just basic, it's just basic biology basically. And so I do think we definitely need diet and exercise and those things that I do think um you know, human connections are very underrated. – [s] I appreciate you bringing that up because it's a component of resilience, uh inter social interaction, what I referred to as emotional nourishment. That's a very important aspect of mental health and I think it will turn out to be a very important part of longevity. – [r] There's just another paper out about that, how, how emotional loneliness is a risk factor for all these chronic diseases and ultimately longevity and their take home message was, you know, make friends have a partner if you have a partner, if you're in politics, get a dog, but get something, you know, but make that connection even if it's with an animal with a pet, you know? But yeah, well Tim how can people reach you on social media? What's the best way for people to follow you?

– [t] Yeah, so I have, you know, a fairly generic name, so I insert my middle initial in there. So I'm Tim R Peterson pretty much everywhere. I think my LinkedIn is actually kind of the opposite of Peterson, Tim R. But I kind of always yeah, like I said, I include that kind of, our middle initial is to stand out a little bit, you know, like Stephen A. Smith is like the famous like sportscaster guy on on ESPN and also generic generic generic name guy, but but you know, puts the a in the air in, so at least, you know, no, it's him.

My companies are like I said, our health span technologies, healthspan.devbio.tech. That's the bio tech is not live currently, but we'll have that live.

So yeah, like I said, everything on social media and stuff is just Tamar Peterson and that's my emails as well, bio tech and um, so yeah, you can reach me in many ways.

So yeah. – [s] Sounds good. We'll make sure people know about all of those ways of connected. – [t] Thanks appreciate it. – [r] Thanks so much for being on the program Tim and we appreciate the work you're doing. And we'll look for you in VitaDAO also say hi, thank you. – [t] Yeah, Thanks.