Introduction and guest background

– Welcome again everybody to the reversing inflammaging, Summit Body and mind longevity medicine today, we're honored to be speaking with Dr. Aubrey de Grey, biomedical gerontologist based in Silicon Valley and I'm happy also to introduce my co host Dr. Robert, Lufkin, – [r] Hi Steve.

It's so, so glad to be here today and I'm so excited to have Aubrey de Gray with us. He never fails to have fascinating things to tell us about his take on longevity, so I can't wait to get into it.

– [s] Welcome Dr. Aubrey de Grey. It's a pleasure to have you here along with my co host, Dr. Robert Lufkin and to kick off our discussion, we the audience would love to know what brought you into this arena and what really excites you about this area – [a] Alright? Yes. Um well, first of all, thank you so much for having me on the show, the two of you. I'm delighted to be here.

So well what brought me into the arena really was the realization at the age of about 30 or so that there were so few other people in the arena. I had always, since I was a young kid decided that I wanted to dedicate my life to One or other of the hardest and most important problems for humanity and in my early life through until about the age of 30.

The one that I ended up working on was the problem of work. The fact that so many of us have to spend so much of our time doing stuff that we wouldn't do unless we were being paid for it. You know, I thought, right, therefore um you know, we should definitely develop artificial intelligence and have more automation so that that's no longer the case.

And at the age of 15 or so I started programming and found that I was pretty good at it. So that's why I went into that area and my undergraduate degree within computer science. And I did a I research for several years thereafter. But during that time I met and married a biologist. And the result was not only accidentally learned a lot of biology, I also eventually began to realize that neither she nor any of the other biologists I was meeting were actually interested in aging, which struck me as completely crazy and I had something that had never occurred to me until that point that biologists would not view aging as by some distance, the world's most important problem.

But it turned out that that was the case and then digging a bit, I found that, you know, the few biologists who were working on the biology of aging were in my view, not going about it very well. So I thought well I better switch fields really and I happened to be in a position where I could do that without much risk.

How Aubrey de Grey defines aging

So here I am, – [r] That's great. Well, I I there's one question we always ask all our all our speakers here just to sort of set the stage in addition to the background and how you got here. But it's it's kind of how do you view how do you view aging? What is longevity?

Why do why do we age? And it's interesting because our speakers all seem to look at this from a slightly different angle. – [a] Yeah, it's astonishing really, that people, even people who are experts in the field have a wide range of answers to that question. You would think that, you know, since we've been preoccupied by it since the beginning of civilization, that there would have been a consensus reached, really, but there you go.

Um So, yeah, I mean, my view of aging, my definition of aging is perhaps a little distinctive simply because of the way in which I arrived in the field, starting out in an engineering discipline, a technological discipline.

So I look at aging, you know, from that perspective, really looking at the body as a machine. Obviously, a very, very, very complicated machine, but still a machine.

And therefore I look at aging in a very down to earth way the way I would look at aging of a car or whatever. Just that it is the lifelong accumulation of self inflicted damage that they created as a consequence of the machines normal operation, and that the machine is set up to tolerate.

It only a certain amount of that damage, therefore eventually that threshold is exceeded, and the machines function starts to decline it eventually to cease entirely.

That's to my mind, a definition of aging that works just as well for living machines and living machines. And then of course there's the question of why it's valuable to do something about it.

And there we have a rather different situation where the analogy with cars and airplanes or whatever doesn't apply at all, because we're perfectly happy with the idea of a car basically falling apart and we just buy a new one, but we don't have that option for our bodies. Um so um of course the question then, is is there some kind of um you know, satisfactory limit to how much how long we would um extend the period before the machine starts to decline in function.

And to my mind it's perfectly obvious that there is no such limit because essentially uh attitude to all of that question revolves around how we want our future to be, which has nothing whatsoever to do with how long ago we were born.

– [r] Yeah, yeah, great, great, great thinking on that. I I one thing I want to share with you the first time I came across your work was I think back in 2006, up in Monterey at a ted Technology Education to design conference and I heard you give a presentation there, that was just just remarkable and I think that It's still available online on YouTube if you want to search it. But uh it was it was very transformative, just hearing, hearing you talk about that in your vision at that time for longevity and negligible senescence, which we'll talk about. I'm curious.

That was that was back in 2006 I think. Um And I'm I'm curious since then, now we're in 2000, 22, 23. What has changed most or what's happened in the intervening time in the longevity space?

Progress in longevity research and funding

That that's most that's most excited you since that talk. – [a] Yeah, take me back. I was I was very privileged to be able to speak at such a prestigious conference um at a relatively early time during my career in aging. Um you know, not very many people knew me until then, but as you say, it was well received talk, it's been viewed I think a few million times and so on.

Um of course, at the conference itself, I met various people who were um who became important supporters and uh and so things have definitely progressed quite rapidly since then, I guess it's not as rapidly as we would like. Of course it never could be as rapidly as we would like. But um yeah, I mean that was when I had just created the Methuselah Foundation along with my co founder, Dave Global. Um and a few years later, of course I created Sense Research Foundation and um you know, things have gone pretty well since then um at many levels. First of all, of course in lab, not only our own lab, but labs all over the world, there has been, you know, steady, well at least, um, cumulative progress towards the goal of bringing aging under comprehensive medical control. Um, and I guess really the most important things for me that have happened that really kind of see changes to the situation has been the arrival of really serious money from various sources.

The first phase of that really started about six or so years ago when a few courageous early stage investors started to get involved. Um, up until that time, essentially all of the important work was being funded purely philanthropic lee and well at the end of the day, investors write bigger checks than donors do. So when people start thinking that they might be able to make money out of this in the fullness of time, then things start to go faster. And so, you know, it's a research foundation. We transitioned very rapidly to a business model where we would essentially spin projects out of startup companies as soon as there were investors who had an interest in supporting them and that, that's happened maybe seven times now, something like that.

Um, of course, that's just the tip of the iceberg because alongside us, there have been literally hundreds of other

companies that have sprung up doing important work in the areas of longevity and rejuvenation. Um, uh, being funded from those kinds of sources and of course it's not just angel investors these days, you know, proper institutional investors with very deep pockets have started to come in.

Um, and then the second thing, which happened much more recently really, over the past two years was the arrival of serious money from the Cryptocurrency and Blockchain community.

The reason that has mattered so much is basically because it's a community populated almost entirely by geeks, by people who think like me about aging, you know, who are basically technologists and who very readily understand the body as a machine kind of thing. And the, you know, the potential for comprehensive periodic preventative maintenance to do the job we want of stopping people from going downhill as they get old.

Um, and so yeah, I have been extremely successful over the past, I'm gonna say five years or so, in enthusing the Cryptocurrency community. You know, I would constantly be invited to crypto conferences where I was the only non crypto speaker just because of the fan base.

And and over the past couple of years. And I say that enthusiasm has very much translated into the writing of text. – [r]Just a quick follow up question on the Cryptocurrency space where Tim Peterson is speaking on our program from VidaDAO What other is it is in the Cryptocurrency space? Is it mainly the decentralized autonomous organizations that are getting traction? Or is it just people with Cryptocurrency investments and a lot of liquid capital that you're seeing coming in, There are really three major things. So the DAos are very important to doubt is certainly playing a big role. And you know, thank you for mentioning them because they just gave a grant of a quarter of $1 million, a sense research foundation just a couple of months ago.

So yeah, they're very cool people and very much a fan of that. Um, then there's the, let's call it the grassroots individual donation community. And the biggest example of their involvement was the Airdrop that happened last year at sens Research Foundation. So one of the real big hitters in the crypto community, Richard Hart, wonderful man who, uh, made a great deal of money and indeed his name creating the Cryptocurrency Hex. Um, he's very much a larger than life kind of character and has a large following of devotees. Um, and Rather than, you know, he's been a fan of what we do for a long, long time.

He actually volunteered at one of my conferences when he was penurious back 15 years ago. But he decided that he would not just write me a big check the way he could have done, but rather he would mobilize his flock.

And so basically last year we were the beneficial the area of 27 a half million dollars coming from more than 2000 donors, Almost all of whom were what they call hexagons, people who have done well out of hex. Um and almost all of whom were completely new donors who had never come across this before. So it was a very big deal and I have nothing but praise for richard Hart and for what he's done in this area.

And then the third group within Crypto is the big hitters themselves, the people who have made literally, you know, hundreds of millions or billions of dollars out of hacks. The first person out of that community who got properly involved in terms of supporting our work was vitally butter in the creator of ethereum. He read my book when he was 14 and was hooked ever since and once he got into the position of being able to help, he started doing so that was maybe five years ago.

But then in the past two years the number of other people have come in and done the same thing. A guy who's not very well known but outside of the crypto community but is very well known and respected within it.

A guy named James Fickle who made a lot of money out of ethereum um started to get involved and put proper money into this.

The SENS damage-repair approach

But also he um he's the kind of guy who likes to um talking to evangelize and so he and he has a number of friends who are even wealthier than he is and a lot of them have been putting money into the longevity and rejuvenation

field over the past couple of years as well. And those investors as well, like the guy who started coin based brian Armstrong created a company funded it to the tune of $100 million. Um, you know, lots of, lots of crypto people have been making big waves in this area, Charles. Hoskin son who created cardin o has just created a clinic in rural Wyoming. Um, you know, – [s] it's uh, it's great that the field is getting so much financial support.

Of course, that's one of the foundations of growing the field. Uh, let's take a step back. Can you talk a little bit about the stands approach to combating aging that I think you helped to develop.

Sure, absolutely. Of course, the reason I wanted to highlight the money at first was because until now it has been an inescapable fact that the lack of funding in the field has been the rate limiter in terms of the rate of progress and now argue that it no longer is. But yes, so the sense approach is very simple really. I mean, as I was explaining earlier, it really just about looking at the body as a machine and therefore looking at the ways in which we will extend our healthy lifespan and therefore, as a side effect of a total lifespan, um, in the same way that we would do so for a vintage car or whatever.

And that comes down to comprehensive, periodic preventative maintenance. That's all it is removing the various types of molecular and cellular damage that the body does to itself throughout life. Um, every so often so that the amount of damage the body is carrying around does not rise to the threshold amount that the body is not set up to tolerate with full function.

And this sounds so obvious that these days that it's astonishing really to think back at how counterintuitive it was 20 years ago when I first started talking this way, the people who were working on the biology of aging were completely focused on the idea of instead simply making the body run more cleanly. In other words, damage itself more slowly than it would naturally do.

And of course that's the kind of thing that we get from, for example, calorie restriction or from for that matter, calorie restriction, genetics and so on. Very, very few people were thinking in terms of removing damage that's already been laid down, even though obviously it's much more desirable because it means that it's actually useful to people who are already in middle age or older.

Um So yeah, that's what the sense approaches and the way it ended up being a plausible approach because of course, I had to say, not only is this what we'd like to do, it's also going to be easier than cleaning, making the body run more cleanly.

Um I had to break it down into describing what the damage is that we need to periodically repair. And um the way I did that was basically to describe seven categories of damage within each of which there are plenty of examples that differ in various ways, but nevertheless the classification has the great utility.

There are generic approaches to actually doing the damage repair that apply across the whole category with differences of detail from one place to another.

So for example, one category is selloffs cells dying and not being automatically replaced by the division and differentiation of other cells. And so of course if that happens progressively, the number of cells in the relevant in the affected tissue declines and eventually there are not enough for that tissue to do its job.

And so I often use Parkinson's disease as the most straightforward example of that. This is a case where of course we have a particular type of neuron that dies much more rapidly than most neurons do. Such that by um old age all of us have lost maybe a quarter of the dopamine neurons that we had in youth and some people have lost maybe three quarters and that is beyond what can be tolerated and that's why they have Parkinson's disease. So of course the damage repair approach is simply to use stem cells to replace these new neurons and restore the number of them so that we restore the amount of dopamine there is and of course um it's been found that when you do it right, it works. And now we're up to the point where there are clinical trials going on doing exactly that took a long time to get here because we needed to figure out things like I. P. S. Things like how to manipulate stem cells in the laboratory before injecting them. But here we they are now.

So that's just one category. And of course there are other areas where cells the last let's take for example the thymus where of course we have a dramatic reduction in the production of naive T cells as a result of the thymus essentially becoming a chunk of fat.

And um again you know there are approaches to doing something about that whether with stem cells themselves or whether with growth factors to uh take the residual cells in the thymus and regrow them etcetera. So that's just one category.

And of course as I said there are different details. You use different stem cells for different issues but still it's very useful from an engineering perspective to have this classification because of course once you've got one stem cell therapy working for one tissue you can reuse a lot of the knowledge that you acquired in getting that one working. So the next one and the one after that much easier and quicker to actually develop. – [r] Before before we get into the details on these.

– [r] And this is fascinating. I wanted to back up one step and something you mentioned just to sort of underscored a little bit, the idea of damage repair versus uh slowing damage creation and and some of our speakers, they're focusing on slowing damage creation. Others are on on damage repair.

And what is it about slowing damage? I mean, uh, damaging repairing damage that is um Much more an appealing target or much more effective than just slowing repair.

I mean, I just intuitively, I think I'm gonna I'm gonna break my arm.

Stress, aging, and cellular damage

It's better not to play on the swing than have a better way of of making the bone heal, but that's that's a naive approach. Maybe you could expand on that little.

– [a] Yeah, yeah, you're saying it exactly right. This is precisely what was the mindset within the community 20 years ago that led them to focus exclusively on slowing down the creation of damage. The fundamental error in it is that once damage has been created, but in the stage before there is so much damage that we get sick.

The damage is kind of inert, it's it's not participating in metabolism, so to speak. It's just sitting there, you know, accumulating. Whereas metabolism itself is of course this enormously messy um and complex network of processes and here I use metabolism in the broadest sense, you know, the entire network of protesters that keeps alive.

So I'm not just talking about, you know, um insulin metabolism for example, or whatever. So yeah, I mean, it's just so complicated the problem is that the creation of damage is inextricably intertwined with the performing of the things that we need the body to do to keep it alive.

So if you try to, you know, disentangle them and try to get the body to not do the thing, we don't want to do, creating damage. You are just going to have unintended consequences that stop it from doing things we needed to do as well.

Whereas if you are attacking and removing damage after it's been created, then you're basically leaving metabolism to do what it has evolved to do and you're not messing with it. – [s] Let me let me get back on that question because as a psychologist, um one of the interesting things that I address is uh the impact of stress for example, as well as emotions, um trauma and things like this Hans cellular, the person who originated the concept of stress in the behavioral field.

One of his earliest studies actually, you mentioned the thymus, one of his earliest studies showed the atrophy of the thymus with stress. And we we could look at other impacts, impairment of the prefrontal cortex and hippocampus where neurogenesis uh takes place.

So how do you, how does that fit into how you see the process of longevity? – [a] Yeah, it definitely fits a lot, but it doesn't fit in the sense of defining what types of damage accumulate where it fits, is in defining how rapidly they accumulate.

So absolutely, we understand as you're pointing out that stress is bad for aging in general essentially accelerates the accumulation of various types of damage, whether it's soul loss or cells or pretty much anything.

Um and we of course know some parts of how it does, so, you know, the impacts of stress hormones, etcetera. And of course empirically we see that if we look at, for example, centenarians, there's very little that centenarians have in common.

Mitochondria, free radicals, and repair strategies

But the one thing that people who study centenarians very often point to that they pretty much do all have in common is nothing bothers them. They haven't really ever necessarily had a stress free life, but when they get into stressful situations they ride the wave, so to speak, they roll with it and you know, that that that that's that's to me a very important observation. Yeah, So I feel the value of minimizing stress in whatever way, whether it's by yoga or meditation or whatever works for you, right? Um is definitely, you know, un arguable, but it doesn't really change the question of whether damage repair is the right way to go.

It certainly still is the right way to go whatever. – [r] So the stress reduction would help both in slowing damage creation, but also in improving damage repair, I guess is what you're saying, then potentially, – [a] I don't know, I mean certainly in slow damage creation, but damage repair is kind of an exogenous thing, it's medicine, right? We we have to actually inject stuff.

So I don't really see it as having all that much connection there except insofar as of course you could do the damage repair less thoroughly or perhaps less frequently if you wouldn't necessarily have different type of damage repair.

– [r] I see. And so the sins approach and, and I'm not sure if we define sins as engineering negligible senescence and all its important concept and that I think you coin, but it's a, it's a beautiful idea.

So in the sense approach, um, you've, you've been successful at, at basically developing these techniques and planning for ways to make, to repair damage. What are the challenges now that, that you're facing? What are the biggest challenges with the, with the sense approach? And and and and as a follow up to that, the sense approach then it sounds like is mainly doing stem cells and tissue tissue repair that way. Is that correct?

– [a] Yeah, not quite so. Well, first of all, by virtue of being a divide and conquer approach where we are developing different techniques to, to um, to repair different types of damage. We are inevitably in a situation where some of these approaches are easier to develop than others and foundation, we've always been very strong on focusing on the things that other people are neglecting and my new foundation that's going to be the case as well.

The point there of course, is that, uh, once something is already kind of far enough along that investors and big, big farmer or whatever uh are up for it. You know, it gets into clinical trials and so on then kind of, you know, um it's got a life of its own and kind of not something that I feel I need to um focus on. And we are pretty much there for some stem cell therapies and also of course for some analytics, drugs that um, that selectively kill offs in essence cells, which is one of the other strands of sense.

But there are other things that are not quite that far along. One thing that I've been focusing on for a very long time is the removal of waste products.

A different categories within sands um distinguished by whether the waste product is inside the cell or outside in the spaces between cells essentially because the approach that I have preferred and promoted for stuff that's outside the cell essentially to get inside using vaccination and stuff like that where because inside the cell we have much more high powered cata bolic machinery in the license especially whereas inside the cell things only

accumulate because they are by definition resistant to degradation by even the lighter side.

And so everyone has to do things like introducing enzymes from other species or other techniques of that sort. So there um, you know, we're not quite that far along. We're close though, there are a couple of the approaches that we've pioneered that are probably going to be in clinical trials within a year. So you know, that's edge out of my area of focus if you like. And then there are other things that are really hard. So for example, one thing that happens in aging is the accumulation of mutations in the mitochondrial D.

N. A. Which happens very much faster than the accumulation of mutations in the nuclear D. N. A. And the approach that I've been pursuing for the past 15 years or more is to make copies of the mitochondrial DNA and stick them in the nucleus modified.

Of course, in such a way that they still work even though the DNA is in the wrong place. In other words, the protein has to be re imported back into the mitochondria the way that well over 1000 proteins naturally are.

And you've only got 13 proteins that we need to do this for. But still it's a very hard job and we're far closer to making it all work than anyone 10 years ago would have believed that we would ever be able to get to.

But we still got a long way to go quick quick follow up question. – [r] That was all fascinating. But it just one I wanted to ask you about is what is it about mitochondrial DNA that makes it mutated at a higher rate than nuclear D.

N. A. – [a] There's quite a few reasons. The first reason is it's a really bad place for DNA to be because it's right next to the respiratory chain which is where most of the free radicals that the cell creates are generated. But there's also more to it than that.

There's the fact that there is simply less sophisticated DNA repair machinery in the mitochondria and indeed less sophisticated DNA protection, there are stones for example.

So there's a whole bunch of things one might ask, you know, why did evolution not care about mitochondrial D. N. A. And you know, it's a good question.

But you know, even though mitochondrial mutations accumulate much more rapidly than in the nucleus nevertheless um they're still you know, they're still relatively low in abundance um even in old age and the reasons for believing that they are important as contributors to age related ill health are still relatively in direction circumstantial. Um They they're pretty strong but they're indirect and the mechanism is still still not clear.

There are various proposals that I and others have put forward for how a small amount of mutations can have kind of can be amplified in terms of its impact.

Rather in the same way that a small proportion of senescent cells have, you know, pro inflammatory this thing called the saSP as they call it. But yeah it's still it's still rather unclear – [s] you briefly mentioned free radicals and I know that's an important concept. Can you explain for the audience your perspective on our place into the aging process and if you're doing anything to address that? – [a] Yeah, sure.

So um um of course, yes, the free radical theory of aging was the very first properly mechanistic theory uh molecular level of how damage is created in the body.

Timeline for healthy life extension

Um The respiratory chain which is the source of most of a T. P. The main in which the cell extracts energy from nutrients um is a really hairy thing that's only evolved once in the whole history of life and it gets things wrong. Sometimes the intended function of the respiratory changes to take electrons from well mostly from the Krebs cycle and to transfer them to oxygen And in the process create carbon dioxide, which releases energy.

And that energy is used to regenerate a teepee from ATP and phosphate, which is all very well. And it was all worked out in 1961 by Peter Mitchell. But um the problem is that occasionally the electrons that are transferred out of the T. C. A cycle do not find their way to oxygen in the correct manner. They find a way in an incorrect manner creating something called super oxide which is a free radical a molecule with what's called an unpaid electron. And after that all hell breaks loose because the process of uh well because because free radicals are highly reactive and they and a lot of things can happen and we have a lot of machinery, both emblematic and in terms of vitamins that minimizes the extent to which free radicals cause havoc, but they certainly do not minimize it to zero.

So this is a major source of the long term accumulating damage not only to mitochondrial DNA, but also to lipids and proteins that that eventually leads to age related ill health and in terms of what to do about it.

Well of course um this again comes back to the question of um slowing down the creation of damage versus preparing it. The approach that Dan Harmon, the creator of the free radical theory of aging first um proposed was simply antioxidants molecules which react just like anything else with free radicals.

But the reaction product which is itself a free radical is unusual, Ian reactive and therefore it kind of detoxified the whole problem. Unfortunately antioxidants basically don't work or hardly work because it seems that the free radical reactions that matter happened so fast that essentially and in in locations that antioxidants do not tend to be able to have much impact on.

Um but conversely if one is going in one step later, one is actually, you know, accepting that damage is going to happen as a result of free radicals, but then one repairs that damage, then one kind of, you know, gets around that problem. – [s] What are some of the ways that that can be repaired? – [a] Well, So all of the things I was just mentioning. So for example, waste products inside cells are very often accumulating in the license own because the molecules in question are so cross linked and messed up that they are simply no longer substrates for any of the dozens and dozens of hydrologic enzymes that the license contains many of the in fact most of the chemical reactions that creates this Gordian knot that can't do anything about um free radical best reactions.

So that's just one example. – [r] I've got a follow up question on that. How soon do you think people will that will see significant healthy life extension effects from the work you're doing or just generally in this space when when are we gonna see a bump things?

It's a great question and I wish I knew the answer but um I think we could be talking only a decade or two away now because the damage repair approach being a divide and conquer approach is clearly going to need to be translated to the clinic as a panel of therapies that are provided to the same people at the same time so as to fix a whole bunch of different types of damage simultaneously.

But we're getting there with these things. And what's most important to remember is that there is crosstalk between the accumulation of different types of damage in the sense that um you know, the more of one type of damage you have, the more inefficient the processes we have, that that limit the rate of accumulation of all types of damage.

So, for example, a decade ago when people first started exploring in mice in genetic models, initially the impact of removing senescent cells, they found too, I think everybody's astonishment certainly mine that there were very widespread benefits to the health of the mice that were given this intervention and you know, so widespread that you couldn't even really hypothesize what the mechanism was for.

Why moving themselves would have these knock on effects. But the knock on effects were very broad, which of course means that we could end up being able to get away with a really rather incomplete portfolio of intervention of damage repair interventions and still get a significant benefit in terms of prolonging healthy lifespan.

So I don't know, but I'm quietly optimistic that we are we are getting there pretty fast.

Mouse rejuvenation and future research priorities

Should actually mentioned in this context that one of the major new activities, in fact, really probably the

centerpiece of my new foundation is going to be to push forward exactly this to do combination therapies. We will only work in mice, not humans because humans are expensive, but we are going to be taking middle aged, nice and doing combination therapy on them. Having had done nothing to them at all until they got to maybe within one year of their life expectancy and the goal will be to reach the milestone that I have historically called robust mouse rejuvenation which basically means doubling their remaining life span.

– [r] Yeah that's a fascinating idea. We we've talked about the interventions testing program and the work on mice there. So your your project will sort of take this to the next level and because they're obviously limited in the number that they can do but how will your project compare with the existing work done on mice and longevity?

– [a] Yeah. Thank you for asking because absolutely this will be another case where we definitely do not just want to duplicate effort. So the main difference is that in the I. T. P. And indeed in other efforts along of that nature such as work that's starting up now at the buck Institute also work in Singapore and Brian Kennedy. Um those those are all basically looking at dietary interventions that orally available stuff and um you know that's great but we firmly believe that that's not going to cut it that we need to go into stuff that needs to be injected in order to really have the maximum effect.

And the reason why that has not been done in these other programs it's basically cost and efficiency. They basically say well if you're going to be doing something every day it's got to be something you can do really easy easily.

But of course the interventions that we want to do do not have to be done every day. Maybe they only have to be done once or maybe only a few times during the lifespan during, during the treatment period. So we believe that is a misguided, honestly limitation of these other programs and we will be focusing very strongly on things that are explicitly damage repair and therefore only need to be done occasionally or maybe even only once. But we are absolutely not shying away from doing stuff that needs to be injected.

– [r] Yeah, that's so exciting. I can't wait to hear the results of the of those projects here. You're involved in so many things in the longevity space. I'm wondering are there any areas of longevity that are very exciting to you that you haven't been there able to get involved in or what's the most interesting things that you're not working on in longevity?

– [a] Well, I mean, we of course, you know, we're a small organization. I mean both my organizations and Research Foundation and the new one small. So we can't do everything. But equally, you know, we are constantly looking to update our priorities.

I have one person working for me, Katie Lewis, who is whose main job is to answer that question, you know, ongoing li you know, to identify and consider new priorities that we may have. – [r] Great. Yeah. – [s] So if for our audience, can you identify a couple of very practical things that are available right now that the audience can follow that would maybe have an impact on their longevity.

Practical longevity advice and closing remarks

– [a] Well, of course, you know, I'm a PhD, not an MD. Right? So my area of expertise is very much what doesn't exist yet rather than what does. And you know, I think Robert is very capably demonstrated, not only not least in his new book, but you know, you should really listen to him more than to me on that matter.

But I really think that the fundamental, the only fundamental thing that can be said is to pay attention to your body. In other words, everybody's metabolism is different and you know, everybody has their own things that they have to pay attention to. I'm one of those repulsively lucky people who can eat and drink exactly what I like and nothing happens.

I don't even need to do any exercise, but and I'm biologically far younger than I really am. Um, but of course, most people are not like that. And so paying attention to what your body wants you to do is really the fundamental thing.

– [r] One quick follow up question to that Aubrey as a, as an expert in this space, would you be comfortable sharing anything that you do personally with, you said nutrition's find and everything. Are there any supplements you take or any sleep habits or he said, you don't exercise anything at all that you do.

– [a] You share this comes back to. I just mentioned that my biological age is very nice. I've been, I've been fortunate to be able to get really high end test of that every few years for the past 20 years and I always come out ridiculously good. So of course that means that for me the right, the rational thing to do is to be conservative if it ain't broke, don't fix it kind of thing. Even things that one might generally believe to be good for most people might not be good for me.

So I'm obviously, I always pay close attention and I'm looking out for any early signs of anything going wrong in my body. But until such signs start to emerge, I'm taking a conservative approach and just living the way I've lived since I was 20, – [r] How can people reach you on social media? What's the best way we're going to put your website and everything in the show notes? But anyone who's listening to this on audio as well, what's the best way to reach you and follow you?

– [a] Yeah, I'm easy to find on twitter LinkedIn facebook. I would give you my email address, but it may be about to change because I mentioned starting a new foundation. But so yeah, social media is the right way to find me. And it's very easy, you know, there's only one will be degrade. – [r] And has the new foundation. Do you have a name for that yet?

Or will that be that will be forthcoming – [a] eventually, – [r] that will be forthcoming quite soon. Well, great, it was, I want to thank you so much, Aubrey and Steve for, for spending an hour with us today, uh, and sharing the great ideas you're doing.

I'm a big fan of your work, Aubrey, and it was wonderful to have you on the program here. – [a] Well, likewise, thank you so much for having me. – [s] Thank you very much.