Introduction and guest background

Welcome to this episode of the Reverse Alzheimer's Summit. I am so thrilled to have Dr. Michael Fussell here today to tell us about his unique approach and unique perspectives on Alzheimer's and how we can find solutions to this awful, awful, dreadful disease.

Dr. Fosso has a impressive background, including both an M. D., Ph. D. from Stanford, and he is also in charge over at Telus. So Dr. Fosso, welcome. Let's, let's dove into the nitty gritty of what you're up to.

I look forward to it. Thanks, Heather. And I look forward to hearing what your listeners are interested in, too, if you're passing along. Yeah, wonderful.

So maybe just start with the premise of tell us why you guys are doing things in a really different way from what most of the conventional pharmaceutical approaches are to finding solutions to Alzheimer's and testing as well.

Yeah, let me give you an example. Let's say that I didn't know what caused COVID. All I knew was you had a cough and you had a headache and had a fever.

It didn't feel very well. But I'd never heard of a virus, didn't know anything about microbes or anything like it. And what I did was I said that I think I can cure it with a cough medicine.

No, I'm glad to help with a cough, but it didn't get rid of the COVID. And I think that's what we do with so many age related diseases and among them, Alzheimer's.

We treat the symptoms, the signs, the down downstream outcomes. So for example, we go after beta amyloid or how tangles or mitochondrial dysfunction without asking what do they all have to do again, like COVID, fever, cough, headache, loss of taste and smell.

Nope, nope. You've got to go after the original thing that caused it. I love it. So one of the things that really stood out you wrote is in an editorial for the Journal of Alzheimer's Journal of the Alzheimer's Association.

Well, as Alzheimer's and dementia, what happened was I had and helped organize among a bunch of others. It was a conference on animal models of Alzheimer's and dementia in Washington of years ago.

Why Alzheimer's needs a systems approach

And I summed up the first day by saying, look, the problem is not that we don't have technology. We've got monoclonal antibodies, for example, or that we don't have targets like beta amyloid.

The problem is that we didn't understand what the disease is. So, for example, we know that if you have tools of of APOE before you have a higher risk of Alzheimer's. Why?

We know that if you get hit in the head numerous times, you have a higher risk of many dementias. Why? You know what? If you get exposed to a certain number of infections, for example, in the brains of latest you have, why?

And, you know, we can go on and on with toxins and radiation and lots of different things and fungal infections. A lot of things have been implicated. Why?

And I said what we need is a model. And afterwards, a lot of people came up to say, yes, I agree, we need a model. And one person said, I couldn't agree more.

Do you have a model? I said, yes. I just didn't see any reason to impose that in others. And he said, Tell me about it. So I did. He said, We publish it. I said, Why?

Who are you? He said, Well, I'm the editor in chief of Alzheimer's and Dementia, the world's preeminent Alzheimer's journal. So I'd never had a paper I wrote that got eight reviewers, all complimentary, all of which wanted me to write more.

And then it did very well, got thousands of reprint requests and invitations to chair conferences and interest from our perspective, from investors so that we can hopefully take this to human trials.

So you much like the way we approach dementia here at my clinic at Miramar, the residential care facility. So we're looking for a comprehensive approach, not taking one of those verticals, not just toxins or just infections, but how can we set someone up, set up an individual who's suffering or looking to prevent? Right.

How can we set them up in a way so that every neuron is supported and not constantly in attack and defend mode, but able to put down the infrastructure of creating new neurons, neuroplasticity, new connections, new memories, and reading your your paper was just so refreshing to see that in such a reputable journal, to see this kind of appreciation that we need to shift how we're doing.

There needs to be a full paradigm shift in this as we approach this disease. So it was novel and exciting for me to see this happening and we hope to be doing kind of the boots on the ground pieces of getting that approach to people out there who are suffering right now.

So at Tiller site, so what you did was kind of took this this theme in the world of Alzheimer's and after writing this paper and then turn that into a business to a site that is hoping to create a really different approach to how we do this.

So, like you said, focusing on the causes, not just the symptoms. So how is this happening? What are you doing different? Actually, we started tell us that before I wrote that paper.

In fact, you know, I've never had any interest in writing papers or books just to write papers or books. The question is, where is the benefit of it? And the reason we did this because again, we thought it would help us get going toward human trials.

But, you know, the difference is it's what I think of as a systems approach. I could just as well as is a gestalt approach or any way you want to look at it, but it you know, I have a friend and in fact, she's become our CEO who used to work with Airbus as an aeronautic engineer and worked globally as an executive.

And he said this is a systems approach. And I said, What do you mean? He said, Well, see, I have a family, single family. It fell in a Rolls-Royce jet engine.

He said, I don't ask what's wrong with that one family. They ask, what's wrong with a fan blade in the jet engine in the Airbus 380 going 100 miles an hour at 35,000 feet, a high temperature air.

Emily, it's not the family. It's the context, it's the system. And I think that's what's going on here. You know, it's not a matter of amyloid how tangles mitochondrial dysfunction.

It's not and dozens of other things. It's a matter of how they all work together. It's a systems issue. And once you begin to understand that, that there are all these upstream risk factors and all these downstream outcomes and in between is still aging.

You have to ask yourself, working with intervene optimally. And if you try to intervene upstream, for example, prevent head injuries, excellent idea. Optimize your diet, excellent idea.

Make sure your blood pressure is good. Your, your, your glycemic for sugar. You avoid infections, you avoid target.

All those are true, but none of those have ever been shown to stop, let alone reverse Alzheimer's.

The same is true downstream.

From editorial to company vision

You know, if we if we go in and we try to do something with amyloid and you look at the data, for example, for an academy and McNamara and all the other monoclonal antibodies, you see that at best, you slow it a bit or or offset the downhill course by a few months.

But you don't stop it. You don't reverse it. So the question is, can we do better than that? No. If you look back in 1950, we talked about polio and a lot of people would try to avoid getting it.

Excellent idea. And after you've got it, they try to give good rehab and try to get people back walking. Great idea. But how about a vaccine? How about actually intervening directly at the level of the cause of it?

And that's what we're trying to do. We're going to see if we can take this and the animal data suggest we can do just as you talked about new neurons and the data suggest you actually grow more new neurons.

You have better differentiation and proliferation of neural stem cells. You regrow brain tissue, you normalize, as it were, cognitive function. If I can talk about that in animals, which I think you can and I think we can do this with humans, too, but to do that, we have to be very careful, want to do it right and to to do it credibly.

You know, there have been more than 2200 interventional trials with Alzheimer's by global consensus. Consensus. They've all failed. They haven't stopped and reversed the disease.

And so if we come out and say we've got going to have 12 patients and we can actually show we can reverse a lot of the disease, no one should believe that.

We have to be very careful that we do it credibly, rigorously and correctly. Right. Right. So how do you do it? Well, what we're actually doing is actually literally reversing aging in human cells.

This has been done in human cells in the laboratory now, 24 years ago, the first time in human tissues, 22 years ago, the first time. Some interesting articles out in science and experimental cell research.

But it was first done and and repeatedly after that with human tissues in the lab. But it was first done with animals now about ten years ago. And the best study was probably by Maria Blasco in Madrid, who is the director of See.

And I go there and she used a technique that we can adapt to human trials. It's the same essential approach that was used by the infectious group in showing we can prevent or cure spinal muscular atrophy in children.

So we're going to use the same basic model and we're looking at another model as well. We need to test both of them and take it to human trials to show that we can reset the aging process.

And cells in your brain have them act like young cells again so that they actually turn over beta amyloid and how tangles and the mitochondrial function improves and DNA repair improves and oxidative damage goes down, all of those things. So that's our approach.

What we're going to use theoretically is a gene therapy. We're not altering the genes. We're simply putting in another another copy of a junior we have that functions to reset telomere length, which resets gene expression, which reset cell function which should reset disease.

Wow. So you're the name of your company's Taylor site. And also tell us, how do I. Use me to look. Now? I don't remember the everyone should remember that they pronounce things correctly.

If other people pronounce them differently, they're wrong, right? I think it's one of those words that I've only read. So but but this is. British friends in the UK who talk about telomeres because maybe they call me on the telephone and watch television.

Which is so telomeres. Explain to our audience what they are and what they mean in the context of aging and Alzheimer's. Well, a lot of misconceptions.

You know, the common one is that people are compared to an egg, the little plastic patient and that shoelace. And they say that, you know, if you if you lose that plastic egg, but your shoelaces unravel.

Well, genes, you know, chromosomes don't actually unravel, but they do as the telomere as your cells divide, which they do. And if I do this, I lose some cells.

The remaining ones divide them. And as they divide the telomeres shorten. We've known that since about actually it was first described in 1970 by a Russian friend of mine, Alexei, a little bit, but well, we've known it really scientifically since the nineties and we know that if we reset that we reset the pattern of expression, but as telomeres, shorten gene expression changes and turnover changes.

So for example, if I'm looking at your mitochondria and we notice that your turnover rate of cytochrome c oxidase a standard aerobic enzyme slows down.

So while damage is continually going on, if you slow down the rate of recycling, if you will, you end up with more damage.

How telomere resetting may reverse aging

It's like what happens in my house, you know, if I have a certain amount of dirt that accumulates in my house, if I clean it once a day, it's a very clean house.

I don't know if I clean it once a year. It's a very dirty house. I don't what you see, the longer I take to recycle, the more dirt I get the same thing in your cells and same thing with the enzymes for your mitochondria or the turnover rate for beta amyloid or anything else.

We look at DNA repair, oxidative damage. As you slow down the rate of recycling, the damage begins to accumulate and you have an old dysfunctional cell.

The result is an old dysfunctional mate. So you talk about cell senescence as a model for this. So you can you could describe senescence and the questions that come up as we use that model to to create a comprehensive approach to dementia.

Well, all of your cells, as I say, as they divide, they tend to get older in a cellular sense. They have this clock, the telomere that changes that pattern of gene expression.

And sometimes when people say cell senescence, they mean the entire pattern of change. From the day I was born, till now, I'm 71 now, sometimes what they mean is the replicative senescence, where the cell has gotten as old as it can get, it can't divide anymore.

So when I hear cell senescence, it sort of depends on which thing you're talking about. It's very confusing literature. People say cells, in essence, and you don't know which thing they mean, the whole the whole spectrum or just the very end of it.

But there are other changes to it, as it turns out, for example, telomere length doesn't matter. It's the change in telomere length that matters. So, for example, you'll see mice that have telomeres at birth that are ten times longer than mine.

But I have a lifespan 40 times longer than theirs. It's not the length, it's the rate at which it changes, which changes the pattern of gene expression.

And it really is the gene expression that matters. You know, if I look at the difference between the cells in my ear and the cells in my heart, they have the same same genes, but they're playing a different tune on those genes.

And that's the same as a difference between me at age seven and me in age 71. It's it's not the genes, it's the tune I'm playing. So what we're doing is essentially just resetting the tune.

Wow. So when can people expect to have access to what you guys are creating? Where are we in the process of discovery and then getting to market? Well, you know, what we need to do is finalize the funding for us.

And we've had a couple of groups who are committed to funding us. But until I see the funds in the bank, it takes us about a year to do the large animal study.

So we've got the toxicity data, the dosing data and so on that we need for the FDA. And then it takes us about a year to do that. Phase one human trial.

And after that we're going to do a global trial for phase two everywhere in in the globe. So after that, I think the the proverbial, you know, what hits the fan.

And so I'm certainly curious, how much does it cost to go through that process? Well, I mean, the the cost the patients in the trials will be nothing that we're going to write.

And after that, I can give you some some napkin sort of estimates on what it costs us to produce it, and those will go down. What I can tell you is that right now, the cost of producing what we need to to give a single dose is probably about a third of what it costs you to to pay for your last year of life and Alzheimer's not having a cure.

So in that sense, it sure is cheaper. Thank you. But we'll have to see how far we can get down the cost. Well, you can. Do here's an analogy here. You know, right now, people panic about the cost of Alzheimer's and it will it make us medically bankrupt.

In 1952, in government estimates were that the US would be, quote, medically bankrupt by the year 2000 because of the cost of treating polio, iron lungs, rehab, nursing care.

And in the year 2000, the W. H. O. estimates were that it was less than $0.10 a head to prevent to prevent polio. I think that's where we are now. That is, everybody is worried about the cost of this in the long run.

But as we as we have as we do, this not only generally lower the cost, but our ability to produce it goes down, the costs go down. Our technical abilities to make it better go down.

I think we're going to find that we can actually lower your health care cost dramatically globally, nationally and personally by doing this. Yeah, and I think what I want our listeners to understand is just how expensive it is to get these things to market right, to get them in front of you, that this isn't something that can happen overnight, that it's not something that costs, you know, millions of dollars, but sometimes tens of millions or even more than that.

So how you spend the money to the here's an example, Anna-Kat, from my perspective, doesn't work. Now, if I had Alzheimer's, I probably take it anyway.

But but I don't think it works. It certainly doesn't reverse or stop the process and, you know, it costs an enormous amount. The Biogen actually spent about $1,000,000,000 on the research to come up with that kind of a we can probably spend something, well, -$100 million, probably more like $50 million to prove that it works.

Doing what we're doing. And the question isn't so much how much you spend or how hard you work, how smart you are. The question is, are you applying at the right place?

The classic example is the old Indiana Jones movie, where they're looking for the the Ark of the Covenant and they're digging in the wrong place. Well, when you're digging in the wrong place, it doesn't matter how big an excavator you have or how many people you have out there are shovels.

If you're digging in the wrong place, you're digging in the wrong place. Whereas if you have, you know, five guys with shovel, you're digging the right place.

So I think that's true about the cost of this. It can cost an awful lot if you're digging in the wrong place and get nowhere or you can dig in the right place and relatively speaking, spend very little.

Now, I don't have $50 million, but that sure is a lot less than it cost to do. Most studies like this. Yeah, that that is really impressive and exciting to know that these things are on the horizon.

So a timeline and are you thinking a few years?

Telomeres, senescence, and gene expression

Yes, there is intel this is available for people to get benefit from potentially. Well, it depends on, you know, in chemistry, they talk about rate limiting steps.

Now, if I'm trying to start a fire, the lemming steps are what I'm burning and how much oxygen we have. And having it, you know, there are a couple of things like that in any chemical reaction and economically or in terms of pharmaceuticals that rate limiting steps to the first one is convincing people to fund.

The second one is getting the data you need. Go through the FDA. There is another one that people don't think of, which is it take to get the quality of what we need to do this safely and effectively.

It takes us something on the order of a year or so to produce the quality medicines we need. After that, the time goes down, but there's a lead in time.

So you have all of these sort of rate limiting steps that you really can't control. Good. And, you know, we did a clinical trial here in my office and recruitment took a lot longer than we expected.

And so we of course, we started in February of 2020 and then the world shut down a month later. So that was not expected either. But these these unexpected little things or big things come up as we start to do these trials.

And certainly that can can delay getting these things out there into the world. We've been lucky in a way, because, you know, we haven't even we haven't even asked people if they want to volunteer, but people have been volunteering.

So we have a registered registry of hundreds of people on it. But most companies, for example, Biogen, Eisai and Eli Lilly and others have been looking for patients with early Alzheimer's and a perfect patient has no symptoms but is going to get symptoms, which is sort of hard to estimate, but that's what they're looking for because they don't think you can stop it, but they think you can slow it if you catch it early.

And we're not we're looking for patients with moderate degrees of Alzheimer's because we think we can reverse it. And I can't very well reverse it if you haven't gotten it.

So it it makes it a lot easier for us because we have lots of places. You know, we're talking about doing the first human trial in Kansas City or actually done at USC.

Keck Or at Harvard, because we had people on our board doing this and they run Alzheimer's centers and they do have patients with moderate Alzheimer's that no one wants in that trial.

We do. Thank you very much. And I'm sure many of our listeners here today will either be struggling or know

someone who is. Is there a way that they can sign up to be on that list, to be considered?

Well, they can they can go through our Web site and ask. But again, until we're ready to to move this forward to FDA human trials, we're sort of willing to.

Do the other work of getting the word out, spreading the word that we need a new model and that you have some solutions in mind. We think we do. You know, we're less concerned about having the wrong model than we are about testing it the wrong way.

We want to make very sure we test it right. We don't make a mistake, give the wrong dose, develop the wrong way. No, we have to make sure that it really does work and that it's safe.

And I think it will be both. So, A, you've already done animal models, is that what I understand? Yes. Maria Blasco has done a number of these in Madrid.

So, yes, it's fascinating to watch, see what's goes on. Amazing. And so if if our listeners could help you in some way, what can they do? How can they learn more?

Help you guys get to your goal faster? We are a community who is very committed to finding solutions to Alzheimer's and dementia as soon as possible. And right now it's fun and we have a number of people who put in convertible notes.

But you know what? We need is not not $10,000. What we need is $10 million. And it's finding the right people to put that money in. We've had a lot of people who want to give us small amounts and it there's it's sort of like going over a chasm.

You know, you can't jump in small pieces to jump the whole chasm or you don't if you jump halfway, you're in trouble. And that's very much true of the funding, too.

We can't, you know, $10 doesn't get it, 10,000 doesn't get it, but 10 million gets it because then we can jump the chasm. Wow. Okay, so this is an exciting time for you and your work.

How long have you said this is? It was before you published this paper that you had started. Tell us. Right. So tell me about that journey. What made you go found this company and and what is the process been since then?

I kind of get where you are now. What was the for. Me, it sort of started back in in medical school, actually, in graduate school, too, back in the late seventies and early eighties, I was teaching neurobiology at Stanford and I got intrigued by aging.

And I found that people would always say it kind of it just happens. Your rush to get old, but expect wear and tear entropy. And I thought that's an awfully blasé way of looking at it.

Maybe they're missing something because usually when people are that blasé, it means they haven't really thought about it. Now it just happens. Now things don't just happen. You wonder how they happen.

But I couldn't find a good way to get involved in the research until the mid nineties.

Development timeline, funding, and trial costs

Early nineties, actually, when I ran into some of the work on cell senescence cells, aging, telomeres, summarization, and I got fascinated because it came down to work together.

So I wrote the first paper, a first book on that first papers in JAMA. And 26 years ago we gave the first lecture on this at NIH, but still we needed the funding and we had an offer of remarkable amount of funding in 2001.

But then the couple who offered it had an angry divorce and they didn't. They hated each other. So there was that something went down. So I wrote the first textbook on this for Oxford Press and then got involved in some other biotechs that tried to do something like this.

But the people had problems as often as it's not the bad scientists, the bad people. But then a couple of years back, my friend Peter Raison flew out from the UK to meet me because his mother had Alzheimer's and he said he'd look through the world's literature.

In my textbook for Oxford, Press is the only one that made sense and had a systems approach which is why he gave me that example of the Airbus A380 and the Rolls-Royce jet engine.

So we started putting it together and, you know, the major problem, of course, has been getting people to understand the model because they keep thinking it's something simple, as it were, cough medicine for COVID, nope, doesn't do it.

And then, you know, the other problem, of course, is that the patent issues are a little different in gene therapy than it was in the old style. In the old style, you know, if a patent had a new chemical, I patented the chemical.

But you don't patent the chemical in gene therapy, you patent the recipe. It's like making chocolate chip cookies. The old days, you patent the chips and the butter and the eggs and the sugar and you know, and in the new days, you don't patent the ingredients, you patent the recipe.

And it makes harder. It's harder for investors to understand when you're actually patenting something that can't quite see for a process they don't quite understand.

So it's been an uphill battle. We're getting there. They're getting there. And this complex of some science approach to me is I mean, you're just we're singing the same song.

It it has always struck me that there is this very complex disease and a very complex system. Right. The human body, the human brain. And we are expecting a very simple solution, a one pill or, you know, a couple of IVs might solve this problem when really my expectation is that the complexity of the solution needs to match the complexity of the disease and the complexity of the system. It's manifesting then and it sounds like you're saying a very similar thing.

However, your intervention sounds like it won't be all that complicated to administer. Is that right? Yeah. Here's another way to look at it. It's sort of an ironic way of looking at it.

People tend to think of aging and age related diseases. Is that simple? It just happens. You get old, you fall apart. Entropy where it just happens, it's simple.

And as a result, they can't figure out how to cure age related diseases. Too complicated. And what we're beginning to discover is that the aging process itself is remarkably complicated.

It's a lot harder than just wear and tear, if for one thing, it's a failure maintenance and it gets even more complicated. But because it's so complicated, it turns out the intervention is relatively simple.

So it's kind of ironic, you know, if you think of aging as it just happens, it's very complicated to fix it if you think of it as it's really complicated, but I see how to fix it simply.

Suddenly things turn around. Wow. So and be familiar with David Sinclair's work. And we certainly use some of the Phenols and AIDS and things like that in my clinical practice.

Are you what is your relationship with with his work? Are you familiar with it? Yes, there's a nice guy. I think of it as a nice kid. But he you know, he's still looking at sort of individual solutions here.

So from our perspective, trying to treat the symptoms is coming up with, you know, if I wanted to cure Ebola, I'd want to give you a blood products and I. V.

presser. Agents and fluids and pain medicines and medicines for your fever. What you really need is an Ebola vaccine. And a lot of people are involved in the kind of thing that you're talking about with David, which is he's going after sort of what we regard as downstream outcomes, biomarkers, hallmarks, not what caused it, but outcomes.

I see. So can you any very briefly, can you say what is the cause of Alzheimer's? Old cells. Of the. Cell, aging. Senescent cells, old cells? Well, it also depends on where you start genetically.

So, for example, if I have to apply to illegals, it turns out I make an amyloid molecule that's not very sticky, so it doesn't damage very easily. So even as I get older and I slow my rate of recycling, it doesn't much matter.

So you get people with these two or two illegals or 90 years old and they're fine. If I have to appear before illegals, they tend to make sticky your amyloid so you have a higher rate of damage.

So even at a young age, as I'm beginning to slow down my rate of recycling, I begin to get more damage. So these people are beginning to get micro aggregates of beta amyloid in their twenties and thirties and beginning to demonstrate symptoms in their forties and fifties, which is unheard of rate.

But we two alleles but pretty common for referrals. So genetics makes a difference but it wouldn't make any difference if your cells weren't getting older.

So what we're going to do is reset the aging of the cells. And that being the case, your brain will act more like it did when you were 30. And it doesn't matter whether you're able before if we two.

So the genes make a difference and they cell aging makes a difference. But we can we can readjust cell aging. And we focus a lot on the signals that basically go into the brain.

So like what your what you described earlier, toxicity, nutrient levels, hormones, how much exercise and blood flow just structurally isn't arriving is enough leaving?

Are the waste being taken out and are the infections being treated? And my understanding is that those are the things that kind of trigger that would trigger the amyloid plaques and the proteins.

Basically the inflammation and the oxidative stress, those defense mechanisms. And what you're saying is maybe those things don't matter. Maybe people don't need to change their lifestyle or don't need to worry so much about those triggers.

If we can just reset the aging clock in in the cells of the brain, is that accurate? I understand actually, that is, you know, yeah, all of those things matter right now.

I mean, if I'm trying to right now prevent Alzheimer's disease, there's not a lot I can do. Most of the things are meant to slow the low.

Origins of the research and patent challenges

My risk, lower my risk. So avoid head trauma, for example, you know, and as I say, avoid toxins and avoid radiation damage and lots of things, infections.

But those most of those things feed into the rate at which your cells turnover. So for example, if I get an infection, I'm causing some immune damage and the result is an increasing the rate of of aging of my immune cells.

So I get more inflammation, for example, and a sloppier and slower immune system. But if I can reset that, then it doesn't matter whether you had meningitis at age five or 20 or not at all, because I'm resetting it so that you're no

longer have that problem with the damaged your immune system because now you have a young immune system.

Theoretically, that's the sort of thing we're talking about now. We're also talking about that with regard to micro glial cells and neurons in the brain, it gets inordinate, inordinately complicated, but it's the same process.

We're basically saying, let's make sure you have the risk you had when you were 20, not the risk you have when you're 80. Right. So you mentioned that you're 71. Right.

But what do you do to protect your brain health? You're obviously very sharp. While lot. But I don't know I you know when meditating for 50 years as I know I'm. Well, you tell me does it got me.

Yeah no. Yeah. I'm not sure. All right. I mean, a lot of that turns out to be genetic. Some of it's luck. It's hard to say. You know, my grandfather died at a date when he was 97.

He said he was robbing the cradle, going out with this 89 year old woman and he was smoking a cigar at the time. Some of this genetic some of it is luck.

Some of it is. I'm not sure what. Well, maybe it sounds like mindset. Sounds like he was having a good time. I think so, too. I have to show you this picture.

This is actually I put it there electronically, but this is the HMS Beagle. But 200 years ago, Charles Darwin. So speaking about gene therapy, I put that up for you.

I saw that up there. And also your paper, the unit, a unified model of dimensions and age related neurodegeneration and Alzheimer's and dementia. It starts with a quote from Charles Darwin.

Yeah, I try to remember the quote because I saw it again this morning. But what he basically said is most of the time when people think something can't be done because they're not thinking very carefully, you want to quote it, we have to find it. And he says, But ignorance more frequently begets confidence than it does knowledge.

It is those who know little and not those who know much, who so positively assert that this or that problem will never be solved by science. Very much so. He was right.

You think something can't be done and it usually means that you just don't know it well. Oh, so you basically turn off any effort that goes in towards looking like.

It can't be done. Why should I work on? It's funny that you mention that because Elsevier Publishing has just asked me to edit and author. I'm an author for the chapters on a new textbook on reversing aging and age related diseases.

And I've got people from Mayo Clinic and Harvard and UCSF and the Anderson and Houston Methodist and University of Innsbruck all contributing chapters on this.

What are basic or basic approaches to think that, yes, we can actually that cell aging causes this and that we can do something about it. It's not impossible.

It simply takes a lot of fun. And it's a little frustrating because we've but as you mentioned in the paper as well, we have thrown billions of dollars and countless hours of very intelligent people's time, not only from the US federal government, but also dollars from the from the pharmaceutical industry.

And so much has been thrown probably at the wrong direction, just going down this tunnel of looking at the downstream effects, not at that pleasant level.

So really. I'm not going to make I'm going to make my one snarky remark and it's a bit subtle sometimes. It usually takes people about 3 seconds to get this.

But most of the people involved in the work on Alzheimer's and aging and so on, I know a lot of them globally and they're very bright, they're very hardworking, they're very educated, they're technically adept, they're well-financed, and they are some of the best 20th century minds I know.

Right? Right. It's not the. 20th century think, you. Know, it's the 21st century. And it's time to upgrade your understanding of this awful disease to a complex system, science model and create a solution that works.

And it's relatively simple but matches the complexity of the disease and the brain that it happens in. This is so exciting. And I know I know that our all of our listeners are going to be just excited to get access to what you are offering when you offer it.

So all of us are going to be cheering for you and manifesting that money to show up so that you can do this very, very crucial work in the world. And and again, if there's any way that we can help it sounds like fun mean is the way to do it and then potentially going to your website and reading it.

I think changing the narrative is also something that it sounds like you're up to, right? You are writing in textbooks. You are sharing this perspective with lots of people in the industry and that's part of it. Right. We're kind of stuck in this, right?

Because the story is that there's nothing you can do that it's too hard of a problem to solve. We've spent all this money, we've spent all this time, and 99.6% of these trials failed.

So changing that narrative to make know we just need a new model, we need to start going down that path is a big piece of. You know, if I look back historically and I say, what are the most important things we've ever done medically to make people's better?

It is not robot surgery, and it's not the latest statin drug and it's not a million other things that tend to increase. The cost of medical care was sort of a marginal improvement in quality of life.

The most important thing we've ever done is not a technical advance. It's been a conceptual advance, and that is microbial theory.

Changing the paradigm for Alzheimer's treatment

For example, the idea that you should wash your hands before you deliver a baby drastically lowered the mortality of infants and mothers, and it costs nothing.

A bar of soap, I mean, for God's sake. And that entire lowered the cost of medical care throughout the world when when the model got instituted and people fought, fought against it tooth and nail, they thought it was a dumb idea.

Not I'm not just talking, you know, washing your hands for deliveries and surgeries, but also the whole idea that we could develop vaccines and antibiotics, that really changed people's lives and made them better.

Again, not just the latest antibiotic, but the fact that you can have an antibiotic and not die, for example, of a little infection. When you go out in the barn.

So that has been an enormous medical revolution and that's the big one. It is. As I say, it's robotic surgery. Fine, but I'm talking about something that really made people's lives better over the last hundred years. Enormously better.

I think we're about to undergo a sort of history's second major medical revolution, which is instead of curing microbial disease, curing age related disease, and it will be at least as big as the first one.

And again, it won't raise the cost of medical care. It will lower it. How long do you hope you live? As long as I you enjoy it, I guess. There was back in 2013, nine years ago, Puma World Trust did a survey in the U. S.

and a year later in Canada, and they asked people, you know, if you could live an extra 20 or 30 years, would you want to do it? And the majority, I think 56%, something like that in the world, in the US said no, because what they were thinking is you're giving me an extra 23 years in the nursing home.

And what we're talking about is not that we're talking about being able to play tennis, go gardening, climb a mountain, wrestle with your grandchildren, go out and cook, make something from scratch.

The sort of thing that people enjoy doing much more than giving you an extra ten years of nursing home, which can't be done anyway. So yeah, you know, if anybody doesn't want to take this and wants to have Alzheimer's and wants to die early, go right ahead.

It's a free country. But no, I think we can make people's lives better and give them the joy that they had when they were 20 or 30 something they do. Yeah, it's fun because we get to see a bit of that doing these lifestyle interventions, see people get to regain their their brain health.

And it it's fun to watch them get back to life and enjoy it and get back to dating in your eighties and thrifting and getting excited about food preparation and time with grandkids remembering grandkids names.

And that one's an important one too. But it is. But it's hard work, right? What we do is very hard work. Changing your lifestyle, changing your diet and doing all of these things that it's not easy.

And what you're offering sounds like a bit of a shortcut. So we will be looking forward to that. Take a lot of work to get it, but I think we can do it.

We just have to make sure we do it right. Most things worth doing and take some work. We do a little bit of thought, a little bit work, and sometimes to make a step forward, you need to step back and reexamine assumptions.

You know, 500 years ago, the Vatican astronomers knew that the sun went around the earth. And you understand this. I see it rise in the east. In the west, I'm not moving.

Clearly, the sun is going around me. Well, reasonable assumption just happened to be wrong, that's all. I think the same thing is true of aging natural diseases.

We think we understand it. No, we don't know. Even scratching the surface, there's a lot could. If we think about it hard. Yeah. And reexamine our assumptions.

Smart minds on it. Like yours, maybe I could set up such a privilege to have you. I know that you were very busy and. And have lots going on between textbooks and businesses and curing Alzheimer's.

Thank you so much for taking some time to chat with us and share all of your wisdom and experience. It's such a. Most of your listeners are very busy too.

I don't think I'm any special in that regard and certainly you have been also, Heather, so thank you for inviting me. It's a pleasure being here talking with.

You as well.