Cognitive Decline Is Now Optional: Here's Why Transcript

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Introduction and Background

Dale, thank you so much for agreeing to be part of this Summit series. Your expertize with Alzheimer's and cognitive decline is just so relevant. So Dale, would you please introduce yourself and explain why you are such an expert on cognitive decline?

Yeah. Thank you, Terry. So I've been interested in cognitive decline. I'm a neurologist and neuroscientist by training and ran a laboratory for 30 years in which we studied the basic research on the mechanisms that drive the neurodegenerative process.

So we've been interested in that for many, many years. I'm a professor at UCLA. Currently, I was the founding president of the Buck Institute and was there for many years.

And so we have then translated our work, and we were the first back in 2014 to report reversal of cognitive decline in patients with Alzheimer's and Alzheimer's.

I love your work and I know you had this international reputation for all of your work with Alzheimer's. And the reason I asked you to come today is that I think Alzheimer's and the cognitive decline with M. S.

are parallel diseases that this year so many of the same problems. And we know that for M. S., cognitive decline is very common. The longer you have your M. S., the greater the risk you are to develop cognitive decline and brain fog.

And it's the leading reason people leave the workforce. So let's talk about the major contributors to Alzheimer's and the cognitive decline that is so common with M. S..

Yeah, that's a great point. You know, I think of these as what we call defamatory diseases because they are both degenerative and inflammatory. And what happens in Alzheimer's we think of as a degenerative disease.

But it certainly has inflammatory components. And as we think, as an autoimmune disease with a lot of inflammation and recurrent inflammation. But of course, as you just indicated, it has a degenerative component.

So in a way, you know, they are related. Certainly, again, with M. S., we think of it more on the adaptive side where you have autoantibodies, you have your adaptive system that's hyper activated.

In Alzheimer's, we tend to think of it more like a COVID 19 where where you have a mismatch between the adaptive system and the and the innate system where the innate system is on.

Shared Mechanisms in Alzheimer's and MS

So, of course, we think of COVID 19 as killing people due to cytokine storm, whereas Alzheimer's is more cytokine drizzle. And I suspect just what you're saying is true, that with M. S., you have the same sort of thing.

So, you know, the big message that we have today is Alzheimer's is now optional. And hopefully the cognitive decline of M. S. is now also optional because we can, for the first time, we can really look at what's driving these things and get at the the upstream drivers of the problem.

And in Alzheimer's, it's for major groups of things. It's anything that causes ongoing inflammation and that's leaky gut or chronic sinusitis or Lyme disease or viral infections, all these things, you know, poor oral microbiome within inflammation, any of those things.

Number two, it's anything that causes ongoing toxicity and that can be organic, inorganic, or it can be bio toxins like micro. Thirdly, anything that reduces energetic cerebral blood flow oxygenation.

So, Neil, things people who have sleep apnea are at increased risk, mitochondrial function and then things like ketones that actually give you the substrates to burn.

And then the fourth and final thing is trophic activity and that's growth factors like NGF and BD and RF. It's hormones, estradiol, progesterone, testosterone, thyroid, all critical.

And then it's nutrients, things like vitamin D. So those are the four big groups and I suspect that they similar things are happening in patients with. Ms.

Absolutely. So in again, Dale, you have such seminal work in Alzheimer's, but everyone who's listening, Dale and I agree that these same concepts are very applicable in mice, and these are the same concepts I use when I'm seeing my patients that I walk through to sort out how we can best support them on their healing journey.

Now you have published a bunch of really interesting papers. When I went to medical school, I was taught that if you have Alzheimer's, if you have cognitive decline, and if you had cognitive decline from M. S.

or Alzheimer's, that there was no coming back. Right. But you've published a number of papers that that called into question. You thoughts about that?

That's a great point. We began publishing after the first example, 2014 and have continued now. And this year, actually, we published a clinical trial in which 84% of the people actually improved their cognition, not just slowed the decline, but actually improved their cognition.

We have people now ten and a half years on the protocol who still have improved cognition from ten and a half years ago. So unprecedented improvements and sustainable improvements.

So that's you know, that's the idea here. And as you said, now we're really being able to take these principles that we discovered in the lab over the years and now apply them to M. S.

and apply them to other things. And by the way, along those lines, we are beginning with other diseases and we have fantastic initial data on wet and dry macular degeneration.

So as you know, there isn't anything for dry macular degeneration right now other than a red two, which has almost no effect. And we've got dark adaptation studies that show dramatic improvements in people with dry macular degeneration.

So we should be able to take these principles and adapt them for Parkinson's. Lewy body, M. S., frontotemporal dementia, cortical basal degeneration. Just go right down the list.

You know, when I think of macular degeneration, that's millions and millions of people. I believe that's the leading cause of blindness right now. And, you know, this is something that I think about a lot.

I have aunts that became blind at an early age and then began having hallucinations and had, you know, cognitive issues as well. And so as I watched their lives unfold, I'm thinking about your work and my work and that this was the root causes in those four big buckets that were driving their blindness and their brain fog and hallucinations.

Are absolutely these same sorts of things. Again, you know, all of these are turning out to have one thing in common. You have a supply for a specific neural subsystem.

And of course, in MSA, you're talking about the white matter, you're talking about your immune status and any sort of pathogens that you're chasing, of course.

EBV has been the big one. There's been so much press on in the last few months, but likely there there may be others as well. So you've got this situation where you've got this balance between the supply and the demand.

And, you know, when you get these diseases, you have a chronic mismatch where the demand exceeds the supply and they're nowhere. Is this more obvious than in macular degeneration, where blood flow is critical.

As you know, if you have atherosclerosis, you're at increased risk.

Reversal of Cognitive Decline

You increase your demand by you, by going to lots of blue light, living near the equator, or decrease your supply by living at altitude, by having sleep apnea.

These all increase your risk for macular degeneration. And you're absolutely right with what you said. There are 11 million Americans with macular degeneration almost twice as many as have Alzheimer's disease.

So this is a very common problem, as you indicated. Yeah. You know, they revised the number of folks with M. S., a million. I think that's still probably an underestimate.

And then if I think about the neuro immune, systemic autoimmune diseases that have a neurologic symptoms or psychiatric symptoms, you know, again, that's probably 10 to 15 million that have a systemic autoimmune disease, that have brain fog, anxiety, depression, irritability.

And then I can think about the folks who had COVID, who are left with brain fog, anxiety, irritability, or having difficulty working because they just can't problem solve as well.

Or they're having difficulty getting along with folks that it's the same type of problem and a cure for them, that they are at much greater risk of developing a new immune problem.

Absolutely. And I think brain fog is becoming a huge issue in this country and elsewhere. And, of course, COVID has brought this to the fore, but in fact, it's such a common problem.

So it's actually, to some extent, a favor that was done for us to brain says, hey, wait a minute, there are a lot of people out there. And as you mentioned, anyone who has neuro immune problems.

So many people just with their the diet that they're doing, the sleep schedule that they're doing, the stress that they're under. So many of these people, and I'm sure you saw this just this week, this new report that people with adult ADHD are at increased risk for cognitive decline, which, by the way, fits with our research perfectly.

I was always wondering over the years, why were people denying that? But it's turned out it's actually true. If you have adult ADHD, you are at increased risk.

So brain fog is becoming a huge issue for so many of us and there's a tremendous amount you can do about it. You know, look at the number of people who are relying on energy drinks to try to get through the day because they're so fatigued or they're relying on a lot of caffeinated beverages to try and maintain their their focus and their attention.

And again, to me, that's just another indication that brain is not function as well, that there's excess inflammation. And so those are the kind of people that I wish would reach out to you or reach out to me because we could help them in a whole lot.

Easier to help early in the disease process. Then after you've been diagnosed with Alzheimer's or diagnosed with your M. S.. Absolutely. Now, there's no millions, probably billions being spent on research for be myelination drugs.

What do you think? Are these drugs going to be any more successful than the drug development for Alzheimer's?

Well, I'm so glad you brought up the drug development for Alzheimer's.

What a mess. So recently we heard that looking a MAB, which doesn't make people better, it doesn't prevent decline. What it did in its trial was it slowed the decline by 27%.

Now, Terry, imagine that Elon Musk said everybody perishes in space, x rockets, they explode, everybody dies. But we have a breakthrough. They die 27% later, you would say.

Really? Really.

Beyond Alzheimer's: Other Conditions

Ilan, is that really a breakthrough? So this is what we've been hearing, that there's this historic breakthrough that slows the decline. So the bottom line is absolutely what you said is correct.

The drugs have not worked well, partly because they're not getting at what's actually causing the problem. And I do think in the long run, we need to have these precision medicine protocols that look at what's actually driving the problem and then add targeted drugs.

That's going to be critical. But doing this as a monotherapy, as a drug, it just makes no sense. So what I'm doing right now is compiling a list of all the things that have had better outcomes than look at a map.

Here's a partial list for you. Number one, extra virgin olive oil by itself. Better outcome than look at a map. Number two, refined olive oil. Number three, Polyphenol alone.

Better outcome. Number four. Well, hang on. Polyphenols. Let's play. What? What is the polyphenol audience? So just polyphenols. Things like, you know, there are and there are dozens and dozens of plant polyphenols that, you know, these are basically multi ring structures that are wonderful and support it, you know, support cognition and support anti inflammation.

And they include things like Quercetin, for example, 5 to 10 things like that. And there are many of these and they're typically plant derived. So these work better than looking mad at which by the way, looking at them has a side effects brain hemorrhage, brain swelling, and in a couple of people so far, death.

So I think we can do better. And of course, the Recode protocol that we use and the protocol that you developed work better than these drugs as well. So I think that, you know, the future is to bring these together, but start by going upstream.

And you mentioned the re myelination idea. Great idea. After you determine what's causing the problem. So start with identifying that. And to me, that is the fundamental of new medicine.

New medicine is asking what caused the problem and then addressing those things. Of course, this is, you know, root cause medicine, functional medicine, however you want to think about it, this is going after what's actually driving the problem.

Once you've got that in hand, then sure. STEM cells and myelination and all these things are great after the fact. But start by asking what's causing the problem and get rid of that.

You know, when I when I talk with people about re myelination, I have to remind them that you can't remind the brain if the microglia are still reactive, still driving the innate and adaptive immune system to attack the threats, whatever the perceived threats are.

And so we in order to do that, that is all about diet and lifestyle, sleep, exercise, stress, relationships. I'm sure you have similar conversations in your Recode protocol that you are talking about the person's diet and lifestyle.

And you know, you brought up a great point, which is microglia. So microglia are the macrophages of the phagocytes of your nervous system. They're sitting in there and they're trying to take up these various things, like these various pathogens and things like that, but also swallowing things like damaged neurons and fragments of myelin and things like that.

So interestingly, two groups reported two completely different ideas. One said all we have to do is ramp up your microglia because then it will sit there and eat away the amyloid and you'll be fine.

The other group said All we have to do is turn off the microglia because they're spewing out these cytokines and if we just turn them off, everything will be fine.

So you can see the conundrum here. The problem is microglia are physiological activators. So as long as they're being activated and inflamed and told, you've got ongoing pathogens, of course they're going to be active and of course they're not going to be taking away the amyloid because the amyloid is there to fight the infections.

It is an appetite, microbial peptide. So this simplistic notion that you either activate them or deactivate them doesn't make sense. You have to get at what's causing them to be activated.

And I don't think there'll be a drug solution that will address that.

Brain Fog, ADHD, and Early Intervention

That's going to be really coming down to what in the environment is causing that increased reactivity. You know, it's so interesting to me that we're all used to now large data sets with, you know, Google knows where you shop and where I shop and where everybody shops and what you're doing and all that.

And yet we're not using these sorts of sophisticated algorithms nearly enough for looking at what's actually driving these various complex chronic problems that we're all suffering from.

And so we need to kind of get into a new world of medicine where we understand what's driving these problems, and we need more Silicon Valley, which is why I work with a software company to develop better algorithms so that we can understand more about what's driving these problems.

So I think what I hear you talking about is having a deeper understanding of my family history, my genetics, all of the environmental factors that I was exposed to growing up as a child, young adult, my current environment.

So a much more robust history then probably a much more robust assessment of biomarkers. You know, I think of the biomarkers that that we we can now investigate metabolites that can look at nearly two so literally many thousands of chemical metabolites that can be correlated with outcomes.

And will that be the future we're looking at by the microbes that live in my mouth and my stool, the metabolites in my urine, my stool perhaps in my blood?

And then we have a much more comprehensive guide on what diet will be best, what supplements may be best, what probiotics may be best. How far is that kind of approach in the future?

I don't think it's too far in the future where in fact we're including it in our upcoming clinical trial. So as I mentioned earlier, we published a clinical trial this year that showed 84% of people actually got better.

We're now expanding this and doing a larger randomized controlled trial. I know you've done a number of clinical trials as well. And so as you know, these take some time.

But the good news is, with this new one, there are all of these new ways that we can now look to get a better handle on what's actually driving the problem.

We have to quit thinking of people as a prescription pad. What do I write on my prescription pad? One little molecule for these complex diseases makes no fundamental sense.

We have to start thinking of people as complex systems.

Why Drug-Only Approaches Fall Short

Something went wrong. Somehow this system got out of whack and it created mess. Or somehow it got out of whack and it created Alzheimer's disease or other things.

And we can now understand what actually went wrong with this complex machine, and we can now begin to fix that network. And interestingly, when you fix a network, you're not using a hammer, you're doing much more like what you do.

You're changing the diet. You're you're telling people what to eat. You're changing their inflammatory parameters. You're changing their hormonal parameters, you're changing their lifestyle parameters.

And then you tweak here and there, as you know, we can, but we can now look at fast motile in the blood, look to see whether there's damage ongoing. We can now look at a beta in the blood to see if there what's ongoing.

If you're likely to be developing Alzheimer's, we can do epigenetic studies to see whether someone has issues with methylation, whether they have what their biological age is, whether they are their brain is aging more rapidly or more slowly.

And we can follow these in real time. We can now look at at electrophysiology, we can look at your p300, which is how quickly your brain is recognizing something new.

We can look at your quantitative EEG so we can get an idea of where did this system go awry? And we can now tweak at the various places. And as you know, you indicated earlier, a lot of the things that you're going to tweak are going to be things that you might not have thought of, like improving your sleep that aren't necessarily a drug that doesn't work and gives you all sorts of side effects.

But Terry, you know, you and I, well, we trained as physicians, we were taught to give drugs for these various problems. And great at certain times, you know, penicillin has saved a lot of lives.

Let's be fair. Yes. But when you any time you throw in a drug, what you're saying is, I believe I know better than nature. And a lot of times that's that's got all sorts of side effects.

And you're now looking at all these drugs. And again, with the ones for Alzheimer's disease, you're talking about brain hemorrhage and brain swelling and just horrible, horrible side effects.

So you really have to use these judiciously and know what the side effects are and know when you're tweaking the system. What's actually wrong with it?

What are you trying to do? What's the outcome you're looking for? Instead of just saying, Ah, this is EMS, I write a prescription for Copolymer know what have you.

You really have what's driving these things? You know, I want to take a moment to remind everyone just the magnitude of the work that you've been doing.

Dale Alzheimer's, I learned, was a one way street that once you're diagnosed, you have progress of decline and to you no longer know who your loved ones are, you no longer know yourself.

You eventually become unable to feed yourself and a terrible, terrible prognosis. But that is no longer inevitable. People with Alzheimer's confirmed Alzheimer's have worked with the team.

They've not all. And for many, just stopping the decline would have been like astounding home run. But what Del's describing is that people are improving, they're thinking that they're cognitive perform, it's their memory, their ability to recognize family, friends, interact has been transformed.

That is staggering. A similar cognitive decline happens with our math that I'm very optimistic that these same kinds of approaches, very similar with addressing the toxins, addressing nutrition, addressing hormones can stop the decline in improve thinking and cognitive performance.

And the earlier, the better. It's so much easier, so much easier. The sooner you come in. Now what what's going to happen in the future for you deal. What's what's immediately coming.

Yeah so what's what's happening right now we're just getting the IRB approval for a randomized controlled trial, really excited to work with six absolutely outstanding physicians on that.

Root-Cause Medicine and Biomarkers

So we have six sites, Miami and Cleveland and Nashville and Sacramento and Oakland and San Francisco. That'll be starting early in the year. We're very excited about that and we'll be able to do the test that I mentioned earlier, which we which were not available before, as well as using the overall protocol to show improvement in these patients.

As I mentioned earlier, also, we're doing something called the ARC Project, which is to now take the the biochemistry and the genetics of Alzheimer's and now change it, adapt it for the unique biochemistry of each of these.

And you mentioned M. S., I think is a great example and doing, you know, what you're doing, doing it in getting people to to be on an optimal cognitive protocol from the beginning, I think is is likely to give you best outcomes is when we see people who either get on prevention or earliest reversal, people who have subjected cognitive impairment, they all do great.

We've not had a single example yet. Out of thousands where people got on prevention but ended up developing dementia, dementia is a late stage of this problem.

So get on early and and do well. Now with the ARC project, we're starting with macular degeneration. As I mentioned, we had the initial data look great.

Then we're going to be looking at Lewy body disease and Parkinson's and cortical basal degeneration and progressive supranuclear paralysis and and frontotemporal and als and all of these things.

And the other thing I'm doing is putting together just single cases of each of these things that have shown clear improvement to show that the approach that we are all now taking is actually the way to go.

So we have these wonderful single cases that really kind of get you started. Alzheimer, AMR's, familial Alzheimer's. That's rare, less than 5% unheard of to keep those people doing well.

We have a great example there. Craig Tanen down in Miami had a wonderful example recently of cortical basal degeneration, a rare degenerative condition.

And that has done very well on the protocol. And then people with Lewy body who've done well and then I mentioned the PCA before, so we have a case of PCA recently.

What is PCI of not real cortical atrophy? And this actually came from Kerry Mills Rutland in New York, who is a wonderful brain health coach and dealing with a patient who has PCA, which is one of the non-domestic presentations of Alzheimer's.

So it's someone who's developing Alzheimer's but who didn't start with memory loss. In this case, it's visual perception loss. And this person is doing very well.

And the cool thing is the MRI. The initial MRI showed parietal lobe at 0.6 percentile because that's where PCA happens. It's parietal and occipital. That's why posterior cortical atrophy described by Professor Frank Benson years ago.

And the interesting thing is the follow up scan showed 22nd percentile, so oh 6 to 20 second, just dramatic and other improvements on the scan. But because parietal is really where this disease lives, we were so excited from a very atrophic parietal lobe to a relatively normal parietal lobe in terms of volume metrics.

So she's continuing to work with that patient and the patient is able to is doing somewhat better. But we need to keep improving the visual perception issues for that patient as well.

So I think this is telling us this is giving us a this is pointing us in the right direction for the future. So and again, anyone who's listening, if you have been told that brain volume loss is irreversible, the cognitive decline is irreversible, I think what you're telling us still is that's old thinking.

That isn't matching your clinical experience in the research experience that you have. Exactly right. And I should say, Teri, when you were first diagnosed with M. S.

Current Trials and Future Directions

, of course, I'm sure people didn't tell you you were going to be doing great and you're going to be doing all these wonderful things and getting better.

You broke the mold. And I think that's what we're all doing as we're getting at what's actually causing the problem. Adele, you may not know this. I'm now jogging.

Oh, my God. So that's that's pretty exciting. I'm very excited about that. Yeah, I congratulate that. Well, you know, this is we're all seeing that if you if we kind of ignore this claim that you can't do it, that there's just nothing that helps and actually look at Will.

But why are we there? What's happened then? We're you know, we're seeing we're all seeing unprecedented results. And were you criticized at first for talking about this stuff?

Oh, not just the first one. Still I'm still getting nasty emails, nasty comments, nasty publications. And it's interesting, none of these none of these complaints says, oh, we evaluated one of your patients and didn't agree.

Or, you know, we looked at your paper and we didn't agree with the data. They all say things like, you know, you didn't you didn't do enough people in your trial.

Well, it was statistically significant results. So apparently we did you know, you didn't include the control group. We weren't. But one of the things is you published it in the wrong journal.

Really. You know, let's let's talk about the data. Do you really care? You know what? I tell my my post-doc that if you have a really new idea, it's very difficult to get it published the first time because it's it's so new.

You have to, you know, struggle through or find a journal, get it published. Then the next paper is a little tiny bit easier to publish than the next one.

A little bit easier. And you if you if you want to do really new innovative stuff, that's the process. It just have to persist, persist, persist. So, Dale, I am so glad that you have persisted, persisted, persisted.

I think you're doing amazing, amazing work. Now, Dale, where do people find you if they want to come check out

your work, if they have a family with dementia or multigenerational, I'm sure that they get tuned in to your work.

How do they find you? So we recommend anyone who's 45 or over. Please have a cognate copy, just like you have a colonoscopy when you're 50. So you can go my cog, NASCAR, FT.com that has some information.

You can look at the books. We published three books, the end of Alzheimer's, the end of Alzheimer's Program, and the first survivors of Alzheimer's. Please take a look at those.

We go through some of the things that can be done. You can look at Dr. Rennison dot com or you can follow on Facebook or Instagram. Dr. Dale Bredeson.

So any of the above, you can see the work that we're doing. And everyone who's listening. Please download Dell's gift that will have tremendous resources for you.

Dell I love you. I love the work that you are doing. Keep up the good work. I can't wait till the next time I see you at one of our conferences. Thank you so much, Terry.

Thank you again for the invitation. Love what you're doing for all these years and congratulations. Enjoy the jog. And great to hear.