Doctor Houstonu2019s Background and Credentials

Doctor Mark Houston graduated Phi Beta Kappa and summa cum laude from Rhodes College with a BA in Chemistry and math. He graduated with highest honors in the Alpha Omega Alpha Honorary Society distinction from Vanderbilt Medical School.

He completed his medical training at the University of California in San Francisco, and then to return to serve as Chief Resident in Medicine at Vanderbilt Medical Center, where he received the Hillman Award of the Best Teacher.

Doctor Houston is the director of the Hypertension Institute and Vascular Biology, medical director of the Division of Human Nutrition, and medical director of clinical research at the Hypertension Institute in Nashville, Tennessee.

He has served as an associate clinical professor of medicine at Vanderbilt University School of Medicine. Doctor Houston has presented over 10,000 lectures nationally and internationally, and published over 250 medical articles, scientific abstracts, and peer reviewed medical journals, books and book chapters.

He has published nine books, including The Hypertension Handbook and his newest book, Controlling High Blood Pressure Through Nutrition, Nutritional Supplements, Lifestyle and Drugs.

Well, it's a great pleasure to have you on the show. Mark, to, talk about your experience and knowledge in the field of hypertension and tie in telomere biology into that.

I know we've had some discussions before at meetings, and I'm always fascinated to get your take on on cardiovascular disease from an integrative, and sort of aging standpoint.

So I just like to get started by asking you how your approach integrates sort of aspects of aging, telomere biology into the management, diagnosis and management of hypertension and risk stratification.

In your in your daily practice.

Integrative View of Hypertension and Vascular Aging

Yeah. Just one of the, the major differences and what I do with integrative management of hypertension and all its sequelae of CVD is we really have started with the basics of why people become hypertensive.

And we factor that into vascular aging and how that is affected by aging in general. So as you know, sorry most recent a man is his oldest is blood vessels.

Right. So if you can get the vasculature healthy then the blood pressure is going to go down. So we have really turned this thing upside down. Most traditional, hypertensive specialist or internist will look at the blood pressure and decide if I get the blood pressure down.

I have solved the problem of cardiovascular disease. And unfortunately, that has turned out not to be the case. And that's been proven in long term clinical trials that it's not just the blood pressure, it's what happens to the blood vessel.

And so how you get there is important. What you use is important, and how the nutrition or the supplement or the drug affects the arterial health is important in reducing stroke artifacts, heart failure and kidney disease.

And when you get to the basics of what it is about the vasculature, that's so important. We're looking at endothelial

dysfunction the glycol calyx and vascular compliance or elasticity.

And then what what is it that affects those three things. What's the what's the basic foundation of cardiovascular disease and hypertension. And it's three things.

And you've heard me preach this long time. There's only three finite responses that the blood vessel has for all the insults that it can encounter. And there's, you know, hundreds and thousands of these, those fat responses or inflammation, oxidative stress and vascular immune dysfunction.

So in in a summary of kind of what we're going to talk about today, we'll be turning the entire prevention, diagnosis and treatment of hypertension kind of upside down, concentrating on vascular biology, vascular health and vascular aging, which in turn is going to improve your overall aging.

And all of this feeds back into all the things that you and I talk about with aging, such as telomeres. Yeah. I mean, I think that's a really important point.

It's there are a lot of studies that were done that show that higher blood pressure is associated with, you know, with increased cardiovascular events, strokes, etc..

But now we have a much better way of, we have better ways of measuring it, I think. And that's when we've learned that, you know, certain drugs will lower blood pressure but probably don't help, like beta blockers potentially don't help necessarily with arterial elasticity.

Might make it a little bit stiffer and, and, you know, the CAF trial showed that, and then you have, so you want to focus on that. The basic pathophysiology, which I think is, is fantastic is almost in some ways a little bit like reticent does this protocol with Alzheimer's disease, you want to fix all the things that can go wrong in Alzheimer's disease, and then the rate of work better rather than, you know, giving a drug that speeds the brain up, but you're really not fixing the problem, which is causing the dysfunction in the first place.

So I'd love to hear more about, you know, how you go about assessing, evaluating somebody that's different for what we do. And in typical internal medicine or, or hypertension specialists.

Right. Attitude. When we talked about the Signal Corps and and then you talked about endothelial dysfunction and you know the glycan calyx, which is, you know, really gotten, to be a very interesting area.

So, yeah, go ahead. Tell us about that. Sure. So that's divided up into two areas of diagnostic therapy testing. Let's talk about, blood and urine tests, which almost everybody can get from any lab.

So the first thing you do is, if you get a hypertensive patient is let's concentrate first of all, on the blood pressure itself and how we're going to manage that.

And then the second piece of the arterial hill so patient walks in is hypertensive. You want to know why they're hypertensive. So there's primary which is usually genetic and they're secondary.

So the first thing you want to do is be sure the patient doesn't have a secondary cause for hypertension. And that's really not that common. Maybe 5 or 10% of the entire hypertensive population.

And most physicians know how to do that. You know, it's 24 hour urine.

How He Evaluates Hypertension in Practice

Cortisol, catecholamines base, many nephrons, making sure they don't have an adrenal tumor or feel chromo that time, and so forth. Well, that part's pretty easy.

The second piece is to document that the patient really has hypertension, but also to find out what their 24 hour

blood pressure monitoring is, because just an office blood pressure doesn't give you adequate information about the patient's risk, about what to do, what kind of treatments involved.

And if you're giving medications, it helps you determine when to give the medication. So we have like 1024 blood pressure monitors in the office that are always busy.

And they monitor not just the average blood pressure, but spikes and blood pressure lability, nocturnal blood pressure. Whether the patient has nocturnal dipping or non dipping surges in the morning.

So all those things are important. And I'll just give you one example of how helpful the APM can be. Let's suppose that you have a patient whose nocturnal blood pressure is pretty high.

Maybe even higher than it is during the day. And they're, they don't dip at night. They don't have a relaxation of the blood vessels of the pressure falls.

Okay. So if you knew that, you would say, well, it would be better for me to give whatever, whether it's medication or something else at night to lower the nocturnal pressure.

However, if you have the reverse, which is the blood pressure actually is very low at night and they're in large column excessive. Dipper, you don't want to do that because you could actually drive the pressure to low and give them a stroke.

So it is crucial to have that information. And I really don't know how you can treat hypertension in 2021 without having a 24 hour blood pressure monitor because of range won't give you the clues to that.

So those things blood, urine, AB that gives you kind of the blood pressure piece. Okay. Now the other piece, which is extremely important, is what's driving the hypertension.

Related to the vascular system. And we have about 8 or 10 tests that we do in the Hypertension Institute that measure everything in arterial compliance, plasticity, augmentation index plus mammography, endothelial function, glycol calyx, just programing everything you can think of.

We measure it and when I get that information, I can pretty well tell you at least two physiologically why the patient has hypertension. So for example, one machine we have is called a pulse wave velocity.

And it's it measures big medium and small artery elasticity or arterial compliance. Almost everybody who has hypertension has very, very still small arteries.

Sometimes they have stiff, larger arteries as well. But if those small arteries are really stiff, what happens is when blood enters those arteries, they don't dilate and they will rupture or clot.

And if that's in the brain, you get a stroke. In the heart you had a heart attack. So you have to loosen up the arteries with nitric oxide boosters like, okay, let's boosters get those things fixed quickly because that doesn't reverse as quickly with even a blood pressure medication.

Right. Because sometimes the medications are good on the blood pressure pretty quick. But it takes longer to get the arterial elasticity better. So we use a lot of compounds in the office to improve, arterial elasticity.

So that's, that's the, large and small arteries. And we have another test that measures endothelial dysfunction. That's the end of that. And then we have another one that measures what's called was migratory, which looks at all the cardiac function.

It looks at cardiac muscle coronary artery stiffness and other parameters. Sympathetic nervous system balance of autonomic dysfunction. And that's another whole area that feeds into the hypertension and the vascular biology as well.

All that supported with, you know, 2D echoes, right. Duplex, ultrasound of the abdomen, renal artery. So all these things will give you a fairly comprehensive view of how is the blood pressure, does it dip?

Do you have nocturnal dipping? And also what's the basis of the vascular pathology present that allows you to treat both nutraceutical with supplements or nutrition and then with drugs at the same time?

Yeah, that's a multi-pronged approach. I mean, yeah, I have a couple of those issues, but not all the better than all the hypertensive, hypertension Institute.

I was just curious about the Glencoe killings. What's the. I mean, obviously, for the endothelial dysfunction, you have things like, you know, A40 and other blood pressure medications, but, what would you use to directly treat the glycol kinetics right now we have a glycol calix booster.

So is it okay to mention names to come. Yeah. This is not CME. Yeah, sure. So, yeah. It's called Arcturus. IL. Yeah, I've heard of that. Yeah. It's made by Calvary Hill and it's, it's very effective to improve like okay, we've actually done several clinical trials with them.

The first one was on hypertension, and we found that the glycol calyx arterial seal actually lowered blood pressure and at the same time improved our arterial elasticity and endothelial dysfunction.

That was without anything else, just purely arterial. So do you, what what kind of a compound is that? It's actually it comes from, seaweed preparation.

It's got a proprietary blend. I'm not sure I know exactly what all is in it, but but it's, it's a it's a group of amino acids and glycoproteins that are, attached together, and they actually go in and repair and replace any damaged glycol calyx, which is the first piece that protects the in the the lining.

So if both of those are damaged, you've got a mess. If you can repair both of them, then you restore, endothelial function very well. And when you say measure the glycol calyx, I see in some instruments, how do they go about doing this?

It was a thickness issue. It is a functional measure. Yeah. Right now it's, it's a, obviously I'd say it's a test. It's evolving. Okay. It's in my opinion, it's testing.

Like, okay, Alex is not quite ready for general clinical use. It's maybe research at this point, but what what you, what you measure with this particular machine that is out there is the micro circulation under the tongue, which is kind of a real logical assessment.

And it's assuming a lot of things that if the rheology in this micro vessels is normal and the red cells aren't sticking, that the glycol calyx. And the other thing, it must be pretty healthy.

But, you know, is that the case for sure or the other factors? And how do you quantitate you know how much damage there is? So I still want to hold off on recommending any glycol testing directly.

You can get indirect testing by using the the machines that we talked about earlier. Well, you know, you know that the glycol calyx is involved. Presuming this just works through the glycol calyx because you got the lowering of the blood pressure, you know, the pulse wave velocity or in the, the augmentation index.

So, I mean, you could follow those.

Glycocalyx, Endothelial Health, and Arterial Testing

So that might be one of the earlier things you do, because I understand that the glycol calyx is one of the earliest things to start to dysfunction. Exactly. Yeah. Do you do that?

I mean, I don't know how many people you see that don't actually have hypertension in your, in your, in your practice. Probably not that many. I don't really know.

But, you know, it's a continuum, right? Arterial aging, hypertension is just accelerated arterial aging, essential hypertension. And so, I mean, I'd be interested for me if but also for, you know, our listeners, you know, to get in, should we be testing?

Is there an optimal level of these, of these, instruments, the outputs from these instruments that we want to keep way before you actually get over 135 over over 90, you know, the target is right now it's actually is 120 or read has gotten into it, which is which is smart at this point.

Yeah. Because I do that in my practice. I want to I don't I want to not only prevent hypertension, I want to keep your arteries at a 20 to 25 year old level, which I have patients who are on the augmentation index.

They're at 20 years old and they're 50, but they are doing all the things right. You know, they're they're running, they're keeping their body fat down or they're, you know, all the information is down.

So that's kind of where I see what you do is sort of it's a continuum. And I try to put that in my practice. So, so you're, you're you're using arterial cell and then obviously you're prescribing exercise, etc..

What's what's the next sort of stepped level on that in terms of so what? You've hit on a very important point. And, and that is the chicken and the egg concept for hypertension, vascular disease.

And what is very clear now I'm pretty well proven is the arterial disease, particularly in a central or genetic hypertension. The endothelial dysfunction in particular the glycol okay.

And the stiff artery loss of compliance actually precedes hypertension by decades. And so what we have found, if we get, say, a 20 year old kid in the office and both of his parents who have hypertension, but he's 120 over 80 and he thinks he's fine, we find distinct abnormalities in most all of the testing I just mentioned, he's already is already getting stiff.

His echo may show left ventricular hypertrophy or diastolic dysfunction. And you know he's already got vascular problems. So the way I think about this is the vascular disease comes first.

The hypertension becomes a marker of a thick artery. And that's what happens, you know, 1015, 20 years later. So the beauty of this and knowing it is you can actually preempt these patients from getting hypertension by aggressively treating them with things that improve their arterial function in health, slows down their vascular aging, and either stop or delay, or if they get it, it's not as severe as it would be otherwise.

Yeah. And that's health care not disease. Yeah. Right. Impacting the health of your arteries. It's unfortunate that, you know, the large societies are not looking at it that way yet.

They're still looking for arbitrary, really arbitrary, but cut offs that don't make that much sense. You know, I was, And the glycol calyx is obviously made of glycans.

And I was talking to Gordon Loucks, who's, you know, the, what, the big glycol biologists. And he has the glycan age test, and he was he was telling me about a study where they looked at glycan age seems to precede by years, the development of metabolic disease, like hypertension, diabetes, and so that goes right along with this dysfunction of the glycol killers.

And this is, you know, just a, the IG glycans, not not the actual like what counts itself. So it's it's really great that we have these tools now and this understanding of this, pathophysiology to intervene at a time when it really makes the most sense.

Yeah. And the, the people that come in and you find this abnormality, they're so delighted. Number one, he found

it. And number two, they realize that maybe I can avoid taking drug therapy by doing all the natural things, nutrition and taking nitric oxide boosters like neo 40 or Glock or Calix boosters like arterial cell.

And those two together, by the way, or very, very powerful to reverse arterial compliance issues. Yeah, I see that. Haven't started using but I'm going to start using arterial cell.

But I see no side effects with neo 40. Really. Are there any potential side effects with Arturo? So, no, I've seen virtually nothing with either of those rarely, the neo 40 people have citric acid problems.

They get a little irritation in their mouth, maybe like a Cartier seal. I've haven't had anybody have any issues with it. So, I know that there is a large, large I think it was 400 direct studies.

I saw, in a read a number of the sort of more seminal ones linking telomere attrition and telomere length with hypertension. And I'm sure that's an area that you're interested in, know about and maybe talk a little bit about that.

And yeah. Yeah, you're exactly right. Telomere attrition. Right. Very short telomeres, correlate with all, all the vascular issues, all of them, whether it's hypertension, stroke, coronary heart disease, aneurysms, all those things are part of the shortening of the telomere, which makes sense if you think about it.

It goes right to the very initial premise that we talked about, which is hypertension really is accelerated vascular aging. And if your arteries are showing aging, they're going to, you know, be associated with short telomeres.

Yeah. The question is, is is it an association or is it potentially causal? Yeah, that's a great question. Yeah. Let me because then would something like to 65 help in the same way that arterial cell does with helping the glycol calyx.

With that help put off either, you know hypertension or cardiovascular disease, coronary heart disease, atherosclerosis. Through the third area, which you mentioned, potentially affecting, immune dysfunction within the, arterial vasculature.

Yeah, I think it's really, a bi directional issue. And I'll, I'll give you the reasons I think that, in other words, short telomeres increase vascular aging and vascular aging.

Shorten your telomeres quicker. So yeah, it's probably like one example. I'm sorry, I'm having trouble with my computer. Give me one second. It's turned my screen off and then trying to fix that.

Well you just haven't figured it out yet. I can't see you until I do this. Okay. You're back. Oh. All right, so here, here's an example. But, you know, as I do that, there's a lot of things that shorten telomeres.

But if you look at the three finite responses that I mentioned earlier, all three of those shortened telomeres, okay. So if you're at a high oxidative stress, or inflammation status, which most hypertensive boys are, their telomeres are going to shorten.

So as a telomeres shorten then the blood vessel gets unhealthy quicker which then feeds back into more hypertension. So that's why I really think it's a bidirectional loop with these things.

So the comments you made, which are if we can increase telomere length by different mechanisms, improve the vascular aging by different mechanisms, we stop that by directional feedback loop.

Telomeres, Aging, and Cardiovascular Risk

Yeah. I was I was curious to see, to read in the reanalysis of the scopes trial where they looked at using statins to prevent coronary heart disease in the individuals who had longer telomeres.

The static didn't really have an effect because they didn't need to. The vasculature was already healthy. What LDL might have been getting oxidized, but it was able to repair itself.

And that, I thought, was another way in which it's tied in I mean, there's other, associations, observational studies looking at telomere length and risk of cardiovascular disease that, you know, taking age out of that and other risk factors.

Still, there is a pretty big, I think, odds ratio, 1.41.5 for the short is third versus the longest third, of that. So keeping your telomeres long, do you think that figures into, you know, who gets essential hypertension?

Has anybody looked at, to your knowledge, whether families with essential hypertension might have shorter telomeres? I have not seen that data. That doesn't mean it doesn't exist.

It makes sense, though, that if you did a a group of, telomeres in normal versus hypertensive, you could easily see a big difference in telomere length.

I'll look into that. I think it's a great question. And there may be some studies out there that have actually done that. I just don't know. Yeah, it would be hard to know.

As you mentioned, it's a there's bidirectional feedback. I mean, if you have, you know, hypertension, you might be causing more oxidative stress and shortening your telomeres.

So vice versa, you know, I'm sure you're aware of probably the tactic trial where they're looking at, using 65 in, people who've had an MRI within the past six months and geographically proven, atherosclerosis and seeing if a year of 65 reduces recurrent arm wise, and through reducing immuno senescence.

So maybe you could talk a little bit more about the sort of mechanics of the immune dysfunction, the kind of inflammation, that we see in, in hypertension.

Is that third response. Yeah. We use, three different, labs, actually, for labs that measure all the inflammatory and oxidative stress parameters and also immune dysfunction.

And I'll just mention those because I think it will help your audience. We use the Cleveland Heart Lab, which has a great, immune, inflammation panel.

We use a pulse which is out of California. We use coral, which looks at, obstructive coronary heart disease. And the newest one we're using is Provincia, which I just learned about about three weeks ago, which is, extremely well validated.

Testing for, arterial health and coronary heart disease. They all measure different things. And so you can have one that's abnormal and the other three normal that because they're measuring different things, you get a really good composite picture of what's going on with your patient.

For example, inflammation markers, oxidative stress markers and immune markers can be present, but they don't yet have a functional or anatomical problem with a disease.

They're just early on. Then the next test, which is, pills. It also is a functional test. It measures, inflammatory markers and growth factors in the heart muscle that predicts the five year risk for myocardial infarction.

Now, once again, because it's functional, it doesn't necessarily mean you've got obstructive plaque, but it means that if you don't get those things fixed, you're going to progress to plaque or some sort of arterial non obstructive dysfunction.

Have heart attack within five years. Oh that's interesting. It's, so this is molecules that are released from the heart muscle. You said. Yeah it's it's interleukin 16.

And then there's a bunch of others that are called eotech. And for us they have very strange names that most people have never heard of, but it's real world validated.

Doug Harrington in California, developed this particular test and and we've probably done, I don't know, 2 or 3000 of these tests over the last several years and really have some validated information correlating with other things.

And so the the usual treatments that you were talking about address, there is a specific way to address the inflammation. Yes, there is we have developed a treatment protocol for the Pls test based on the markers.

And most everything is supplements, plant based diet, nutrition, and occasionally a drug that treats the different markers. And if people follow the regimen, they do have their markers come down.

It might take a while, it might take for about six months or longer. But almost all the patients do have improvement in their, functional markers, but would be something like the top three supplements that you might know.

Let's say we use a lot of curcumin. Quercetin, omega three fatty acids. Resveratrol. And one of the drugs we use is an RB like cordis, for example. Oh, okay. Sure.

It has some really good effects. So McCarthy's will reduce these inflammatory cytokines. Yes. It's through the, blockade of the AT1 receptor, which cuts off the oxidative stress inflammatory pathway.

Yeah, I was up. You know, I've always talked to patients. I mean, when they're worried about taking an ace or an RB. You know, it's I don't want to take a drug.

I mean, it's almost like if you choose the right one, these are almost vitamins for your arterial system, because, yeah, you're really getting at the root of the problem that's lowering blood pressure.

Lowering blood pressure is a side effect of exactly what you're talking about. The reducing the inflammatory the inflammation. But you know what? I explain it to them that way.

They're more willing to, to, to use it. Would you even in the absence of hypertension if you couldn't get maybe you you always do get but get the enough of a reduction in these early markers.

Would you, would you, would you add a mechanism or a, or an Ace inhibitor or something like that to, to even before hypertension? Okay, okay. I'll tell you a big secret that a lot of people don't know ARBs and Ace inhibitors or anti-aging drugs.

Yeah, I mean, I've always but most people don't. I think you're absolutely right. And metformin is probably an anti-aging drug as well. Mean you out Basel is going to try to prove that with the team trial hopefully.

Right. I found out about aces and ARBs in, 2003 at a coaching meeting. It was a researcher from Puerto Rico who've done it in mouse models, and he slowed down the aging in mice by 30 to 40% with Aces.

I mean, it was unbelievable. So I said, well, I'm not waiting for argument study, which will never happen. So I started taking my card. Is whenever it came out of an ace or an hour early on.

I've been on one of those since 2003, and I, you know, I'm kind of a self guinea pig. I check my telomeres and all these other tests, and I really think that it's slowed down, aging, dramatically, in me.

And I've done it in other patients and seeing the same results. So the answer to the question is, yeah, I use a lot of Aces and ARBs in people who don't have hypertension for other reasons.

Inflammation, Immune Dysfunction, and Treatment Protocols

For vascular health and our vascular aging. I mean, I think that is, you know, probably going to be the future. I've toyed with the idea of my my cardio agent, you know, we use was comes off of the, the augmentation pressure and the augmentation index on the Signal Corps instrument was always in the, you know, 20 to 25 year old range and less exercise lately.

And hopefully I can get it back. But I'm going to be 62. So maybe, you know, I don't have hypertension, but I'm thinking about getting started on, on one of those, either an NB or an ace, depending on, you know, if I get a call for the ace and I can't do that, but, but so you're using the then and yourself.

Yeah. Yeah. So. And is it a lower dose? You just, you have to watch to start low unless you've got high blood pressure and still get some hypertension.

So what I did, I started to like ten milligrams and took it right before I went to bed. So we can get dizzy. And then I work my way up and eventually I was able to tolerate a 40mg dose.

And we don't really know how much you need in human to do all these things, but 40mg in my car today is a pretty good dose to take with really no side effects.

And but probably, you know, people talk about lowering blood pressure or causing problems with heart. Erectile, erectile dysfunction. Probably the opposite. Probably helping them.

It it actually, corneas increases nitric oxide levels and probably helps ed. And once you kind of get used to it in normotensive patients, it doesn't tend to lower the blood pressure very much at all.

I'm curious about quercetin. That is, you know, kind of thought of as a analytic, which, you know, a weaker one. I think physicians are a little bit more powerful.

Use it chronically because some people talk about cycling it. Do you think it's working through a set of lytic effect or through, gritty oxidant effect?

Do you have any thoughts about that? Yeah, of course it is an amazing supplement. I take it every day. And also take both the nice thing with it, which makes it even work better.

I have one bio bio, fitness team. Oh, I heard that. So I take, 500mg twice a day. It is a signal that it. No question. And it does get rid of all that stuff that ages you quicker, but it also has other effects.

Mast cells and prostaglandins and, immune function and allergies and all those things. So it has really all the effects that we talked about. The three found out responses are probably treated with quercetin.

Wow. And the reason of course, ten over 15 or just the, together use them together. There's a company, that makes them together. I think it's called life extension. I think it comes together.

Yeah. Great. So those are those are the top three, and omega three is, of course, you follow omega three fatty acid levels with the Cleveland heart testing.

It's they have one of those, and, any thoughts about I mean, there's, I mean, killer length has been associated with longer telomeres associated with higher levels of intake of omega three fatty acids and of course, cardiovascular disease.

I forget like, his name right now. I think it's Harris that's shown, the relationship between omega three. Fatty acids levels, and, and cardiovascular disease around the country, around the world, in different countries.

So obviously, are you in the do you do it by measurements or are you just to two grams a day, four grams a day? And if I'm doing prevention, I will measure, their omega three index and try to beat it at eight or so.

If I'm treating a specific problem, I push the dose higher and the omega three index is going to probably go higher, but I don't get to worry about it.

So the dose can be all over the place. You know, it could be anywhere from 1 to 5g a day. And the studies actually show that if you wrap it around four grams a day, that has the greatest risk reduction for or infarction, there's a

there's also this is probably a good time to dispel some myths.

Talk about all the crazy that have been out there. Yes. And we've had otherwise for years, different companies and universities battling each other. But don't take and take which one, how much DHEA versus EPA?

I mean, it's all over the place. And then as I said, I've really looked into that very carefully and tried to skew out the bad studies like the good ones.

Here's what I think. I could be wrong, but I've looked at it carefully. The first issue was are omega three is good or bad? Well, I think it's been proven now. They're definitely good.

I think that B is gone now. What omega three should you take? Now we have the argument that we need more EPA than DHEA. It seems to be more biologically active.

And if you took too much DHEA, it might counterbalance the effects of the EPA. That remains to be proven. That's a theory at this point. But some of the formulations that I did, ten years ago were mostly EPA because I had talked to a lot of omega three specialists.

The one that I use, which is from biotics, is two thirds EPA and one third DHEA. That's one point. The other very important point is that, maybe three fatty acids become oxidized not only in the bottle, but in your cell membranes.

And you say, well, how do I prevent that? Because I don't want that happening. You put gamma delta tocopherol in your cell membranes and in the capsule.

So, there's a certain does she need for that? And it's gamma delta tocopherol, not alpha and not beta. So you have the right dose in the right to control.

And then the fourth point is when you give high doses of EPA, DHEA, you deplete GLA. Most people don't know that either. And that's not good. Just GLA is the least poor up the higher link there, is a good omega six.

So the formulation with biotics, which is EFA supreme, has all that in there in every capsule. So you just have to figure out how many you want to take in your home free.

And it's good quality. Now then we have the next thing that popped up, which is atrial fibrillation. So all these studies now are suggesting a small percentage of people on omega threes get AFib.

Well the question is what type of omega three were there taking. What was the quality. Was it balanced.

Omega-3s, Supplements, and Anti-Aging Drugs

How much EPA, DHEA, tocopherol, GLA was in it so that that whole thing about AFib is out there. But I think we still need to be careful about assuming that that's a big problem until we start saying, let's really look at high quality studies with high quality make threes, make sure that we're okay.

Well, you made a bunch of fascinating points here. A couple of follow up questions. The first one is about atrial fibrillation because I've been seeing more of it in my practice.

I do use a lot of fish oils. I'm not aware of that study. So I just love to know if just briefly what the what you know, what the author of the citation, so we can I can just look it up on published probably. But you know, that would be interesting to see the other point.

The interesting point is the from a vascular standpoint, EPA potentially could be more important, but you talk to the neurologists and the Alzheimer's people and they're like, yeah, is only important insofar as it gets turned into DHEA.

And you know, if you have too much TPA, then you might, you know, might mess that process up too. So, I mean, I think it's probably they're both true, maybe from an inflammatory, prostate gland and standpoint in the vasculature.

EPA is more important. But within the brain, DHEA kind of rules. So, I think most fish oil, the omega three products are about for whatever it is, 60, 40, something like that. If you can't get one that usually has more DHEA than EPA, right.

So you're going to that's going to happen anyhow. But, you know, it's kind of interesting. Do you have any thoughts about the the EPA versus, you know, the Isocal I, I Pinto versus, and whether there's really any benefit I mean, farmer wants us to think there is, Right.

So. Well, those are both mostly EPA, right? Yeah. It's, but the problem is they don't have any GLA and they don't have any tocopherol, so it's still an ox about oxidized form.

So I don't typically prescribe those. I usually use the biotics preparation because it's two thirds EPA and one third by Richard Point is exactly right.

You ask yourself the question, why am I giving you omega three to begin with? Well, if it's for eye, brain or blood pressure, DHEA works better than EPA, right?

But maybe for other things. EPA better. So when I developed this and I talked to a lot of people, I said, can you tell me what's kind of in nature? I mean, if I go eat cold water fish, what do I get?

They said it's about two thirds EPA and one third I said, yeah, exactly down there. That's what I'm going to do, right? Yeah, I think that's, that's that's probably a good way.

And, any thoughts on krill oil? Some people talk about krill oil being better than, than, than, omega three from fish oil. I know the concentrations are different.

There's less of it in there. But they say there's some benefit to it. And krill oil, I'm not a big fan of it because there really aren't a lot of clinical studies with krill oil or anything.

Right? Lipids, blood pressure or food. That's not to say krill is isn't good, but if you've got to make it threes that are proven, I would go with that over krill oil.

Anytime. Yeah. There's a lot of people that say, well, it's because it's better because they're lower in the food chain and so there's less chance of concentrating.

Mercury or other other, pollutants in it. And, you know, they distill these things. There's no mercury, right? Need to take care of that. Well, that's out there.

But the supplement industry is very happy to talk about, so in particular the crab people, well, so that's, that's, those are really good points. Particularly fascinated about the Arby's that you started.

In terms of what you actually do with, protocols and whatnot with, with to 65 and telomere testing. How do you approach that in your patient population?

Right. So there's, there's one company in the US that does the PCR telomere testing and that spectra. So and then the other one is, you know, is out of Spain, and they actually measure short telomeres.

And I think most of us feel that the shorter the short telomeres are really what drives the aging process rather than the average. So if you're going to do telomere testing, you can pick which one you want to be.

You got to know, you know, what the difference is. And then we measure it once a year. And we do all kinds of things a lot, much of which we talked about, you know, controlling all the risk factors to 65, nitric oxide boosters,

galactic boosters, exercise, plant based diet.

And we do see improvement, in telomere function over time. And it's very highly correlated with, vascular aging and all the cardiovascular events. Yeah.

I, I, I definitely see people and I've been treating people with 65 now for 14 years. You know, most of them don't have any loss. Some have an increase. In telomere length.

I would give you a third option, though, for, for tailor length testing, which I think is is as good or better than the other two. Is repeat diagnostics in Vancouver, Canada.

Oh, good. I didn't know about them. They do the same sort of flow fish type tests as, as life length in Madrid does when you mentioned, which, and they're both good companies, but you get both the granulocytes and the lymphocytes from repeat diagnostics, which gives you a measure of, you know, all the slings and arrows from an oxidative stress.

Viral infections, psychological stress standpoint, causing shortening in the lymphocytes. But then the granulocytes, which of course of the neutrophils only circulate for a day.

And so you really get a reflection in the bone marrow stem cell telomere length and your inheritance. And you can see that gap large and getting larger between the leukocytes of the brain of sites.

When you, see spaces to have a lot of low allostatic load, as they call it, from infections, from, from stress, from, you know, bad diet, from being overweight.

So I think that's pretty useful. I would also say that while, PCR is great for large, studies because you batch it, there, you know, it's, inter subject, variability and trough temporally is pretty is pretty significant.

So I take a look at repeat diagnostics. They do all the, the children's hospitals, field length testing that they have, I think really a good coefficient of variation around, 2 to 3%.

Wow. That's great. Okay, I want to get there. I'll definitely look at that. Thanks for that information. So, so I think that you, you know, dispelled myths given us Perls.

And your approach. Is there anything more you'd like to talk to me about? Which one other major point it's very important. When you're treating hypertension and the blood pressure is definitely elevated by, the criteria.

We've talked about office ratings in ABM or home reuse, whatever you want to use. You don't want to wait to get the pressure under control. So people will say to you, well, can I try diet and weight loss and some supplements first?

And obviously I'm I'm all for that. But here's the problem. If I weigh even, you know, one month or two months, you'll never catch up and be as healthy as if I started today.

So my approach is let's get you on a good drug for blood pressure.

Genetics, Resistant Hypertension, and Early Treatment

Start your other lifestyle programs, get the pressure to 120 over 80, make sure you're dipping appropriately and all those kinds of things. And then if that's done, you can then back off later on the drug if you're getting healthier.

And that's that was there's a lot of studies that have shown that. So the point I want to make is the earlier the better the normal. That is the really fascinating I think a good point.

I mean, that goes against what most other doctors, I'm sure do, which is to say, you know, if it's not 180 or 200 over

120, they're like, you have some time, you know, you might have a stroke out, but saying you're damaging your arteries with this high blood pressure and you're never going to be as healthy if you wait even a month, which that that's incredible, is, I think a paradigm shift in some doctors there may be listening to this, and most of them probably, and I myself, even, who was very focused on subclinical disease.

You know, I'll have that conversation with patients. But I think, you know, I you've changed my mind. I think that's that is the right way to go. Really.

And that makes the most sense. Yeah. In patients, if you if you approach it that way, they won't, give you too much trouble about saying, I don't want to do what you're saying.

They're all for getting healthier. And and then you demonstrate that it works. So they're all in. That's right. Like you told me you were draining their 401 K when you could wait to sort of slow down the draining, but we can stop it now.

And they'd probably say pump it now. And that's. Yeah, it's a biological 401 K. Yeah. The other thing I'd throw in here is when you have a drug naive patient, we stratify, their hypertension into high renin and low renin and that's easy to get.

Quest labs does that plasma in an activity and, out of serum levels in the blood. And you do them at the same time, drug patients and tells you which group they're in and it tells you what drug class is going to work the best.

Like for how renin hypertension, ACS and ARBs work the best low rate and hypertension, some of the better diuretics and calcium channel blockers work best, so that gets you straight into a quicker analysis of what you need to give them is going to work better.

The other thing we're doing routinely now and in all of our hypertension and CVD patients is genetic, cardiovascular testing. And I want to give you the name of the lab that we use.

It's the best in the country. It's called vibrant, vibrant America, vibrant in America. Labs throughout a San Francisco. And I developed this genetic profile with them about five years ago, measured about 25 different snips.

And I got to tell you when you get this back, you can figure out much better what type of hypertension they have. What do you use to treat it? You've got their coronary heart disease in my snips or diabetic snips or lipid snips.

And you know exactly what to do to turn off the gene, which is called gene expression testing, which we can now measure with all those tests we talked about.

Right, right, right. Exactly. Those are things that should become routine, I feel. And hypertension management. And how about even in because there is basically when we talk about hypertension being accelerated arterial aging, there are people who don't have hypertension when you're seeing them at age 40, but probably will have it at 60.

They have a little bit steeper trajectory towards hypertension than the average arterial aging, which is, you know, making it to 8090 without high blood pressure.

Should we be looking to see what they have? Maybe some of those snips and sounds like we could. Yeah. Because, what I see, because I see so many referrals, I get all the resistant, hypertensive.

They're coming on five drugs, and they're just totally irrational. And I said, is anybody ever checked your genetics to see why you're hypertensive? And, of course, none of them have, get the vibrant technology genetics on them.

I did have, one snip that's related to a specific drug that would work, most of which they're not on. For example, some of these the some of your listeners probably never heard of a metal rod.

All right. Yeah. But so yeah. Yeah. Right. Well, I mean arise become the new kid on the block. Is that right? It walks

the sodium epithelial channel and about 30% of people have resistant hypertension, have an upgraded sodium epithelial channel in their kidney but also in their arteries.

So their arteries are full of sodium. The body's full of sodium. You. Nothing works. You can give them everything you want to, but you put em on a metal rod by itself for like 6 or 8 weeks and the pressure comes to normal.

Usually you may have to give a diuretic with it for a while, but most of the time you've replaced five drugs with one drug. Wow. That's one. You see what I mean? For 11?

That's the snip you get. And you say, oh, I've got it fixed now and then. The other one is huge is, one that's related to Alice. Tyrone. Yeah. These people make too much Alice. Tyrone.

But they don't have primary elbow, okay? They, they they look like it. But when you do their testing, you don't see these really high levels of Elvis.

Tyrone. They may be up a little bit, but not huge. But their, their arteries are very sensitive to just around the receptors. They make too much because the Alan Alda serum synthase gene is cranked up.

So you get that snip, you put them on spironolactone or platinum. And about 6 or 8 weeks later, their pressure comes to normal. That counts for another 20 or 30% of resistant hypertensive.

Those are all absolute pearls. We, amyloid is an old boulder drug was used in pregnancy, right?

Book Promotion and Closing Remarks

Oh, gosh. Yeah. It was it was, made by Merck. It was, hydrochlorothiazide and the metal rod. Right. Together. I don't remember the name of it, but that's where.

And you can still get it and be by itself. And it's an incredible agent. It's very popular here in Nashville because we prescribe some. Right? The pharmacies, they keep it in stock.

Then they do. Yeah. Sounds good. Well, listen, this has been a really fascinating conversation. And, you know, as always, learned so many new things. Whenever I go to one of your lectures.

And today I've been able to ask the questions has been, a pleasure and an honor to have you on the show. Look forward to seeing you at the next meeting.

Probably before him or. I know you're always flying around doing something, so, again, thank you very much for being on the Telomere Summit. Let me ask you one other thing.

If I may, can I do the plug on the blood pressure? Good for us. Oh, sure. Yeah, I forgot about that. Absolutely. Yeah. So I'm going to show you the book.

This is, see if I can get it to make that right there. Close your camera. I got a copy on Kindle. And it's, Yeah. Great. So there a little upside down there.

So you got this right? Just a little lower. Perfect. Controlling blood pressure through nutrition. Nutritional. So this is, published one month ago. You can get it on Amazon or most of the major bookstores.

And it's got everything in it that we've talked about, plus a whole lot more nutrition, supplements, lifestyle, drugs, diagnosis, treatment, testing. I mean, literally everything we've talked about is in there.

It's written for both the light public but also for physicians. And, hopefully you'll find it helpful. Yeah. No, I, I got a copy of it. I, it's it is written in I really understand it was laid out in a really, really nice way and then practical as well.

So I highly recommend it. Again, great talking to you. And hope making a cross pass real soon. Appreciate your time. Thanks for the interview.