Introduction and Shoemakeru2019s Work

But I've had. Yeah. So welcome. Welcome back. This is, another edition of mycotoxins and chronic Illness. And my pleasure to continue a conversation with Doctor Richard Shoemaker.

That's it. For those of you who don't know, Doctor Shoemaker is the, pioneer in, in in toxin, in mycotoxins, illness. But, especially in, the areas of water damage and just chronic inflammatory response syndrome.

I mean, he's the man who coined the term, and he's the man who has, developed the treatment tools that we all use every day. And, someone who I have learned from and continuing to learn quite a bit.

He has developed a, with the help of, Jimmy Ryan, a, PhD scientist. He has, been working on the gene test, and we've talked about that at length. Our first episode, we're going to talk just a few more minutes about it, because I want to talk a little bit about, touch upon Covid, hyper coagulation and then a few minutes on glucose, and then we will talk a little bit about and wrap up talking about water damage, buildings and, what is it in them that may be causing our problems?

So, which let's, the Covid, I mean, what I said I've had, not much experience with Covid patients, I guess. Thankfully, though, I do feel like I was left out.

But when I did see a handful, and I got blood work on them within the first week of either symptoms or exposure or positive test, and I was very impressed with how elevated, their D-dimer were and, and their ferritin.

Right off the bat, I mean, these in some of these people, their D-dimer were, you know, not high enough to get a hospital. Stay excited. They're only like two times normal or three times normal, but they were essentially asymptomatic.

And so to me, it looked like I think a lot of at least perhaps a lot of long term Covid symptoms are because of these, maybe initially silent micro emboli or micro thrombotic events.

And I was wondering what your take is on that and what you what if you've seen anything that correlates with that, looking at Covid patients and the genie test.

I'm looking at a graph or a table. Excuse me. That is going to be this is in our new paper on post-Covid syndrome. There I asked the question is post-Covid syndrome really just Chris?

COVID, Coagulation, and Post-COVID Syndrome

And as far as the markers, it is a serious. I think so we look at our percentage of control patients who have coagulation abnormalities in gene upregulation, 5%.

That's that's not not zero. But it's not 10% either. In our full blown surgeries, people water damage buildings 54%. So big difference cases versus controls.

But interestingly, other people had Covid and six weeks later did not have symptoms impairing activities daily life. The abnormalities was 14%. Okay. So they did have impairment of of excuse.

Do I 36%. So the p value is less than 0.00. 0.4. So it's not as good as we want 36 versus 14. Sounds impressive. We didn't mention the null hypothesis.

But it's not enough to show that Co egg is the main problem that we see in in in Covid compared to where it's much

more dramatic. Right. Okay. Well, it's I said that was a very small end of mine and just an observation.

But especially in acute illness patients, I'm looking at people six weeks after their Covid is over, after they had a negative test. They feel well enough to be able to go back to work.

But yet, if you well enough to work in the lab, human activities, daily life, and if Covid, you still have coac abnormalities 14% of the time, there's not a small number.

It's a lot. So when we look at what Kimmi showed in 2016 with cytokine elevation and Co elevation in our Sirs patients, yes, 54 is more than 36. But our control patients were five.

And in control patients here was 14. Yeah. The control patients were small and group was able. Symptoms control patients out of 37 symptoms 2.7 is a mean.

As we published the surge 22.3. That's a lot published that. But six negative people were not brought back to work what have you. 8.2 symptoms compared to cases of positive 18.2.

Now that was significant at 0.013 18 versus. Yeah. So just to remind people is PC is post Covid and PC as is the post Covid syndrome. Just so they know who we're talking about.

Thank you. Yeah. And so this is you know what I, what I, what we'll see over time. I hope it doesn't happen. But those, those people with that the 14% with the abnormalities in the coagulation pathway, what happens when they've been walk into a water damage building.

The point is, is that we're likely to find, you NPC's -14% out of, you know, reactivity. I just mean it appeared that 56% of those were full blown illness and 87.5% had precise, specific causation.

And so we were really looking now at a landscape approach, what happens to a Covid patient who walks into a wet building and stays there? Stay more than two days.

You're going to have some medicines who have normality. The turbulent enclosure that I mentioned is 66% and serves patients less than 5% of controls. That's really pretty dramatic.

But in PC positive cases, 50% wow. Wow. 50% have got problems. But then we say, well, that says there's going to be a lot of proliferative physiology.

Yeah, I'm saving the best for last. If we have zero in our control patients, 0% in molecular hyper metabolism and 85% of untreated surface patients, their treatment naive have molecular metabolism, you know, how about negative?

Do you think they'll have a low number of molecular metabolism? I would hope so. But I have a feeling the answer is no. The answer is zero. Okay. Oh, really?

Oh, sorry. I thought it needed ten. But how about the PCC's? Positive. What percent of our 14 patients is a small study had, PCOS positive in hypo metabolism?

100%. Wow. Okay, so. So these people. Yeah. These are your patients. I mean, that's that's been my healing from as soon as, as soon as post Covid hit.

I mean, it's like, you know, it's chronic fatigue all over again. And to me chronic fatigue is the same thing is chronic inflammatory response syndrome with flake with different flavors sometimes, but pretty close. So what do we know.

That was surprising. It goes back in with the PKS positive versus negative for KD three D defective antigen presentation 86% of PKS positive patients had suppression of CD3 d 86%.

In our Sirs patients we have 96%. Look at that PKS negative still 14% effective antigen presentation. And we're telling every one of those patients to get a Covid vaccine.

Let's see what happens. So do they have it? Have you have you checked it? Do they develop titers or, you know. No, no. No one's doing titers tonight. No, no.

Yeah. So be interesting or T cell activation test and see. So a to to do required. Yeah okay. We want I won't go into that now let's but this is this is amazing.

And you know I mean there's there's so much money. So what the the NIH is going to spend $1.5 billion or something on post-Covid syndrome. Do you think I'll throw you a few pennies?

That's the number I got. But still, you know, if they spend the one five doing some gene testing, they would know as fast as I did that doing what they've been doing is just wrong headed.

And yet you can't tell me my age is not invented here. Means they didn't think of it, so who knows what that means? Yeah, yeah, I know that. That's that's really a shame.

Why don't we have decent answers? Because people are protecting their turf. Yeah, and many times it's. It's sad if people don't understand, you know, they only know their turf.

I mean, that is always the problem with the NIH is that is that when you bring something new to them, they don't know what it is, you know, so they just judge.

It's the old story. If I didn't hear of it, it can't possibly be true. I mean, we see this in medicine all the time, you know, it's just, until I until I heard about it from the person who I know knows something.

But the problem, I always say with experts in, in, chronic diseases myself included, is that we're often expert in what doesn't work. Because if we knew what worked, we wouldn't have chronic diseases.

Who we have treated them anyway. So, talk a little bit. I just want a few minutes about glucose control. I mean, because, you know, blood sugar. Well, insulin, I mean, really, to my mind is the bigger issue, which is, you know, tell me a little bit about what you've been finding about insulin and, control of insulin production and how it affects the, the fat cells and, yeah, metabolism.

In the metabolism paper that was published in September of 2020.

Glucose, Insulin Resistance, and Metabolism

I go through that in some detail. It's a free download from somebody on the website. In your spare time. Maybe you can take a quick read. It's the density for you, of course, jargon filled paper I've ever written in my life.

But having said all that, what was interesting is that the problem with proliferative physiology is it's bad for people with molecular potential isn't.

But the problem with prolific physiology is truncated tremendously. If you don't have MHL. And what I mean by that is that if you are not breaking down pyruvate in in the mitochondria, but you're delivering glucose into the cell, what will happen is some of the glucose is diverted as it's metabolized into the biosynthetic pathway.

It's designed to treat and create the ability for cells to divide. Yeah. I mean, look at the mucous membranes. We have, you know, in our body, how often do those cells get replaced?

Well, if there's one on one corner and one on, quarter down, another quarter, that's four corners. One of those cells be replaced every every four days.

Yep. Now our mucous membranes will fall apart because they pop. Ptosis is proceeding along nicely and beautifully. But if you have apoptosis, you've got to be making another mucous membrane cell.

You've got to have proliferative physiology to a certain extent. So when you look at anesthetic glucosamine, it is able to bring about, ubiquitination and phosphorylation and all these different changes in proteins, including proteins that are involved with insulin resistance in fat cells.

It's not in monocytes, it's not in anywhere else. It's in fat cells. But that situation is now giving rise to our ability to measure genes that are found in white blood cells.

Oh, to you know, TG, you know, if you want to have, insulin resistance, turn on, okay. If you don't want to some resistance, turn on RTG and all these different mechanisms the cell has that we can access.

And if we can diagnose it and show the maladies, then we will be able to create gene solutions that we haven't had for obesity and diabetes. Type two.

Yeah. And this makes me really I mean just one of the things you said about, you know, how often we see either a failure to turn on or off apoptosis. And I mean we always think of it in terms of, cancers, but my God, you know, almost every, you know, just even things like, osteoarthritis and, in degenerative tissue, diseases of all kinds, you know, we're failing to turn it on and turn it off, keeping it in balance.

Balance seems to be the key. I had a lady yesterday that I had not talked to for nearly 39 years, and she is her husband has passed away. And so she's been kind of doing other things.

But her problem is it must be ferocious. And she's taking some prolia or some drug advertised on TV shot at risk. But then the question is, where did the osteoporosis come from?

And I said, have you had your TGF beta one measured? Oh, what's that? I'll teach you. Theta one turns off osteoblasts and turns on, so your class now will do it.

So if you want to rot your bone, she's going to have a hip surgery done because of osteoporosis. She just had a lumbar spine fusion with rods put in six weeks ago.

And your shoulders are falling apart. And rotator cuff problems. You know, this actually is kind of a malignant form of last year paralysis. And they could be using calcium, but it turns out that she is going to have to pay better. One is probably going to be 20,000.

Well well right. And the amateur response syndrome and how to treat that going gonna be able to help her with the IPA. Those are. Yeah. You know it's it's and it's just I mean I think that that's another course in itself for you to I don't know if you've done this one or not, but just all the places in medicine when we should, where we should be thinking, which is basically everywhere.

Sears is happening. Review article in Jama. Not too long ago, maybe six months or two. That is going by fast, but it was at the Covid. The view from cardiology covered you from neurology covered the view from home.

Pulmonology. There was a bunch of allergies, and every single one was something that my docs take care of every single day. Yeah. So this is this is what we see and this is what we treat.

But, since these are the these these are the patients who've always had normal blood counts, conventional medicine has usually ignored them, until they get to the point where their, their system is broken and then they can give something else to suppress it anyway.

So, Yeah, so, so fat, fat and glucose and glucose metabolism, is, is just such a huge issue. I mean, that was something that you talked about early, early on in your career.

One thing you think about is that if it's a good thing for for proliferative physiology to have the biosynthetic pathway active, that's going to be necessary.

If you've got pyruvate reduced entry into the mitochondria, if pyruvate is increasing and entry is dependent on here, then we don't need the biosynthetic pathway at all.

But that's not the only way that insulin gives us trouble. Iris too. And some receptor substrate two opens group glucose channel one good one and glucose channel four group for these cycling pathways.

Let glucose increase at the entry to the cell through wide open pores and it's cell membrane. But the other mechanism where it really has to do with water damage, buildings and insulin resistance has to do with insulin bound to an epsilon receptor on the cell surface, holding on to the glucose in the hot little hands.

That grouping that moiety of three different structures is engulfed painlessly ptosis, rando psychosis, whatever you want to say. And that little bubble will come into the cell where it'll stay a bubble, keeping insulin away from being recycled until that bubble is acidified.

And if the cell pumps hydrogen into that bubble, the bubble will open up, release glucose from metabolism, release the receptor, be recycled, release insulin receptor insulin to be recycled.

But what if there is medicine or mine, like the little mice in present in that cell? They're acting in mice. These compounds, they work by preventing acidification of that vesicle.

So if you don't acidify the vesicle you don't release, you don't recycle insulin. And so receptors. So what happens is that with normal blood glucose you are insulin resistant.

Right okay. That was big back in the 80s back when they they put one ounce in a chicken feed. I used to laugh at people who had weight loss desires. They were going to eliminate red meat, but they still eat chicken.

So they're guaranteeing the chicken to eight minutes is 100mg per ton. The you can see that's a lot. And you go through a bunch of tons of chicken. And then one incident nano molar amounts will hurt you.

They're actually creating more insulin resistance by eating chicken than they were. We're eating fatty beef. Well is that's that is no longer being used in chicken feed.

No, no. Hardly at all. Well, they switched over to, using inoculations and vaccinations and someone's going to be upset about vaccinations, but that also has taken a toll because now we don't have moments and killing I'm area, which is an AP complex and parasite that kills little chickens and little, little little pigs.

We also have an avian cells, the, psychosis this being a problem, we're seeing an explosion of the VCA, IG and IG and positive tests that are false positive because they're not killing.

I'm area with the weather with an antibiotic. Like when insane. Okay. We can't wait. It's interesting. So yes. Guinea just, Yeah. Acidifying those, makes me start thinking about, I won't go there.

Oh. We used to use plaque when able to do that in the old days, when we use, actually, we still do sometimes when we use, the macrolide antibiotics. I wonder if it if it has the same effect.

That would be an interesting thought. But, so getting back to, I say, the real heart of the matter for a lot of our, our listeners is the, you know, we've, you know, everyone has focused really on mycotoxins and, you know, your recent papers and, you know, your work the last years has been that wait a minute.

It's water damage buildings. It's these actinium, icy species, that are producing, the, the, the chronic inflammatory response syndrome. And the problem that we get most of our patients are having, just, just real quickly how how did that how did that, metamorphosis happen, you know, how did you see how did how did you, realize what was

going on?

Water-Damaged Buildings and Actinomycetes

The expression we've used a couple times today is that we go as far as science lets us, and the newer diagnostic techniques that are the basis for new insights into causation of these illnesses is something we didn't have 4 or 5 years ago.

So we are told in to and beat in our head that mycotoxins were pro-inflammatory, and that if you had exposure of instilled mycotoxins into a test animal, usually a rat or a mouse, there would be this surge of inflammatory responses and arrested poorly.

And they died if they had done the same studies in pigs, they would have seen even more efficacy of mycotoxins killing things. The Chinese were able to show that in Chinese that the pigs are the most sensitive mammal to the effects of mycotoxins, but if they supplemented with glutamine that the micro toxic toxicity disappeared.

So I think, in the normal American diet, about eight eight ounces approaching the day, how many milligrams of glutamine are there that we're eating? At least five milligrams a day. Yeah.

And who gets micro toxic toxicity? Is primarily it the papers that say this is a horrible thing that's starving horses in, in Russia or people who are getting aflatoxin in Africa.

And what are the people look like who get aflatoxin in Africa? They've got bloated stomachs and they've got quasi work or their protein diet. If we give them five milligrams a day of glutamine, then the micro toxin toxicity clears up.

Wow. And you have, I mean, have you have you tried that clinically? I had everybody get an extra protein right? It works. Okay. And. Right. And but yet, you know, we still seeing.

Let me let me just continue. Yeah. Sure. Issue was still dominant despite the to the Chinese finding about glutamine and protein deficiency creating a problem.

But when we finally started finding that actually those were present hugely in buildings. And it turns out that literature started in in 2010, it was not a new literature.

When I wrote Surviving Well in 2005, Christian wrote more words and surviving in 2005. We didn't have the information about that in us. We certainly didn't know about endotoxins.

And in 2010, when I wrote Survival Mode, we're just starting to barely see that remediation by ways we've approached it for years didn't work like the toxins we would would would be removed where people still would stay sick.

What was left was that us? You would knock down some of their their substance, but not much. That tendons are different than mycotoxins. It was like an alcohol environment.

But they also like company for being sitting there by themselves on a piece of drywall. They release a compound, a fair amount, if you will call trimethylamine or TMA, and you know about another type of trimethylamine oxide and smelly fish and histamine release and all that.

But specifically TMA will attract other other, to those other bacterial species to grow a pseudo part of new fake leg, so to speak, that let them rise up and explore.

They can cross over glass membranes that are barriers. They travel great distances to join to their friend who release a DNA TMA release locally, creates an alkaline environment.

Do mycotoxins grow well in micro toxin in any environment? No, but what you have is actions that you can't see

versus mycotoxins. You remind them of that mold.

You can see a lot of the time, but acting as you can. But whatever there is, the species diversity of 16 plus showing the TMA is active. You know, I have TMA is active.

Then the mycotoxins are involved in secondary metabolism, making river toxins like crazy. They also turn on compounds that turn on TGF beta one signaling devices.

Oh my God. These organisms make every antibiotic known to man with every biochemical known to man. But they also turn on TGF beta one. What TGF beta one does is then create the precise, specific causation of the presence of mind of other acting. Us.

So I mean circle, what we're doing is using the physiology in growth phases of actin is the answer questions are why are they so common in buildings? Why do they hurt people so much?

Why do they cause so much pulmonary fibrosis? Well, there you are TGF beta one. Signaling mechanisms involved turning on some of the exact same genes that insulin does.

So if insulin is developing resistance or we're how come we're not seeing resistance TGF beta one again. Well maybe we are. Just fast yet. Yeah. I mean, you know, it's and that another conversation is Sunday is just the whole I mean, TGF beta one is one of those fascinating molecules.

I mean, it does so many different things, milieu dependent, you know, it's just amazing. It's, ability to send to regulatory cells in a tissue that's inflamed and.

Well, and if the tissue has reacted retinoic acid receptor, that signal will change the T cell into a T effector cells. It's called classes Z plasticity.

And they plus the plasticizers T effector cell releases more TGF beta one to send more T cells to their death in conversion. So what do we get rising TGF beta one and lowering T rex cells.

As I said, we can start measuring that with geni now. And 17 will follow from rising TGF beta one and two. Egg deficiency will give an increase quotient between 2 to 17 over 80 red cells.

What happens here is cardiovascular acute coronary acute carotid syndromes. Keep the aneurysm syndromes like crazy. And the days are coming because of the the the proteomics of 72 look like sirs.

But it's not sirs. They don't have the same mechanisms. They don't have the same, proteomics. And this is they certainly don't have a they have a group the same symptoms, but they're not the same thing.

So as we learn more about sirs, we're learning more about lookalikes. And the ability to manipulate T regulatory cells has got to be something we go to in the future.

Yeah. Yeah. I mean, I think you said it in the beginning of our first conversation. You know, regulation, lack of regulation, lack of regulation, lack of regulation, of lack of regulation.

And, yeah, you're you're you're you're you're immune. Dysregulation to me is where, again, these chronic illnesses all, have in common is you've got inflammation that should be transitory or and it's not I mean, if we can get it to go to, to be, you know, it's what keeps us alive.

If we can turn it on and turn it off. There's an analogy from biology just to throw in, just for sake of discussion. And that is the toxin antitoxin system.

Perhaps you've heard of. Those are 6000 different species of bacteria that will use a toxin to survive intracellular life or stressed life, and then they can turn on a antitoxin to block their toxin, to stop the the hibernation or torpor.

You know, one of your papers with Bob? Oh had a of the summary about dower. Right. You and Neil had to do some work. Something to do with that paper. But what you had was dower what you were describing as an aerobic glycolysis.

It's not it's an anaerobic glycolysis. So a fundamental mechanism because dower does not do the same things that hyper metabolism does. You're talking about down and down is not the same as what we're talking about.

Right. Well I think it's just stages, you know I mean I think that that that is the point is that, I think what Bob was referring by the hypo metabolism, but he was, I think, fully aware of and interested in, in the area you're talking about is, a cause with the, aerobic metabolism.

Right? Aerobic hypo metabolism is what he I said would he describes, you know, very clearly the DNA at all in his paper, I think. No, no, no, he wasn't talking but he was looking at it from the, from from the, looking at, from metabolites.

And I say that's something that you to, I think would be a great conversation. In that it it's apparent it's there, there's it will shed a lot of light on other fields, but I think at the end of the day it's, you know, one of those chicken or the egg conversations, you know, in the case where I'm not well, I'm not so sure.

Well, it's, you know, the, the, you know, whether the metabolite is the signal is the primary is the first signal or the, you know, the change in, in, in, gene transcription is that is the first signal.

It's like they both signal each other. So it's really hard to, you know, I mean, like, you need them both. Bob could control for translocations and control for.

Obviously that would make a big difference because those are two external factors that were not discussed that have a great deal to do with entry of pyruvate into the into the mitochondria.

Right. But I mean, he's just saying it's the mitochondria sensing the difference in the pyruvate levels, amongst other things. And amongst that, that, that then, get the mitochondria to, to change their shape and change their function.

And we'll get you into, that, that, that iPhone but involving nuclear encoded mitochondrial genes. Yeah, yeah. That's a that's what I'm saying. It's like you're saying you're it is the chicken or the egg which came first.

And, you know, it's I think but what would interest me is not so much who, which came first, but how each one will illuminate the other is because we might find things that we can, modulate through, the metabolites and things that we can and ways that we can also realize with, with your model is.

Future Directions in Environmental Illness

No, which level to be attribute to be addressing because you're looking at he needs to do is to fix transfer cases and VDC. I think history is this is where it's all going.

Well, yeah, the question is had it is is how to go about doing that. You know, is had it is how do we restore cells to, you know, cellular regulation.

I mean that, that, that is that is the question. The IP is one of the is one of those ways that that, obviously does it. You know, we're using a lot more other, peptides.

The interesting I mean, I think that's interesting is, you know, at least, the IP is one of those peptides that actually has genetic control, direct genetic control.

A lot of the peptides that we're using clinically now are probably more, and, derivatives of albumin and other and other proteins. So, I mean, that's what makes it so interesting is that it's directly it's got direct genetic control.

Right? But, you know, it would like point is that, I just think it's, you know, the chicken or the egg and the real issue is what's going to give us, the ability to modulate the system.

I mean, for Bob's world, you know, he's he's looking to control, to modulate the ATP receptors, or the pure energy receptors, and just give the cell a timeout, a chance to return back to a no more normal physiology.

But, I said I'd like to leave that conversation for the two of you. I think it would be much deeper and, would, you know, I think you both you you both see things, I just want to share a couple words about the future. Yes.

We've been in this business for a long time. This is my 25th year. And I think you probably you encounter fatigue with what it was in 1996. You started.

And I've seen a lot of ideas come and go. At one time, I had kept track of all these ideas. And when they when there was there were 12 references to what was a new guy on the block and XM was exciting.

Judy. Yeah, I mean, it was just one us. There was one after another. It was it was it? Oh yeah. Yeah. And the constancy in, in my approach is still been to avoid exposure and remember that we are mutating organisms inside water damaged buildings constantly.

So you heard a lot about mutations and variants for, for Covid. I think they call them variants because they don't want to alarm people saying mutations.

But we are creating monsters in our indoor environments where the chemicals we used, we saw with Benadryl and, substituted beds, medicines, all would do bacterial resistance and mutation.

We're doing that with Esther Connors as well, which is abnormal. Or it's just it's just wrongheaded so that when as long as we're going to have concern about human health, I think we need to have concern about environmental health.

And it means the physicians need to be more aware of what's going on in their environment compared to what they do and what what they don't do. So that as we go forward, I think it's going to be important to expand the training not only of the patient population, but of the medical population, to know what the difference is and not just, say, pesticides.

We come up with mechanisms for insecticides and herbicides and fungicides. This is three main classes. And as patients should be demanding the positions who are involved and giving them chronic care have a broader, more landscape approach to what environmental illness really means.

Absolutely, absolutely. And I think that's where, you know, I think the natural paths have been leading the way, you know, trying to, you know, for years, you know, Lynn Patrick and, you know, beef before her.

But, and, we just unfortunately passed away a few years ago, you know, really trying to get that that knowledge out there and getting people to ask an environmental history, you know, really, really where you live, what you're exposed to, what's in your, kitchen cabinets, you know, what are you cleaning with?

What are you using for for the common household things. I mean, this is an area that, Yes. I think by and large, physicians have. I mean, they they just, you know, having paid attention, if the if it's the FDA, not for the, if the FDA says you can sell it, they assume that it's safe.

And, I think we can really see the results of that. It has not bode well. I mean, on some level, I think, you know, I believe that that's why, you know, the the level of, of these chronic diseases has skyrocketed, you know, and also the autoimmune diseases, I mean, we just didn't see these.

I mean, back in the 70s and 80s, we just didn't see this level of autoimmune diseases. You know, I mean, it just and sick kids. And I'm not willing to concede that point.

I think the clear mechanism of awareness in society has changed. For example, the word gomer was one I heard a lot of men. Oh, sure, go to Gomer. Well, there was someone who had 25 symptoms and nothing was wrong.

But they could find in certain measurements. And Gomer stands would get out of my emergency room. So the medical profession as a whole is acting with a sense of arrogance towards the illnesses they don't understand, and creating in examples one after another with with psychiatric diagnosis when they just didn't know what objective parameters real Mr.

Genius coming in and making a big difference. So you need to will make more of a difference. This is going to way the future, when people in the medical practice and the medical profession are involved in learning and resistance to learning new things, and we really have to novel because there's something new coming out of the woods every single day.

Yeah. Oh, absolutely. But, I'm hopeful because we have a lot of young physicians who are excited and really want to learn new things. I'm always a little less less optimistic because we have a system that is now run by accountants, and doctors are no longer running the show.

But, you know, accountants and lawyers are running medicine or and many doctors who have taken on administrative functions often think the same way as the accountants and lawyers do with how to minimize risk rather than how to, optimize therapies for people, you know.

So on that note, my friend, I'm going to leave you. Yes, it's a pleasure. A pleasure to talk with you. Looking forward to, seeing you soon. I'm going to make sure I sign up for your, training.

I think you're going to do a short one for people. Was it June 4th for doctors? That's right. That's the next big event for surviving. Well, you can get information about attending Friday afternoon.

This was be, I agent had some really good things. Jimmy is and prevent present. Jeannie to present the, the speaker. We've got a whole host of other good, good speakers.

It'll be quick, it'll be factual. It'll be true. Data driven. Opinions. Sorry. You have data when it doesn't mean very much. It was. Thank you again. I really appreciate your your work.

I it's it's been something that has, you know, changed my life and the life of my patients. So thank you very much. And take care of your mother. Okay. Bye bye.