Introduction and Guest Background

Welcome to the Telomere Summit. I'm your host, Doctor Joseph Rafael. And today, I'm very pleased to have doctor Mark Rosenberg on to talk about his work in, really a new approach to oncology and treatment of cancer patients.

We're going to have a very interesting discussion about his, recent, work and, a new phase one trial that he's that has been initiated, that will be initiated in the first quarter of this year.

Welcome, Doctor Rosenberg. Thank you. Doctor Mark Rosenberg received his undergraduate degree from the University of Pennsylvania and is an output degree from Georgetown University School of Medicine. In 1988.

He completed his residency in emergency medicine, where he was awarded Resident and Teacher of the year. Doctor Rosenberg has been a director assistant director of several emergency departments, including Walter Reed Army Medical Center.

And approximately 17 years ago, after diagnosing his mother with metastatic lung cancer. Doctor Rosenberg began treating patients with advanced stage cancer and became a cancer researcher.

Doctor Rosenberg started a pharmaceutical company, which merged but merged with a spin out of Harvard, and a phase one trial will be initiated. As I mentioned in the first quarter of 2022, with a new drug that targets cancer stem cells.

Doctor Rosenberg recently filed a patent on a device, and I'm really interested to hear about that will decrease the ability of cancer cells to leave the circulation, thereby reducing metastatic seeding.

I'd like to just have you start, by talking about your journey into becoming going from emergency medicine into cancer, research and, and, cancer treatment.

Would you tell us a little about your journey? Sure. So, as you mentioned, my board certification, was emergency medicine. And, I was also a consultant, for poisons and overdoses.

Throughout Walter Army, Walter Reed Army Medical center. So whenever there was a toxicology emergency or or suspected overdose, I was consulted on the case.

I had written a book. And back, in residency, for the treatment, for novel treatments for, toxicology and overdoses. And, so, while I was working in the emergency department approximately 17 years ago, my mom walked in, complaining of chest pain and shortness of breath.

Plain chest X-rays showed a large, right upper low mass. We ended up doing a CT, chest, abdomen, pelvis, and I had the misfortune of diagnosing her with metastatic non-small cell lung cancer to liver, spleen, bilateral adrenal glands, and left hip.

Oh my God. At that time, I didn't know anything about treating cancer. I brought my mom up to Philadelphia. As you know, I, I went to Penn undergrad, and that's where they explained to me because they knew I was a doctor and they said, well, the truth is, conventional chemotherapy had extended survival, for advanced stage solid tumors by about two months over doing nothing.

Yeah. And I was appalled. I really couldn't believe that in all this time, with all of our advancements, in medicine, that's all we achieved. And so that's where my journey began.

Yeah. And, there's often stories like that that makes somebody switch their focus and and find a passion for

something. So what happened after that?

From Emergency Medicine to Cancer Care

Well, I was told, or we were told that my mom could do chemotherapy. Maybe she'd live six months. No chemotherapy, maybe four months, but who knows? So, I hit the internet and and, this is back in 2004.

So, yeah, 17 years ago. And I came across the name Ralph Moss, who was a famous journalist in, in, in cancer, in science writer. And he sent me about a 400 page dissertation on all the treatments available throughout the world.

Non-small cell lung cancer. So after going through all of that, I asked my mother what she was willing to do, and she said, I. V. vitamin C, nothing else.

And that was my first time dealing with intravenous vitamin C. My mom lived 11 months. Wow. And, And then what I started doing is, you know, my treatment has evolved.

So I consider myself a cancer researcher. Generally, spending, after I finish, office hours, it 3 to 4 hours a night researching the literature, and it goes on and on and on.

What a the best way to sum up what I do. I'm going to tell you what I tell most of my patients now when I have, consultation and I kid around and say I have no friends.

And the reason I say that is I don't like both sides. I don't like the fact that conventional, most conventional oncologist, do not want to step outside of the Nccn National Comprehensive Cancer Network guidelines, even if the treatment's not working.

You follow the rules, and if you want to leave the rules, even if it's not working, you either do a clinical trial or you go to hospice. And that's the way it's being practiced.

I don't like that. And I don't think they're receiving the best treatment they can. On the other hand, I'm going to be honest and tell you that I'm not happy with most of the alternative cancer doctors.

I've yet to meet one that is a cancer researcher or scientists. And they most of them that I've met are not aware of the data, and they don't seem they have their rules that they live by.

And and everybody gets hydrogen peroxide and UVB and ozone and they just do their thing. And so that's why I say I have no friends. So what I do is, I, I present to all the patients, all the different options with the available data.

And so I've kind of become well known around the world for using repurposed drugs, but also using conventional chemotherapy in a different fashion. So, I'll, for example, I'm a big fan of using poly chemotherapy.

So for example, if you use instead of two drugs, if you use four, 5 or 6, you can increase synergy. There's a lot of synergy. Now the conventional oncologist will say, well of course that makes sense, but you'll kill the patient with chemotherapy.

And my response will be, we'll lower the dose of each of the drugs and they'll say, no, no, no. The guidelines tell you, you don't lower the dose, right.

So I use chemotherapy and I'll do literature searches and and I will come up with very unique combinations. I will also use the, the immunotherapy drugs now except again often use lower dose and I use lower dose based on data.

Even though the the standard guidelines are no you use this high dose. But when you look at the data you have to say to yourself, why are we using a dose higher than what the data suggests is effective?

And and so in addition to that, I, I do, you know, I use supple mints, but I tell patients for most of the supplements I don't have data. So I, for example, we know there are a lot of supplements that may be helpful, like high dose curcumin, like melatonin.

But then you have to say to yourself, what is the effective dose? And then there again lies the problem. I have to tell patients I'm not saying there's not an effective dose, but nobody's going to fund a study on a supplement and lose all this money because it's very expensive to do a large clinical trial.

So we don't have that data, right. And probably the, the, the, the the other thing that, I would say that I started really over the past couple of years is I have aligned myself with an interventional radiologist.

And what we're doing together is injecting drugs such as the immunotherapy drugs directly into the tumors. And so when we combine all these, these modalities, and by the way, I also do use local hyperthermia, local, to potentiate a lot of what I'm doing.

And, and the device will penetrate 5 to 7cm deep. And it she's achieved temperatures of anywhere between 107 and 111°F. So but but again, that's only getting where I'm applying it, and and it's sparing the skin.

It's only getting deep to the tumor. So, you know, the the success has been remarkable. And I will tell you some interesting things.

Rethinking Oncology and Treatment Strategy

I treat a lot of patients from Sloan Kettering. I won't name drop, but, the chief of, of one of the divisions of Sloan Kettering has been sending me patients for 13 years.

And, I, I am, you know, another very interesting anecdote. There's a local, neuro oncologist who most of what he treats is glioblastoma, and glioblastoma has a median survival, with the best of standard of care of about 14 months.

And I get patients from him. And, I asked the last patient who came to me, and I said, how did this all go that you got referred to me from, you know, from the neuropsychologist and and he he's an electrical engineer and he's very well-educated.

And he, he so he went to in our oncologist and he said, look, I, I get it. I've read the data and I have an expected survival about 14 months. And the oncologist said, yeah.

And he said, but you know, we have the best equipment here. And we'll do everything we can. And the engineer said, I want to do better. And he said, okay, let me have you see Doctor Rosenberg and and he, he's wait a minute, I'm sorry.

You tell me that you offer the best, but you're going to send me to someone else because I want to do better. And that's where you got an explanation.

Well, Doctor Rosenberg has creative ideas. And he's very successful. But we are not allowed to step outside the guidelines. Yeah, it's crazy. And, and and I'm sure everybody realizes it's not that you're not allowed the the FDA, approves or disapproves drugs, but they don't make the protocols.

But but you understand, it's, you're working within a system. Yeah. And everybody's following the rules. So it's not that you're not allowed, but when you step outside the box, there's a price to pay for that.

Yeah. I'm sure you've been living it. But I think you're. From what I understand, your approach is born out of a different paradigm for what cancer is.

This whole idea of the cancer stem cell and treating the soil rather than the seed. Yeah. It's remarkable, remarkable

books written about chasing down that last cancer cell.

And you don't have to do that. You have to just make sure that metastasis doesn't occur, and that's what kills the patient. So tell us a little bit about that, that different paradigm, which I think others have written about.

But, you know, the Jerome Seed thing, but I found it fascinating and it makes a lot of sense. It probably informs your lower dose treatment and your multiple, there are whole modality treatment to, And then I'd love to hear some about the, you know, the, the actual diagnosis, test for the cancer stem for.

Sorry. Circulating the the circulating tumor cells. Yeah. So, yeah, you make an excellent point. You know, this the seed, and soil hypothesis. And basically, you know, you have cells that are circulating, and then they look for the appropriate soil and then they see.

And what we are aware of now is that each tumor microenvironment is unique and it's unique within each individual. So you can have two individuals with the same exact type of cancer.

And Gina, typically I mean it doesn't happen, but even if they're Gina typically identical, there's going to be unique environments in one individual versus another.

And of course, you know that, for example, this individual's gut microbiome looks like this. And that individual's gut microbiome looks like that. And they're going to have completely different immune responses.

So the microenvironment is unique to each individual but it's also unique within each organ. So for example the tumor microenvironment in the liver is different than the tumor microenvironment in bone.

And a great example I have a lady who's being seen at Johns Hopkins that I spoke with today, and she's been on a chemotherapy regimen. And her, her bone disease has been steadily decreasing on this regimen for tumor markers coming down.

And she feels great, but the liver lesions are growing. And that's not uncommon because the microenvironment in the liver is different. As a matter of fact, there's a lot of data.

And a lot of publications written on the resistance, the resistant milieu in the liver. And as a matter of fact, a recent article talked about the liver literally filtering out T cells, filtering out the immune cells so it can't do its job.

And so her oncologist at Johns Hopkins says, we'll give you one more month. If the liver lesions are still growing, we're we're going to change your chemotherapy.

And I said, well, that doesn't make a lot of sense to me. So you have a nice response in your bones to this treatment. But the liver lesions are grown.

So what I would do is I would continue that treatment. But we'll add liver directed therapy. So for example there are different techniques like it's a type of radiation called y 90 that goes right into the arterial supply.

And wherever that's supplying the part of the liver, it will kill that tumor. You can do chemo embolization right there. You can do cryo ablation. And I said it makes no sense to me to throw something out that's working when you could continue that and take care of the bones and get liver directed therapy.

And we can possibly chief need or no evidence of disease. And she said, I don't know why my oncologist didn't say that. And, you know, unfortunately, the thinking has gone from oncology and in medicine because we have algorithms to follow.

And, and, and it's very unfortunate. Now, it is FA is circulating tumor cells. I if you don't mind, I'm going to get into this whole conversation. So how does cancer go from being a focal disease to a systemic disease?

Well, the key is that cells break off from their primary location. And these cells, when they start to break off, very often, they become, you know, typically unique and then phenotypically unique.

And so as they start to change, it allows them to migrate. And we call that EMT or epithelial to mesenchymal transition transition. So they stop resembling the epithelial cells.

It like to stay home and be adhere to a basement membrane. And they act more like mesenchymal cells, traveling. And then these cells in Travis head into circulation and they are predestined to go to their new homes.

So, you know, cells are earmarked based on their liver. I'm sorry, based on their markers. So they're based on their markers and we're learning about that now.

So we know so some cells are predetermined to go to brain predetermine to go to liver predetermined to go to lungs. Now if you talk to an oncologist and say I want to look for circulating tumor cells, as I've learned from them, oh, that's a late finding.

That's you wouldn't look for it in stage three or stage two or stage one. We only see that in stage four. Well, that is incorrect. And there's a very interesting study that was performed where they took patients with colorectal cancer with metastases to the liver.

And the goal of this study was to find out when did those cells get to the liver. So they studied the lineages of those cells. And they found out that 80% of the cells in the liver arrived there before the primary colon lesion was large enough to see. Wow.

So it was an early phenomenon, but we couldn't see it on a scan. And to put it in perspective, it takes approximately 1 million cancer cells to form a one millimeter lesion.

So, you know, we we have you know, you may have 300,000 cells here, 750,000 cells there, a couple hundred thousand cells here. And we say there's no evidence of disease, but it's incorrect.

So it sounds like what you're saying is that metastasis occurs early on, in a lot of tumors. But then the immune system takes care of it or they, you know, they don't survive in their milieu.

Very good point. Sort of one of the ideas behind, you know, other physicians approaches to this where they want to change them a year. So this is I call us this is less, capable of being performed by the, by the cells, the tumor cells.

So, that that does change it all in its head. I mean, the cancer is metastasis, not a leap phenomenon.

Cancer Stem Cells and the New Drug Program

And you just have to accept that the cells are coming out. And so what does that mean for us? In terms of, you know, all the diagnostic tests that we have now to for early detection of cancer?

Sure. So, you know, we are heading, someone in the right direction. Now, I want to clear that, you know, not all cancers, have circulating tumor cells early, but the ones the the even the early stages, when they do, when they when we do find circulating tumor cells, they are very likely to recur and have metastatic disease.

But there are some cancers that are sitting at home. And it is a very late finding. But what we need to do is we need to be more proactive and find out, you know, who is actually sitting already.

So, for example, how many times has an oncologist, how many times have I seen what we think is stage two or stage three cancer. So you take a stage three cancer patient for rectal cancer.

And you go to you go to Memorial Sloan-Kettering and you get your six month you they respect the colon lesion. You get your six months of adjuvant chemotherapy and they say you're going to be fine.

And then they scan you and say sorry it's all over the place. Well you know if we were really proactive we should have known that probably much earlier.

So how do we start detecting that? Well, number one, there is a test that's been out for quite a while now called cell search. Now, it's not a great test, but it looks for circulating tumor cells that express what's called an antigen called EPP Cam or epithelial cell adhesion molecule.

Now, even though there's a lot of data showing that the number of circulating tumor cells is a much better prognostic factor with regards to survival than even a scan, even the imaging study and the tumor markers.

So why are our physicians not ordering that? It's simply not part of the Nccn guidelines. Now in addition to that we have a new diagnostic technique. And there's a company called Natera.

And they have a test called Signatera. And what they do is they access some of the patient's tissue from their cancer. And they sequenced the genome. So they know exactly what that genome looks like.

And then they isolate any circulating tumor DNA that's floating in the bloodstream that matches that genome. And then they quantify it and they'll say, we found ten molecules of circulating DNA.

And I apologize. My dog is barking in the background. Don't know if you hear that, but they'll say we saw ten molecules of circulating tumor DNA per ML of blood or we now we retest it.

It's up to 15. It's up to 20. That is turning out to be a very early finding. So for example again remember 1 million cancer cells makes a one millimeter tumor.

So we often will see circulating not only circulating tumor cells but circulating cell free tumor DNA going up in the blood before you see anything on scan.

Now, what would a conventional oncologist do with that? Nothing, because you have to wait for the scan. Right now, in my opinion, that's unacceptable.

Now what? I'm what the way I handle this, this will change in the future. But as you know, it takes a lot of money and a lot of years to change the guidelines. Yep.

So anyway, that's, you know, that's the direction we're headed. Now, can you, do you mind if I. If I break for a second, just get my wife to, stifle my dog because it's not that pesky factors that bother you.

Oh, no. If you're okay, it's not that bad. I don't think people are hearing it that much. Okay. Thank you. So, do you want me to get into my work? That my new research project?

Yeah, I mean, absolutely. That's the things started to move in that direction. I just want to help for the for the listeners that aren't as savvy about oncology.

You know what the cancer stem cell is and how it works, and it's not, you know, it's sort of different from the the vast majority of the tumor. Let me be a little bit about that and then move into your, your work from there.

Sure. So there's argument about whether the cancer stem cell exists, but there's no argument about this. You can call it whatever you want. It's all about nomenclature.

The bottom line is there are cells that have stem like, features. And these stem like, cells have the ability to self-renewal. So not all the cancer cells have the ability to self renew.

These cells do. And these cells are inherently resistant to our treatments, including radiation therapy and chemotherapy. So these cells are genetically unique.

And the problem one of the problems is we don't have a drug or treatment that targets the cancer stem cells. And as you can imagine, if we if we get rid of almost all the tumor or even everything we can see when you have stage four disease for almost every cancer, even if you get a patient to need, which means no evidence of disease, we all know that there's cancer still there because we don't know how to kill those stem cells.

And then with time, those stem cells will repopulate the tumor. So there there was a company that started called various stem where they wanted to target.

There are many companies trying to target cancer stem cells. And so what I, as you mentioned, what I did is I looked at a drug that, in vitro and animal studies showed to really target cancer stem cells.

And we ended up, merging with a spin out of Harvard that developed a novel nanoparticle technology that allowed us to put this drug in there and preferentially target cancer and preferentially target cancer stem cells.

So now that doesn't stop, of course, all the seeding from the blood stream. But to complement standard treatment, it it really should be a, you know, if not a home run, certainly, a double or triple.

And you can you say with the drug, is it in there? Well, so what we did. Yeah. When it now it only has a number. But we took the drug still in the mice and now it's a little mice.

And it's really a macrolide that is used as an antibiotic in chicken and pig feed to treat toxicity. Osis. And the problem with it is inadequate doses.

It's very neurotoxic. And so what we did is we figured out a way to get it right to using the nanoparticle, get it just to the cancer. So it's not neurotoxic to the entire system.

And it allowed us to lower the dose very significantly while getting great results. So, you know, in our mouse studies, at, at Harvard, we, we treated ovarian, metastatic ovarian, breast, prostate, pancreatic, lung sarcomas, glioblastoma.

The results were phenomenal on every single model we studied. Wow. That's sounds like it could be in your. You're having a the trial is this is a trial is coming up in the first quarter of 2022.

Phase one. Correct. Yeah. Should be, could be potentially groundbreaking. Yeah. I I'm we're certainly all very excited about that. And, you have a device that also, can do.

Yeah. Over the past year and a half. I, you know, I, I've been for many years thinking about, you know, this the concept of circulating tumor cells and, to explain a little more of that.

Circulating Tumor Cells and Metastatic Seeding

And you would actually hit on this, the individual circulating tumor cells very often floating in the bloodstream, they'll get they can they can succumb to the hydrostatic forces, of the blood flow and often get killed.

The immune system may kill it. So most of the circulating individual tumor cells don't make it. On the other hand, the clusters are what causes this problem.

And what's been demonstrated is that clustering doesn't happen in the circulation. It happens from the primary tissue where some of the cells will leave as a cluster.

Now it turns out that. So how does that happen? How does that happen? A large cluster of cells leaves because it's hard enough for one cell to, you know, enter in travel seat.

Is there some other. I mean, how big are these clusters? Well, so by definition, a cluster is a minimum of two cells. Okay. Now you can have 15, 20, 30 cells.

You know, to give you an idea of size, one cell would probably average around 15 microns. So you're talking at least 30 microns in size, but very often you'll have 50, 70, 500 microns.

So now and you say how does this happen? The amount of crosstalk, the amount of communication that goes on between the cancer cells and our own cells is mind boggling.

So they come from us and they literally instruct our own cells to get help. And I don't want to waste people's time talk for, you know, I'll just give you one example.

There's constant crosstalk between these tumor cells and platelets, and they will exchange exosomes and RNA, and they will co-opt the platelets to work for them.

And for example, there's a site on platelets called a silicon site, where cancer cells can ride on their backs and travel around. Wow. There's so much communication.

And and again, we look at it as the enemy. But the problem is the enemy arises from within us, right? They speak and so yeah, that's that's perfect. They speak the same language.

And so yeah. Go ahead I'm sorry. No are going to talk about the device unless it's some kind of a filter or something or. Well so the clusters. Now when I talk about clusters, it's not only the cancer, the tumor clusters, but neutrophil clusters.

So if you were to Google neutrophil and lymphocyte ratio and cancer progression, you see as cancer progresses neutrophils goes up and lymphocytes go down. The ratio goes up.

When you have neutrophil clusters they they will follow the tumor clusters the tumor cell clusters and help them. Very often we'll see a combination cluster of tumor cell cluster with neutrophil clusters.

And those neutrophils will protect them and accompany them to their new homes and help them set up shop. Now there are also many other types of clusters.

There are macrophage clusters. We we call them, you know, cancer associated macrophage like cells. And they form large clusters. There are cancer associated fibroblast clusters and they're important for nourishing.

There are approximately eight significant clusters that have been identified. And they all help set up shop and build the milieu or environment that the cancer needs.

So I, you know, I came up with a concept that if I could eliminate these clusters, this could be a game changer. Well, it turns out that people have looked at filtering some cells off.

Actually not even clusters, but individual cells. It's been very difficult. It's been very difficult. And so, what I decided to do, after drawing many, many, many different diagrams, is to develop a filtration device that you can hook your arterial supply up to this filtration device, and we will break up every cluster.

And and it's complicated to filter and I'm not going to get into the details. And actually they don't want me to get into the details. Right. But the bottom line is I will make sure that everything that leaves that filter when it now that there's an inlet, an outlet.

So arterial supply will feed into this filter, the filter will eliminate the clustering of everything. And now only individual cells can return to circulation which are are harmless.

And that is basically, you know, the concept behind it. Now to support this, this concept, there was one mouse study that was done. And what they did is they didn't look at clusters, but they injected green fluorescent protein into circulating tumor cells and they injected rose Bengal.

And when you shine a 430, actually, I don't remember the number. I better back on that, but it's a it's a specific laser with a specific frequency. It will activate the green fluorescent protein with the rose Bengal stain.

So it causes massive oxidative stress to the circulating tumor cells, and they'll die. And when they did that, the mice live so much longer and all, much less metastasis.

So that is the first proof of concept in an animal. If you can eliminate circulating tumor cells, well then the patient will live a lot longer. And so to extrapolate that further think about this concept.

If I no longer allow clusters to leave circulation, then there's no more seeding. In essence, what we'll do is we'll turn a systemic disease into a focal disease because it can't see it anymore.

So now your chemotherapy and immunotherapy and radiation and surgical resection works better because there's no longer sitting. So what I ended up doing is I filed a patent about two months ago, a provisional patent about two months ago.

We just brought on, an engineering team. And one of the groups, is that has a lot of experience in circulating tumor cells is at University of Michigan.

But they've been only on the diagnostic side. They they've been looking at isolating circulating tumor cells. So we can come up with a drug to treat it better.

So they got very excited when we they heard what I wanted to do. And so as a matter of fact, I have to send the NDA back today. And we have an agreement, any IP that we develop on the diagnostic side, we'll give it to them.

Any IP we develop on the therapeutic side comes to me. And so what we're going to do is we're going to start building this prototype device. And we're going to take animal blood with many clusters in the lab at University of Michigan.

They have all the equipment we need to measure these clusters before and after. So we'll measure the clusters before. We'll run it through the filter and we'll measure them after.

And there's a lot of tweaking until we can say okay, after one pass or three passes or five passes, some clusters are gone. And so that is the basic concept.

And the idea is that going ahead in the future, certainly you can have your blood cleaned as often as you need. You know, we may say, you know, well, we'll measure, you know, once a month.

Well, we'll measure circulating tumor clusters and we'll say, you know, you're good. This month, next month. Come in. You know what we found? Two clusters per per, you know, 7.5ml of blood.

We need to clean your blood again. And so we're extremely excited about that. And that's version 1.0. Now I will tell you honestly what happens, you know cost a lot of money to bring this into practice.

Likely a large device company will buy us out once we, you know, we'll publish the first animal study. But there'll be milestones. In other words, you can't show this if that company doesn't meet milestones.

It comes back to me. Version 2.0. You shrink this device down, insert it in the inferior vena cava, superior vena cava with a chip and an app to your phone so you can conceive, receive feedback.

You look at your phone and say, okay, let's see. This month I've captured 30 clusters and it's time to change the filter in two weeks. So this this is this is very doable.

Now, this is all conceived by my mind, but I really don't have to develop new science.

Cluster Filtering Device and Future Directions

I can use everything we have to make this happen. So it's like home cancer dialysis. Yeah. Let's. It's funny you said that. Nothing is an accident. Because the company we formed one of the the, one of the people we brought on wanted to call this cancer dialysis.

I nixed that idea, but you're right. That's what we're talking about. Well, I mean, it's, you know, it's kind of like a farriss kind of thing, but, you know, so. And dialysis has bad connotations to it.

So that's why you want to nix it. Because that's basically a life's a short life sentence. You know, but yeah, it's a very, you know, interesting, interesting concept.

So you can in fact, I mean, this kind of brings us to the larger question of this whole idea of curing cancer. It sounds more like what we're talking about is managing it as a chronic disease.

That doesn't kill you. You know, and, you know, maybe the certainly you can have cures and we have successes like that and Gleevec etc., but, but if you can keep the person alive, just like with HIV, you know, there's no evidence of cluster circulating, no evidence of viral load.

It is that success? I mean, exactly, exactly. Now, again, I think it is possible to this could lead to curing because if, if if it's no longer able to seed and then you can wipe everything out.

You know, we'll now add a, a, a drug that targets cancer stem cells and we wipe everything out. We can see, and there's no longer seeding going on. You know, then you use like, I also use a repurposed drug called ammonium tetra from elimidate, which is a drug that lowers copper and prevents angiogenesis.

So I think, you know, initially this could lead, of course, to managing this as a chronic disease indefinitely, but it really can lead to a cure. That's really, that's fascinating.

I mean, we've been trying to have that cure for cancer for a long time. We got to the moon before then, which most people wouldn't have predicted. It's a pretty complicated.

I think our paradigm has been has been wrong for a long time. And and you're right about the, I mean, I understand why the the guidelines are in there.

Because the studies are done. You have to have a large enough study to get the, the, significant results and then you stick by it. But I'm always very kind of disappointed when patients come back to me and they're like, well, you know, they want to do this and, you know, breast cancer or prostate cancer.

And it really is not tailored to the individual situation. And what we're learning is that cancer is a unique disease in each person, not only because of the genetics of the tumor, but because of the soil of the person.

You're exactly right. And that's, I think, important. And I also tell patients that and this is kind of interesting for me, which I don't do any cancer therapy in my practice, obviously, patients are always worried and their doctors are always worried.

You know, colleges are always worried about, you know, to prevent cancer at all costs, you know, and the truth is, is that cancer and aging are two sides of the same coin.

Your ability to regenerate tissues is what happens. You know, that's the seed for cancer potentially. Sure. But but maintaining optimal performance means, no, not completely shutting down.

And for, you know, because you don't have a growth phase with a worse. Yeah. And so if we can, you know, allow for therapies like, for instance, you know, I don't think hormone replacement therapy causes cancer, but, that was a big worry.

But, you know, estrogen is an important molecule. So we have therapies for allowing patients to have the benefits of those and then being able to deal with it when it becomes a problem that that then lets us have longer and very, you know, healthy lives.

I mean, somebody who sees patients with androgen deprivation, I know you use that sometimes in metastatic prostate cancer, but it does a number on the rest of the body.

Right. And they often and you may even be aware and this is it's getting more press even at Johns Hopkins. So, for example, there's something called bipolar androgen therapy.

And what they do is patients who have metastatic prostate cancer who have failed all the guidelines. Then what they do is they slam them with huge amounts of testosterone every month.

And and I won't tell you. I mean, I can get into my theory on how it works, but very often cancer starts receding. Probably, I think so maybe it's a sort of step because it's gets a rubberized into estrogen and then there's an extra dial.

You know, that I think I would call it that could potentially stop it, slow it down. Well, you know, that that's been shown not to be the mechanism, but you're right as a matter of fact.

So in, in a, in a, in a third world country instead of using the powerful second generation, androgen antagonists that we use now, they still use these diethyl still best strong.

And so you're absolutely right. But very likely the way the high dose testosterone works is what you end up doing is as cancer, progresses and, and and the prostate cancer becomes what we call castrate resistant, meaning it doesn't respond to androgen deprivation anymore.

But you still have a few cells that still respond to testosterone, but they're there now. They're overwhelmed by the resistant cells that don't care. And in my opinion, you know, how do I think giving testosterone is working?

I think you start recruiting back those cells that are more sensitive and and less aggressive and so by giving testosterone, you can recruit the, the relatively benign cells as opposed to the resistant cells.

And they do the same thing with estrogen. So with breast cancer same concept patients. And this is old medicine. But when patients have metastatic breast cancer is nothing working.

You give them a lot of estrogen and a portion of those patients will start to regress. Yeah. So maybe they're getting a selective advantage because they're getting they can respond to the estrogen and how compete.

And interestingly along those lines with both breast and prostate when you give them all the testosterone and it works for a while and then it stops working, what do you do.

You block the testosterone and it works again. Yeah. And you can go back and forth and back and forth. Very interesting. Well, it's been a fascinating discussion.

Mean and this whole area of medicine, I think is, is really changing it in, you know, in a way that makes more sense biologically and physiologically.

It's exciting work that you do. Any, closing words? Well, you know, I just, I'm you and I have been doing we've been in medicine for a long time, and, you know, I, I think it's your role and my role to try to make positive changes.

I think probably many of us are not happy about the direction, and I'm not, I'm going to go past cancer now. I think many of us are not happy with the direction that, medicine is going in.

And I get it, you know, the the pharmaceutical industry is really writing medicine, and it's writing the algorithms for

medicine, and and I'm on both sides now.

I see, I get it. But what's really sad is that, the thinking part is kind of going away. So, for example, doctor DeVita, wrote a book, on, on cancer. And he was in the 70s.

He was the chief of oncology at Yale and, and chief of the NCI. And in his book, he talks about perhaps the biggest obstacle to curing cancer. Our oncologists, ourselves.

And, it's, you know, we're we're hoping and it's your role and my role to to help, spur this movement on, to bring thinking back and not simply follow algorithms.

And so my parting words would be to encourage everybody to, to use what you've been given and to think. And when a rule doesn't make sense, you know, it's and it's scary for someone like me to say, I'm not going to follow the rules, but we owe it to our patients because that's why we became doctors.

It's fantastic. Parting words. It's, very sage advice. I mean, it doesn't make sense. Then it doesn't make sense. And you gotta you gotta let it go. Been a pleasure talking to you.

Look forward to maybe seeing at the next meeting. Or, if you're ever in New York City, we'll drop by. Excellent. Thank you so much, Joe. I appreciate the time.