Introduction and Guest Background

Hello, everyone. Welcome once again to the Prostate Cancer Summit. I'm your host, Doctor Geo Espinosa, and I am so excited with my next guest. And I know, I know what you're thinking.

Well, Doctor Geo, you say that all the time. No no no no no, this is this is real. This is real. Doctor Sanjay Juneja Doctor Juneja is a triple board certified hematologist and medical oncologist.

He's internationally known and keynote speaker at the largest followed oncologist globally for his social and news media education at doctor or the ONC, doc at doc @theoncdoc.

You want to follow him if you have any interest in really getting it right. I got to know Sanjay prior to the recording, and when you hit it off after a certain point, you, you, you know, you're hitting it off, and we did.

And this guy is, integrity. He wants to change the world in medicine, in oncology, and I think he will. So you want to follow him @theoncdoc? He's also the host of his own Target Cancer podcast, spanning 2 million downloads in over 110 countries and is the, an editorial board, member of the peer reviewed journal.

I the precision of him oncology Sanjay, first of all, how do you have time for all that? It sounds like your resume sounds so packed. I'm like, okay, dude, what's your self preservation situation like?

Do you take care of yourself? I would think you don't even sleep. Sanjay, thank you for being on. And, Yeah, let's let's start with that. Let's start with how do you get to do all that?

Well, one, thank you for having me. Very humbled and honored. And and two, it's, you know, I think the world just equips you with things that you look at in two ways.

It's like, do I want to do this? And I got this and I'm lucky, or it's like kind of a hot potato responsibility. Like what? What is the purpose of this? Right.

And and I kind of pride myself on being, you know, considered a layperson of the medical degree in a very challenging field of oncology, very scary field, very misunderstood.

And it's really passion based as far as your, you know, self care question. And I'm actively working on that to try and make that a thing. Right now it's a very little sleep 4 to 5 hours.

But but people need it. And I just feel, you know, like all of us responsible not even to change the world ourselves and be a part of a change that's necessary.

And I'm, you know, just humble with people that are listening like this, that, that are so proactive and want to know the things and want to have the best care and are probably sharing it with people.

Like what I mean, that replaces sleep to me for now, but sleep is one of the most important things. Even relating to cancer, so I need to fix that ASAP.

I can tell you've been listening to a few podcasts on sleep. There you go. That's it. You know you almost when you listen to so many podcast, listen to a few.

And I have one myself and you almost it's almost like, I like, for example, I tell people, well, you know, get an ordering, right? Get an ordering to measure your sleep.

Well, you know, what I learned the hard way is maybe you shouldn't get an ordering. It's just too stressful. Every morning you wake up. Oh, my God, my score is so low.

Okay, I'm going to try again next time. And you kind of fighting to try to sleep better. Same thing with listening to many podcasts, right. It's like it's just so much information is like oh my God, you know, am I getting this right?

Am I is it not enough time in a day? And it's that little passion, right?

Personal Motivation and Work Ethic

We were talking before we started recording, I think. Passion. Is the energy source, right? How much sleep do you really need? I know that you've. You asked, experts.

Walker. Wright. Walker. Why we sleep, author things like that. I'm like, I can't listen to this guy, but there's no way I'm ever going to get eight hours.

It's just not happening. But passion, man, is something about passion and trying to make a difference in the world that that I think there's a healing element to that it's not I don't go to work, I don't work, I live my passion.

Monday, I love Monday mornings. I love Monday mornings. I love going on vacation. I'm in Costa Rica last week and you know, I'm by the beach and you know, my wife is reading her magazine.

I'm reading journal papers. Yeah. And I and I love it. Right. So you, you and I have kindred spirits in that way. And we, we got to know each other. And I really appreciate everything you do.

Oh, likewise. You everyone people listening. It's we're all we're all, you know, intertwined in a web of electrons and protons. And the more synergy we have, I think the obviously the bigger waves we make.

Absolutely. I have to I have to ask another personal question. You live in Baton Rouge, Louisiana. Okay. I, I mean, I'm surprised I don't see in your background only purple and yellow, right?

I now know, like, what? Right. Somewhere here, it's like there's a signed helmet from, you know. Oh, there you go. Oh, you can't see it in the corner.

But but yeah, huge fan and, very, you know, a lot of pride. It's. Yeah. Everything around you that made you who you are. So it's really hard to, you know, hard to leave when when the investments been made.

Now you got to give them their ROI. Great school. You undergrad, medical school, residency. Good stuff, good stuff. I love that, Sanjay, I think your main focus, I love your main focus, which is where medicine is going.

Where medicine should go is personalization. Right? Like I always tell people, look, I could see ten prostate cancer patients. They're all different. Not the same.

And the protocol is not the same. And I would I would even argue that the natural protocol as well is not the same, you know, age, interest. What's important to you.

Right. That matters. And we could talk about histology for that cancer. We can talk about the biology of your particular cancer. So give us a general overview of your views on personalizing medicine.

Where are we now as a whole? Let's just say in oncology as a whole. And where is it going. Yeah. So this is a difficult thing and I don't mean to understand, but more so to figure out how we can make this change.

Because the way we traditionally for decades guide therapies for cancer diagnosis is basically large buckets of tubes, buckets of this is what the, you know, cohort is the fancy term for the patients in the study.

And we only knew what we knew when we did the studies. Right. So when you said histo Pat, that means when you take a biopsy of a tissue, I considered a macro thing, meaning, yes, you need a microscope, but you're just looking at the tissue, you're looking at the actual cells, the cancer.

And so we classify everything this way. Adenocarcinoma. Right. Which is mostly prostate prostate usually then squamous. And these couple of receptors that we stain which are proteins and everything's based on that still like chemo and and everything is what you're seeing under under a microscope.

But then along the way, we all of a sudden discovered molecular testing or next generation sequencing. And now when you're talking about those things, we did not have them on these bucket studies that we have.

And they go two ways. They go one way in the tumor. So what molecular is, is not the the house that is built. It's the blueprint for the house. And we realize that there's a lot of things in the blueprint that may not be appreciated when you're just looking at the house.

Of course, an architect is listening to this and saying, of course not, give no idea the data. Right. But but now we have all these properties on a whole different level that we're somehow trying to integrate and work in to these buckets.

The other part of that is the same blueprints, otherwise known as DNA and RNA and all the all the things that make the receptors that that make, you know, ultimately downstream PSA and all that stuff.

We also know it on the person. So we know what faculties are working, not working and how they play a role. Like I can look at stairs in a house, but I know that there was calculations and some stability factors that if I just think I can take out one ball because it's visually not great, the stairs can feel like it's a whole new wealth of knowledge that we're trying to integrate. So when you get your guidance, what happens?

You're usually like the biopsy showed this molecular is usually not brought up. And we're talking about PSA. And those are all the best we had at the time.

Personalized Oncology and Molecular Testing

When you talked about personalized and molecular therapy and precision therapy. Now we said, okay we have all these things. Let's start talking about them.

Like you had mentioned earlier, lifestyle matters. What you do matters. You know how you much you sit or don't set matters if you play golf or not. If you're flying all these, all those are also personalized factors.

But let's just bring it distill it down real, real simply and that's we're talking about the molecular properties and the features of your specific tumor.

And then also of your, you know, genetics, not just what you're born with but what you currently stand with. Right. And then that's what you're talking about when you talk about Brac, and Parp and targeted therapy, you know, you're talking about these molecular properties that actually go into the coating.

Now, another reason that we have difficulty and you may say, well, I just get this decision and it just seems we're kind of, you know, putting our finger in the air and saying, which way the wind blows is because the best we had when we talk about Gleason's, you're again looking just at the properties of the tissue itself.

And you don't know for sure the Gleason score helps, your age helps. But there's all these other factors, which is why we don't know, because they're all grouped together and they weren't partitioned out.

This is where and we can talk about it later. I think artificial intelligence will be humongous in helping us have more kind of precise choices based on a better sample of the person like you or like your tumor.

Now as a whole. That's where we're at, and I think that's where and why. There's a lot of confusion and debate and gray area. But as we start introducing, closer and closer and closer upfront, then we'll be able to really have this kind of targeted concept, especially when it relates to we have something valuable here.

It's a PSA like there's no other tissues that make this prostate specific antigen pick up on a Psma scan. Sure. But but it's very fortunate. You know, people obsess over Sears for colon cancer and CA one 2599 for pancreatic and ovarian.

Nothing is so precise as prostate. But the way we're treating it, we're not actually honing in on the cancer cell itself. We're depriving what makes it grow, even though we know that eventually it won't care and can grow despite not being given food on the plate.

It just gets up off the table and goes, fine, I don't know. So there's a lot to be done. I think, in treating prostate cancer, and I appreciate that, your response so much.

From a, macro perspective, I'm just super interested. We're definitely going to hone in a lot on prostate cancer, but I'm just in. Do you think that personalization and artificial intelligence at some point will be able to detect, cancers, that tend to be aggressive when they're found earlier?

So pancreatic or, you know, stage four colon or. Yeah. You know, I think colonoscopies are great, actually, and they do a good job. But, in terms of finding things earlier, but, you know, pancreatic cancer comes up, right?

Lung cancer, are we going to be able to, your best guess to get find these things early and perhaps take action? Absolutely. So, you know, there was a hallmark thing that happened in the last month, and that's that we actually have a screening test for as early debatably as stage one, not debatably.

It says stage one, but but but stage two is and stage three, which are all generally mean and curative approach. We have a blood test for that you know and that's garden.

Garden came out with it. Now an FDA thing where you can actually check your blood for cancer. Now, I know you're thinking and like, yo, you just said you're checking your blood.

That means the tumor cells in the blood. That ain't one, two, three, I think curable. We're not checking for the tumor, but we're checking for is the sweat or the must, or the hair and skin cells.

It doesn't mean, you know that that the humans there, if you go in a house and you see dust with hair and skin cells, it means that the human is somewhere.

And that is called circulating tumor DNA. And all of our cells live and die, right? They grow. And the older ones die, and the new ones live, and it's usually pretty fast.

So what you're checking for in these blood tests is as those cells die, are we can we pick up on those exact blueprints that I was talking about, like you say, like, oh, whoa.

You know, they had some coding for this, like somewhere. And this is not coding that you do for normal cells. And then that's what that blood test is.

That's one way. And the more you mention the company Giardia is that was guardant is the one that got approved for for a colorectal grail slash gallery.

They've been doing it for that kind of shotgun approach. That's right. For 50 plus, you know, tumors, they're looking at all all the DNA stuff that we know has to do with the coding for unregulated cell growth.

That's what cancer means. That's what stands for unregulated cell growth usually happens in one of two ways. It's usually the on switch is stuck. You're like, okay dude, we're going to turn it off or not.

Oh no, it's not, it's not turning off. And it just keeps growing. Right. And so that's unregulated cell growth. And then also the stop signs, the blow up signs, the ones that says you know, this looks a little sketchy or we've we've we've done what we need it.

When you look at your nail, how does your nail and your cuticle know to stop there? Why isn't our cuticle going all the way back to our knuckles? Everything's in order.

Unordered, unregulated cell growth so it doesn't blow up. And that's usually a Tp53 thing that has to do with with with kind of saying something about sketchy.

Remember your cancer cells or a colony of cells that have properties different to their everything else around them. They've gone rogue. It's you know, I hate to I don't mean to be insensitive, but talking about, you know, cults and things like that, like they're they're around you, but they're but it doesn't mean the people that are around are just like the same thing.

Right. And that cancer cell is a column, the constellation of things. And that's why we, you know, traditionally want to remove it. We want to say, take out that bad player. Yeah.

Or that area. That's that's the I'm not talking about cults now. I'm talking about cancer. Oh. And we just remove it all, but hopefully everything around is removed of this.

That's why you do a lumpectomy with breast cancer. That's why with the prostate cancer, you know, in general, if it's aggressive, you take it out. You're like, I just hope to get it all.

And that's the overall concept of cancer in cancer management. Take out the bad colon. You take out the polyp on a smear, take out the colony on the cervix that has, you know, potentially abnormal behaviors.

It looks like it's being sketchy. Now, I had a gentleman on my podcast that blew my mind, Michael Levin. And he almost and I don't mean this insensitively at all.

He almost made you sympathize a little bit with cancer cells on a biologic level. I don't mean this instead of balls or close people cancer, but that colony is just doing what our normal cells and evolution has done to make us survive today, which is it does everything it can to grow.

It doesn't feel connected with its environment. And so it starts to have it equip all of these things to continue to grow and foster. That's why treatments fail.

That's why that eventually they stop working. That's why, you know, its properties are so different because it just wants to grow, which is exactly how, you know, any species has evolved.

And so you're trying to battle something that has all of the properties and tools and weapons that have ever been, you know, surmised and needed for a humans to be humans today.

And you're trying to battle it and it has similar properties to your own cells. So that's why you have this concept of chemo and poison, because you're like, yes, it's a poison to you to some degree in your normal cells.

But because it replicates fast, a lot of chemo is a work on the replication cycle. They screwed up so bad that your body's like, yo, this thing is sketchy.

This will never work. If an architect, you know, in undergrad or graduate school, first year ends, that thing does. And this will not stand then. Then they scrap it, you know, the blueprint and then have to make a new one.

That's what chemo does. It screws up the DNA. That's what radiation does. Screws it up, makes it it makes it not stand. That is the concept behind so far.

The best we can do to treating, you know, cancers is either make your body decide to destroy it, which is what we're doing with immune therapy kind of opening their eyes, taking off the stop sign off of tumors, which is a big strategy. PDL one CPS.

These are ways that they use to stop signs that are good. Cells use to just say, oh, nothing to see here. So and all of a sudden, you know, you take it off, you block it and their immune system can kill it with, you know, little when it comes to like taking away the hormone androgen deprivation therapy and the pills you take, you're just like, not letting it, you know, fester as much and then temporarily as a two year window, roughly where the antibiotic resistant right ADT.

Right. Radiation therapy is very. So you know what. Which leads to the next point probably which is like these suckers are actually smart. They're trying to be resist.

It's almost like bacteria that is resistant to antibiotics. They trying to figure a different way out to to survive in your body. And you try one thing and it's not working, and then you try.

The next thing is like, what's happening here? ADT is a perfect example of that is only like a two year window before, you become resistant to it. Now you got to do the next thing.

So yeah. Yeah. So there's two ways to think about it because I said that. And then Siddhartha mukherjee, Pulitzer Prize winner, wrote Number of All Maladies.

We are we are so connected I, I literally in my head I wrote it myself, I know it. I gotta bring up Siddhartha mukherjee because when you started talking about the environment around it, you know, I was going to make the point that, you know, a big part of what I do is treat the micro environment, which the author talks about extensively.

Yeah. And I and you brought him up. I, I just feel like the root of it is this guy. Are you are you are your mind reader as well. No it's chemistry. So that's why.

So when I said that said was on my podcast, he just called me out. He's like, they're not smart at all. Whatever, Michael Levin, other people disagree.

It depends how you define smart. They don't see something and say, yo, let's change something and then and have a way to escape this. That's not necessarily in that sense what's happening, but to your point and what Mike 11 and others would say is there are things that are happening in the environment around the destruction of cells that do signal evolution or variation or adaptations.

Obviously, cells adapt. So whether that adaption is considered smart or or you consider it, you know, preprogramed to adapt. I think it's a matter of semantics.

But yes, it is. It is a it is adaptive cancer is. And that's why, you know, I think multimodal therapy or combination therapy is a thing and will be the biggest thing in the future.

We used to have this whole thing. We stack our treatments. We got 3 or 4 lines, one at a time. Why would we use two at the same time? We're getting used to both of them and then we want to have something later.

But then we realize, whoa, wait. But it kind of makes people more people actually potentially get a cure or it delays much further together. Not just additive, but synergistic on how long before you have to change something.

Early Detection, ctDNA, and Cancer Evolution

So now we're like, maybe combination works. Why? Because you're going with different strategies at the same time to hack away the most you can. The thought behind this is the one I think is very important to understand.

If anyone has said it's sketchy at about traditional medicine because when I had cancer all over my body or my loved one, they said they couldn't see anything.

Well, after they did the usually combination therapy and then all of a sudden something comes back in one place. How did that happen? Right? It's the same way that with prostate cancer, if you have a spot in your long, they came back 4 or 5 years later.

It didn't just spontaneously get created. What happened was 99.99% of the tumor was sensitive to whatever you gave upfront, but there was a colony that did not die that that from that moment did not die.

But you need about 200 million cells to see it on an average Cat scan. So if you have 100,000, 10,000, 1000, a million, 5 million, it's clear, the scans are clear.

And it's probably not enough to make PSA a circulating tumor. DNA. And what matters is the doubling time. And in general, prostate cancer takes a long time to double like a replicate compared to small cell cancer.

Small cell lung cancer, pancreatic cancer, things that replicate very fast. So you it's this time frame before you see a recurrence, either as in the PSA or as a spot or in any cancer that it just now got big enough to appreciate and see again.

But the reason we do multimodal is you want to just knock out as fast as you can the amount that later starts to evolve and like, grow and evolve the same way.

Covid is alpha, the zeta. There's different versions because as it replicates, you get little changes in your DNA and they all look a little different.

But in the same vein, cancer is the same way. So the further you are along in your cancer process, when you become cancer, you know, castrate resistant, or in the third or fourth line, you have areas that respond in areas that don't because now they're different, now they're colonized.

Your behavior in New York is different than us in Louisiana, right? We're all Americans, but we're all a little different. If you decide to poison Cajun seasoning, you don't necessarily knock out the people in Illinois or Chicago or, you know, boring food places.

Actually, Chicago is great. I'm joking, but you. But but but Louisiana is going to get hurt. Whereas if everyone's more uniform, you try to you try to hurt it all upfront.

And that's why we're bringing all the things forward and doing multi, either multimodal or multiple things at once. Bring immune therapy in with chemo.

Let's fight and kill everything with 2 or 3 different you know mechanisms and chemo and liberate the immune system to recognize as these cells die and disclose their contents like oh they wear red ties.

Now the immune system because we're giving immune therapy, recognize the red ties and make all the things better because you're killing it with chemo.

Also, rather than just gambling on the on the immune therapy hoping it sees it, let's kill it with chemo and immediately say no. That's why you do radiation and chemo together when it comes to, like, lung cancer.

Right? We doing concurrently? Some GI cancers. That's where we're going. Try to get as much of it as you can. And that's why I want a 2%, 3% of the time of stage four is cured.

Like, truly cured. Because you got lucky there wasn't a bob. No colony was able to outsmart the multiple therapies you did upfront. And this concept is very important and also leads to precision and personalized medicine.

That's why molecular testing has gone from stage four to now. Stage 3 or 2. And now to like early and early earlier because we want to discover and uncover all of the different factors for what we have therapy for.

And when you apply AI, you can partition out everything that may matter, like just a statin, you know, to give us that in matter or not in prostate cancer, you know, a bit of debate does aspirin matter?

Doesn't metformin matter? But again, we can't have studies for everything. But what you can do is have machine learning and crazy neuronal networks to be able to process and just pick up patterns.

AI is pattern recognition. Yeah. I don't know why when there was that in aspirin. Their survivals are actually twice as big or time before getting cancer resistant than others.

Like they could tell us all these things and until then, that's why we have a lot of these whys or maybes or what should we do? Because yes, the Gleason tells you the behavior.

How aggressive. But over time it can evolve. And maybe the Gleason does get up because now as it gets old, you know, replicates and gets more sinister and gets the mutations, it's starting to behave more ugly.

If that's a word. I like adverbs. So you want to knock out things as fast as you can. The lift is also considered. Obviously if you could knock out a prostate cancer and not have the side effects, potential side effects of surgery and radiation, everyone would do it after Gleason, right?

It's like I get cancer, do it. But the reason you don't is because what is the lift? That's why we're less. That's why we're overcautious. On taking out polyps.

That looks sketchy in the colon. What's the lift? You clipping it off? Lumpectomy. What's the lift you got? You know, your breast tissue and then, you know, there's some implications, but you take out anything that's inside you, right?

Right. Skin lesions. Pre-cancer. It's all about lift. Trust me. If if something was invasive cancer and the lift wasn't debilitating, the debate becomes less.

And that's why I think it's also going to help as the tools evolve and they get better and the lifts get lower, the threshold gets lower on untreated invasive cancer.

But we will only appreciate that when we know that stuff. And that's why behavior matters. Gleason is a behavior property. It is using our retrospective data to say, chances are this is not going to be a problem.

The next one is if it behaves the way we studied it, but who knows, it could take a turn. It could turn into small cell or high grade neuroendocrine, right?

That's a real thing. Prostate cancers get a high grade in your indica tumors. That's a whole different thing. That's a whole different dynamic. That's a whole different prostate cancer.

That's evolution night and day. Right. Exactly. And that's the point. Tumor evolved. That's why the PSA stops mattering. I don't get it. The doctor said the PSA is better, but my cancer is spreading like wildfire. Why?

Because it's not doing PSA, bro. It's not sweating it anymore. It's turned into this aggressive thing. So we know tumors evolve. And I hope that could help people appreciate why it's complex, why we change therapies.

I think one day this is my opinion. Just keeping something on and on and on and on, like when you have a great response, is it really helping? Because if there's something that's not listening, is it like just got to declare itself later and now you have unnecessary toxicity?

Like we can figure out, we got to figure all this out. And the way we do it is pattern recognition. Full time job 20 47I in what takes us 200,000 years.

I believe it was a number or 200. I don't know, for one person to study everything that's on the internet. I, you know, can do in minutes or a day like that's that's the processing speed of understanding and recall.

Of course we want that for cancers. Of course we want that for what constellation meds they on. Are they Cuban. Are they Indian. What are their genetics.

What is the tumor. Gleason. And then have a better idea rather than using this ultimately what 12 numbers are? Gleason I mean, think that's not how we're that's how we're partitioning. Is these like big just by a number right now and not factoring these other things?

You know what a al he's you know, that patient is Cuban. Maybe all he needs is more rice and beans and he's. That's exactly what's right. More beans. Maybe, you know, who knows? Who knows?

Really? Who knows? You know, I say that with with with you say who knows with rice is a very interesting phenomenon. I, I've been doing diet work for over 25 years.

Read it like I probably every book out there. And, looking at the research and, you know, the thing with rice. Right. Well, you know, stay away from white rice, white rice, brown rice, brown rice.

So it was most of my life is like, okay, I got to do I'm doing brown rice, except when I go to my mother's, she's not going to do. Yeah. So you're going to stick to the traditional way, right.

Interestingly those not to completely digress here, but brown rice is actually, most of the brown rice is very high in arsenic. Arsenic is a heavy metal that, can increase the risk of prostate cancer.

So actually, brown rice, which I've been saying, you know, has the fiber and thing. So diet is, as you talk about AI and how to personalize more and how to make it how to be more prescriptive, making this micro environment more hostile to these cancer cells.

We really have to get the lifestyle element right. And what's the truth? What's the truth? Because I've done diet stuff for 25 years. It is complicated.

It is it's but the thing is, it's it's but it's arguably a lot of it is is all truth is just a matter of. Because the moment we make it binary or bucket it, that's when it gets complicated, right?

Yes. It has like, you know, increased amounts of arsenic that we know can cause breast and bladder cancer. But at the same time, you know, I had Jason Fung, who wrote, the obesity code, cancer code, diabetes code, looking at me, and he really hit me hard.

He's like, even if you're not diabetic, he's like, what's happening? You're you're surging out insulin to pull away all this glucose that you're subjecting yourself to with fast food and stuff in a way that we've never been exposed before.

We've never had the concentrations in our physical, in our biology and physiology that we subject ourselves to regularly. So it's like pushing an engine to 8000 RPMs, you know, revolutions per minute, like in third gear before going to fourth.

And yeah, you can do it every now and then I have fun. That's why I said like, bro eat, you know, eat the fast food, whatever every now and then. Like sometimes you want to eat it hard, but if you're doing it over and over and over again, it's not good for the engine, it's not good for the car.

And so insulin is a growth factor, right? He's like, that's what he says. It's a growth factor dude. And what is cancer. What do we say earlier unregulated cell growth.

So that's why we're like I'm not diabetic I have cancer. But you had a ton of insulin spikes. You kept looking at the

9000 rpms and third gear like you're you're you're whipping growth on all your cells.

And again cancer is unregulated cell growth. So so that's where we can really have these tailored customized things. You know if you have a Brac a mutation that arsenic matters as a male because you know your chance of prostate cancer is a lot higher.

But if you have a whole very heavy history of insulin driven tumors, the tumors, when you look up and say, diabetes matters, which one? I think one day we'll say hyper insulin matters.

And we know it's linked with this kind of cancer type. Now all of a sudden, brown rice may be more appealing if your risk of prostate cancer is low. Like it's like it's a risk reward, but it's never a one size fits all.

And it's also the poison. The poison is in the dose, even from an insulin perspective. I had this conversation with a Cuban guy who just arrived from Cuba to about two years ago. I think you'll find it interesting.

He said to me, you know, you Americans go crazy on sugar. Like, what's wrong with you guys? I was like, yeah, because, you know, there's a problem. Insulin.

He's like, we live on it in Cuba. We literally well, scarcity. We don't have a lot. So we are either playing baseball all day or wrestling, you know, Cuban sports or in the farm all day.

And literally this is what fuels us. We don't have a choice. Literally sugar and water. And I thought about that. Sanjay. And that's a very interesting and you know, the, the the mortality rate is more or less analogous to to, to us here. So.

You know what your body does to food.

Lifestyle, Diet, and the Tumor Microenvironment

I always say my my common answer lately is hey, Jill, does meat is meat bad? Hey, Geo. It's always it depends. It depends. Personalized precision. What you're saying it depends.

Yeah, exactly. A sedentary person versus an active person versus a very active persons are three completely individuals, metabolically and what they do with all food.

Because now we can also make the optimal well, you know, protein you know, and certain amino acids, you know, signals and stimulates the mTOR pathway and it can cause cancer.

It depends. It depends. Are you lifting weights? Are you doing weight resist? Because if you are doing some level of weight resistance exercise, the protein amino acids go in a different direction, not just the liver.

It has to start feeding all these muscles. Yeah, right. Sugar. And literally I always have to say I'm not recommending anyone to just eat white sugar or drink white sugar or anything like that, actually say get away from that.

But the point there is that, you know, white rice or, you know, fruits or whatever, that it depends, it's, you know, when you, when you, when you have an active body, the, the sugar goes actually right into the cells without even insulin, you don't even insulin in a person that that's active, you don't get those, glute transport proteins are not even necessary.

Just kind of goes or a different pathway goes right into the muscle. So a lot of it is it depends. And so I'm looking forward to, I have some ideas, but I'm sure that, you know, I is going to be able to kind of tweak this a little bit, and up and down.

Yeah. I want it because I my goal with what I do, Sanjay, people say, you know, urologists, I work with all urologists

and urologic oncologist from all over the country.

Gee, I just saw your patient, and he's doing so well how you know, he you active surveillance. He had cancer and now his biopsy is negative. What's happening? What are you doing with them?

Well, first of all, I don't know that I treat cancer, I treat the person, I treat microenvironment. And so I'm trying to be extremely prescriptive based on what they have their, their age, their, type of cancer with their diet, with, you know, how much wiggle room can you have with their exercise regimen?

It's not just go. Exercise is not get on the treadmill or just, you know, do aerobics three hours a week. It's not it's way more prescriptive than that.

Sleep. We're talking before. Well, I only do five hours. You know what I know what I do with patients and myself. First of all, can I do it? Do I do it?

I'm at a point because some people have in insomnia, say, look, it becomes more stressful to try to sleep better. It's almost like the guy that's trying to meditating.

Hey, Sanjay, go meditate. Why don't you go try meditating? All right? You know, I say, hey, stop your meditating. Give me a second. Don't talk, you know, so they're like, stressed about meditating or the process of meditating. Same thing with sleep.

Could be stressful actually, to try to sleep better. I said, you're good for five hours. You're good. I think that if you go to sleep the same, same sleep time, same wake up time, based on some experts that I've spoken to, that's actually probably even better than eight hours, right?

Sleeping every day, same sleep time, same wake up time. If it's five hours, 12 to 5, you know, I mean, the point is, this is this is exactly what personalized precision carry is, right?

That's your point. You're like, it just all depends. But they all depend. Also, it all depends and they all depend. Meaning when we talk about the intake to your point, the same way I talked about cancer predominantly one of two things.

It's the on switch that's stuck or it's the blow up that doesn't happen when you're talking about what's going in. That's just one of the two, but you're not talking about the stuff that is anti-cancer, right?

Like diets, the fiber that you're talking about, the fruits, the vegetables, the things that are colored. That's the other side that can very much change the dials.

You can have way more, you know, less discretion, I guess, on what on carb and take this out. And the other, if you're giving your microbiome and your gut health and your immune system a robust exposure, in my opinion, to vegetables and fruits and all the fibers, that needs to be robust. Why?

Because there's a lot of, belief and data coming out to suggest that diets directly affect our immune system, which we know that's a fact, has a very big influence on permitting cancers to evolve and become invasive or not.

We know that, unfortunately, Aids taught us that we saw horrific cancers very quickly when we know that the lymphocyte antibody system goes down. So whether you're talking about sleep, whether you're talking about, you know, adipose fat, tissue consumption, exercise, they all they all have an effect on your immune system.

Right. That what you eat again how much you work out. Do you need glucagon. If you're working out a lot your body's pushing glucagon. Bring out more energy into your blood which is great.

That's healthy. You're burning the stuff that you stored away. And so if you had a lot of if you have insulin, when I say insulin, I mean your body's insulin and endogenous insulin, not that you're taking it if you're making it, if you're

needing those spikes, but you're also balancing it the other way, a lot more tolerable versus somebody that isn't ever needing that glucagon surge to bring out, you know, to to burn the fat so that they have the power to do that run or whatever it is, and it's only insulin as an insulin.

That's a different story. Yeah. And all goes to two things like, you know, again, immune system, the good and the bad, the pros and the cons and how much that you're beating it up for to weather the storm or I mean, and inflammation because inflammation we know down regulates the immune system.

And then chronic inflammation is a whole thing. But but these things is these things are what we talk about when we talk about precision and personalized cancer care that we are going to.

It's not here, but we are. When you hear the things that say, you know, multi-omics or, fino mix all these x, they mean let's start considering everything together.

The blueprints, which are in your cells that you are coded with from your mom and dad, the ones that you have today that have nothing to do with what you were given.

Because, remember, 90% of cancer is not due to something as far as we know. And now we can sequence the whole genome. As far as we know, it's not related to something that you are inherently given inherently, but they are changes that occur that become the new coding in your lifetime.

You're not passing those down on your kids, but the cellular changes happened in an area of your body and those cellular changes happen to exposures. To your point, the arsenic that you're seeing is exposing the cells down your prostate to arsenic regularly, right?

I'm not saying for brown rice. I'm saying in general, if something is for sure, then then that is not going to pass. Your kids and I had nothing to do with your parents did.

It was the fact that you did that. What's worse, smoking tobacco smoke as it goes down the bladder. So people are going, but I don't have a history of cancer in my family.

They may not have been smoking tobacco and peeing out all the byproducts for 60 years. And so that environment got screwed up. And then they started saying like, yo, we're not part of the system.

We got to grow like they're all against us and they start growing and then it becomes more invasive and become stage 2 or 3. And then it's like, we got to grow.

We got to grow. And then like somehow, you know, you learn that it knows how to put a thumb up to hitch a hitch, a hitch a ride and go somewhere else.

And so you and then I don't even want to get started on the concept of we guy, especially in a stage or, you know, scenario where there's a lot of cancer spots all over the body.

We guide it on one colony. So like if the cancer has been around for for 12 months, 15 months, we're only looking. We only took a, an evaluation of that colony.

Like, how do we know it's not different below the diaphragm if that was a lung lesion. But that's where blood tests matter because blood tests you'll notice if your doctor is getting it.

As an ecologist you'll notice a whole bunch of mutations. Sometimes that's like it doesn't mean all of them have all of them, right. The ones you're seeing. But it could be here and there.

And we got to figure that out, because if we're guiding it on one mutation, which is targeted therapy, a lot of the target therapy stuff that's sexier that you're hearing about your guiding it, but you don't know if all of your cancer cells, the billions now in different places express that that's the other at the time is partial response.

So what do we do? We give you a systemic therapy, meaning a pill or something to the IV to kill most of it. But what I do and many people do academic centers sometimes is if one colony is behaving differently.

But but it's growing the liver. This this one is keep growing, but everything else is shrinking. Then they go for that liver lesion with radiation, or interventional radiology will drop chemo beads because you're trying to now keep that fire down.

The water doesn't work on that fire. And all of this is personalized precision. And to your earliest point on the on the ordering, when we say data, it's data like you have to be we have we won't be able to figure anything out unless we get the molecular blueprints and know what works and doesn't.

The ordering I always said forever, I don't need it because I can't change anything right now. But what it will help, what it will help. I'm just going to put this out there with AI.

When we talk about wearables and things, it helps us, appreciate things we don't even know matter right now that the increase at 10 to 12 of beats of in your heart rate doing XYZ down the line, we may discover that's actually something that leads to a heart attack, you know, fold or whatever.

And then when they come out with that technology and you don't have the data to populate it to get the insights that we didn't even know mattered all of a sudden, like, like you're a little behind, then you get the rain.

So that's why now I'm about to order an hour after I discovered this last week, I'm like, I need to know my longitudinal data today. The one of the benefits, aside from the sleep score, which is stressful for for a lot of people, as I said.

But, the heart rate variability data is even more important than in the sleep score. And, yeah, I want to know that I slept, but how much? REM and deep. Right.

From my talk, do I want to know that?

AI, Data Validation, and Closing Remarks

But the heart rate variability data huge. And I think we're going to see a lot of links, if you ask me. Theoretically, Geo, what do you think? Man up.

You know, ash Tiwari, you're you're wrong. Prostate cancer, Mount Sinai. We're good friend, and he loves my work. I love his work. One of his conferences, we talked about the the love hate relationship between the nervous system in the prostate and that the nervous system around the prostate is actually a transport mechanism that no one talks about to to get to get cancer, to metastasize, which is the ultimate thing we're trying to avoid, right?

We we want to keep it there and then do something there if we have to not not have it, metastasize or spread. And he says this is an interesting phenomenon.

I look at the research like, wow, there's a lot of data with, you know, think a thicker sympathetic nerve, nerves around the prostate that are associated with more aggressive cancer versus thinner sympathetic nerves around the prostate.

That's heart rate variability. That's that's the only way to measure sympathetic, nerve response through heart rate variability that we know now. So we can kind of measure that better.

Our rings or even I watch I have I'm surprised I don't have things on my right arm. I'm always I'm sure I'm looking at everything. That information is valuable.

I want to be very sensitive to your time because I know that you have to attend clinic. But I want to ask you one more question, if I may. Artificial intelligence.

I'm a I'm a I'm a I'm a layperson here with AI. How do we know? Right. So you said it's all about pattern recognition. How do we know that the system is getting the right information and not bringing in the information that's invalid or inaccurate and bringing it in, in and giving you the information?

You know, a mixture of good information and bad information. How do we know that? Yeah, I love it. So when you talk about, you know, ChatGPT open AI and some of these things, it's a great point.

They just looked at everything, right that they want to be. They want to be your personal assistant rather than Google being a you tell the personal assistant they they do all the work and then find it for you.

That is what the GPT is doing. When you think about these general ones. But when you're doing AI, when it comes to medicine and health care, you're training it on.

You're choosing the data. So you have to train it somewhere. So I'm taking a Harvard course that's that's, AI in healthcare and I don't know, implementation or something.

And they're showing us all the different models and a lot of them, they've trained like the study is in the Harvard institution or, you know, if it's Hopkins or Mass General and you're only giving it what all the physicians are doing, it's not pulling from the internet, in another words, or it's not pulling some other subset of data.

It only it can only process what you give it. So they would give to say, all the prostate cancer patients, all the breast cancer patients or whatever, those, you know, inputs and labels are.

And then it just that's where there's all kinds of what's called, training or learning. There's supervised, there's unsupervised, semi-supervised. There's a lot that goes into making sure that it knows what it's doing.

And then what happens is now you have your output, now, you know, like say, you know, they observe that statins matter at this large institution. Just just by looking at the survivals, you can you can say, give me the data points to where, you know, the survivals are more of that.

And then you can fact check that, that part's easy. But the key is to your point, how do you know? How do you really know. And that's called validation.

That's the next step. Now that it's now that you're happy with when you feed it the next, you know, person or patient, it's actually spitting out correctly and consistently.

And you've studied it for months now. You go to a different institution and now everything can change. And you have a lot of variability or the physicians doing it right, or they testing the right things.

Are they up to date on their meds. So it does. It's going to take a lot of tailoring, but that is the way that you are making sure it's trained and you are selecting what those, data points for the analysis are.

Yeah. I mean, I could talk about forever about it, but that's, that's, that's where we're headed is to say like, you know, is it aptly trained and what is the purpose of it at the end of the day, to of that specific model?

Lovely doctor Sanjay to Nasir, I should I should note Juneja is like my partner in Asia and there you go at the ONC doc and the podcast is, where is it?

I have it here on the podcast. Go ahead, say it's target cancer podcast. Target. Okay. I wish I took that podcast out of it. That is part of the title and it's on.

Yeah, I think I it may be for me is that Doctor Geo podcast just like kind of you know, is he flows in that right doc with your podcast. Yeah. We always think about those things.

Sanjay. I cannot tell you how grateful I am that you've decided to be part of this summit. And the fact that we've

connected, I'm super grateful, to meet people like you, how you work, how you think, how you look at the world, how you look at medicine, and, you know, the future of health care.

So thank you so much for being on. We'll stay connected for sure. Let our folks know where to find you and anything else you want them to know about you or your work.

Yeah. Yeah. I mean, mostly the just the Instagram and TikTok handle is where I put some content out, which is theoncdoc? Pharmacology oncology doctor, I guess.

And then, Doctor Cancer podcast is on YouTube and it's on Spotify and Apple and all of this, and and yeah, I mean, I just, I just pump it out there and, trying to make my life again.

I do think sleep is extremely important. Figure out how I can do what I think we're responsible to do, which is given information that we've been privileged to receive an education observation, whatever.

And everyone listening, I mean, you all now, the hot potato responsibility or duty, potentially, if you think of it that way, too, like you have knowledge that you want to share with other people that are either stressed or misinformed or or whatever, you know, we do this together.

I just I just believe in synergy so much. Never an individual thing. Love it, love it. Thanks so much Sanjay. Thank you, Doctor Geo. Thank you. Thank you everyone for tuning in yet to another episode of this prostate cancer summit.

Look, we bring the goods each and every time. Amazing people coming on, sharing their expertise. So thank you for tuning in and I'll see you at the next episode.

So long, rock and roll. That was awesome.