Hack Your Genome: The Vital & Inescapable Role of Genomics on Your Health Transcript

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Introduction and Speaker Background

Hi, everyone. I'm doctor Sanjeev Goyal, and this is the advanced Anti-Aging and Technology Summit. I have Doctor Mansoor Mohammed with me. I'm unsure.

How are you? Pretty good. And yourself? Sanjeev. Very good. So I'm just going to give you a little. I'm going to do the bio. You've pretty extensive bio here.

Doctor, Mansoor is the chief scientific consultant at the DNA company, a leading and innovative provider of comprehensive functional genomics testing and consulting and an industry first individual customized supplements.

Under your guidance, the DNA company has pioneered the term fluency in the language of DNA and is revolutionizing the use of genomic testing towards optimized health.

You're considered a pioneer in medical genomics, has been a recipient of multiple academic and industry awards. He is the holder of several, patents in the general fields of molecular diagnostics and genomics research.

As one of one of the most sought after national international conference speakers in the genre of personalized medicine. So yeah, I'm so thankful you had some time for me today.

And I think it's such an interesting field, and I know our viewers are going to love it. I've spoken to you before. I have a couple of questions, but maybe let's just start with some of the basics, like what is the genome?

So we can kind of get them up to the level that we want to get them to. So let's start with some of that basic stuff. Tell us a little. Sure. You know, one of the things I like, Sanjiv and and we've, we've we've had I've had the pleasure of sitting with you and the awesome way that you view medicine and you view the human body.

And I think from that perspective, getting a definition of the genome. What is the genome? It's the operating manual of the human body. So if we can think of it from that perspective, you know, we all have a basic understanding that we inherit these genes, that we have this inheritance that make us quote unquote, look sometimes even behave like our parents.

And we sort of look sometimes more like mom's side of the family or dad's side of the family. And we may even have certain traits, behavioral traits, quirks, personality, you know, things that define us that someone can be a boy that's just like your uncle, that's just like your aunt for your grandmother or your grandfather. So what does this mean?

It means we understand that we inherit certain things and this inheritance, which comprises our genes and in totality, our genome, that for what I've just said, that genome, that thing you've inherited contributes and in some cases defines the way that your body behaves and looks like, I should say, the way that you look and behave.

So in other words, we get the concept that, you know, this nose is more like my mom's, like my hair type, or my color is more like my dad. But what we need to understand is that your genome in conclusion, control is not just your outward form, but your inner form.

It controls or it contribute significantly to how your cells behave.

What the Genome Is and Why It Matters

So in conclusion, your genome is this amazing operating manual that you inherit, that defines or contributes or spells out how all of those little inner workings of your cells, the efficiency with which you do this, or the inefficiency with which you do this.

That's your genome, and that's why it's so integral to the type of amazing practice that you practice, which is this personalized precision anti-aging medicine.

Because to know the genome is to know the capacity inclusive of the efficiencies and inefficiencies, the optimum abilities and suboptimal abilities of the patient that you're dealing with.

So so I know that you run DNA company, which offers these, functional genomic testing to so people can find out about their genome. I mean, people can also go to 23, and me and I know there's a difference.

I want you to kind of explain to the viewer why what's happening, the DNA companies different than what's when you go to 20. And did you know, I actually just gave an interview with another incredible personalized precision medicine doctor.

And she said to me in the interview, she goes, you know, and I did 23 and me, before I did, she had done our testing. In fact, she'd done our testing simply as an interested clinician.

And after she got her results, that was when the sparks sort of the, the, the, you know, the real moment of wow, okay, this is different. And so she came to us having experience what we offer.

So what is this difference. And in her own words she says, you know, I found out in my 23 in me that I was more likely to have a unibrow. I found that in my 23 in me that I was, you know, like coriander.

Maybe. But she actually said, I found that I was more likely to be one of those people that can smell, asparagus when you eat it in my urine. I, I and she was laughing in the interviews. Me she goes, well, for the first, you know, I knew that was going to be the case.

I just had to look in the mirror when I was drawing up, you know. And for the second, equally, if I was inclined to, I could smell my own urine and determine if that was a problem.

The point being, 23 had me has done an incredible service and continues to do an incredible service. By what? By making genetics and genomics and a person's, you know, this the coolness.

They've they've made genes and they've made genetics cool. And they've made it the topic of a, you know, coffee conversation and dinner conversation. And I think this is a good thing.

I think that by letting people know that, look, there is such a thing as your genetic manual, there is such a thing that you that ferreted this awesome way in which your body works and learn about that.

So they've they've at least opened the door to that coolness. But what do we do differently? Well, clearly. And I say, you know, if you want to know the color of your eyes or the type of hair you have, go look in the mirror.

What we want to do differently is we want to get in here. So here comes the word and which is what defines what we do. Functional. Yeah. We want to read the human operating manual with the perspective of putting aside the cool factor of these things we've just mentioned.

But I want to know I want to know for myself and I want to know for my patients and for the people that come to us. I want to be able to tell you when you get exposed to certain things in your life, certain environmental toxins, when you eat certain things, what are your metabolic processes?

How is your body more inclined to making the hormones that are so vital to your optimal performance? How is your body more inclined to absorbing and transporting the nutrients that are fundamental and pivotal to optimal health?

What forms of those nutrients are you more likely to respond to? I want to be able to tell you something functional, operational, I want to be able to tell you, or I want you to be able to tell me what are the top ten things as you're going about trying to optimize because, you know, we're we live in a time where access to good nutritional information.

I mean, we have the whole spectrum, good and bad nutritional information. But the point is, we live in a time of data, and we live in a time of access to information and even one of the silver linings, Assad, is that my song of the pandemic is online.

Information is becoming rapidly available. Health information I'm speaking of to the consumer. So good, good. We live in a time where health information the good, the bad and the ugly of it, the intelligence and the unintelligent of it is available.

Fair enough. But what we want to do is we need to realize that it's not a one size fits all. And anyone who's pursued health, anyone who's been in a fitness program with their peers and their friends, been in an attrition program with their fitness and, you know, with their peers, they've realized that things that might work for some may not work for themselves.

So they realize this concept of individuality. Personalization. Once we enter into the realm of individuality and personalization, we enter into the realm of your unique genetic operating manual.

So what I want a client to tell me is I want a client to tell me what are the what are the top ten health goals that they've been working towards? What are the things that have seemed to be the Achilles heel to these goals?

What have they attempted and failed up? What have they attempted? Seeing it work for others? Eating habits, fitness habits, anti-aging goals that they've they've tried, but it didn't quite work for them.

And what do I want from the human genome and what does the DNA company give? We want to be able to give them that functional, intelligent understanding so that can be applied, not just superficial, cool, superficial, but not the superficiality of genetics.

We want the functionality of genetics, which is what we do. Yeah.

Functional Genomics vs Consumer DNA Tests

And I mean from my understanding, what I really like about my report, which I'm going to just pull up in a quick second to kind of I have a couple of things that I want to get into, but the idea that you're looking at things together, like not just this gene at a time, but these things are all inter playing.

And I think that piece is really that brings a whole lot other layer, which you cannot get from from some of these. And this, by the way, Sanjiv, so brilliantly that you bring it up, that's the that's the base that's the the foundation of functional genetics.

Nothing. If we understand which is what I just said, that our goal is the functional performance of a cell. You see looks and all genetic makeup defines how our cells work, our cells behaviorally.

How do cell how do our cells absorb the nutrients that they need. How do our cells get rid of the waste products, the toxins that they produce both internally and that they're faced with because of our environment?

How do ourselves build the proteins that we need? The neurochemicals, the hormones, the things that we need to function at the cellular level and therefore the organelle level, and therefore at the holistic level.

This is the human being. And we want to be able to read that manual and infer these awesome functionality, these things that make us who we are. Well, the moment we get to that perspective, we need to understand nothing in the human soul works a single genes doing their own thing, rather all of these beautiful behavioral concepts of a

cell.

How good is the cell of doing such and such? How efficient is the cell at detoxifying such and such? It relies on gene pathways. It relies on gene networks.

It is the inclusivity of multiple genes, each doing their important functions, but doing them in a coordinated circadian rhythmic pathway manner. And once you are able to read the pathway, once you're able to read the interconnected interconnectivity of the genes, that's when the magic happens.

That's what we do differently. Yeah, I think that's exactly what's so well, so well said. So this is a longevity conference. I'm particularly interested on how the DNA of the DNA company test can help guide somebody on making better decisions if they want to prove their longevity, decrease inflammation in the body.

I was looking at some of the some of the gene markers that you're looking at, inflammation of the lining of the endothelial cell. I believe it's one of the genes.

And, I'm actually interested in, you know, another one, you noticed is there some genes that can tell you what type of diet might be better for him? So I don't know if you want to talk about you know I and this is the thing that makes your practice so amazing.

And obviously being a pioneer and leading these type of conferences is that you get that. I used to think this was superficial a few years ago, because I would hear it in some circles, I've now come full circle and realized that it's not superficial at all.

If someone wanted to ask me, when is it even possible to use one word or one term that defines the greatest obstacle to human health? And I would say I again, I used to say, no, it's impossible.

Now I think it is possible. And I would use the word or the term chronic inflammation. I okay, it seems like an overly superficial catchall, but we've now by studying every form of human disease, whether it be disease that is internally meaning something that is not happening because of our genetic value, by the way, efficiently.

And so we're, we're we've got this inheritance of this predisposition to certain health outcomes, or whether it be because of a challenge, we might have the best genes on the face of the planet.

But after all, if we're constantly you switched constantly being bathed in wrong, in the choice, you know, toxins, environmental purposely, you know, purposefully or not, or whether it be diseases based on infections very important in the times that we live in a baseline.

And by the way, we're including things like cancers and when including things like metabolic syndrome, diseases of diabetes and so on and so forth, that what is, is there is it even possible that at the baseline, at the background of all of what appears to be these diversified diseases, is there a common denominator?

And the answer is yes. Actually, there is the concept of cellular inflammation. And so let's dive into this very quickly because it will be the roots of anyone, anyone who is intelligently pursuing the concept, whether as clinicians for their patients or patients for themselves.

And of course, as clinicians, we want to practice what we preach. So we do it for ourselves. If we want to practice optimal living, optimal health with a viewpoint of longevity and not longevity here just, you know, I want to be 120.

I want to be 100. But longevity that we age well and that we're still functional at 80, at 90, whatever age that magic number is, we've got to understand this concept of cellular inflammation, right.

And the capacity for it. So let's quickly dive into this ultimately synergy. If you break it down, what do souls have to do. Each one of our cells, whether they be a retinol.

So, central neural cell, a skin cell, even the lining of the respiratory, the epithelial lining of the respiratory system, the endothelial lining of the blood vessels or kidney, cell, liver, cell or cardiac.

So whatever it may be, a cell. Ultimately, let's break it down. It needs to live. Cells need to live. In order to do so. They need to absorb nutrients, right?

They need to respire. They need to absorb oxygen. They need to combust the oxygen with the nutrients that they absorb create ATP or the equivalent of it for energy so that that cell can live.

Bottom line basically know when a cell does that. Yeah. Depending on its function. Maybe it's a peripheral muscle cell a peripheral neural cell. Maybe it's a kidney cell a nephron Excel.

Maybe it's a liver cell or hepatic cell or vascular cell, whatever it may be. This is just done. Its job of living. Now that it's living now that it's able to take from its environment.

Oxygen, nutrients, respire, make ATP. Now that cell has to do something okay. And the things that that cell has to do might include making the slew of different proteins that it contributes to the function of the cell that contributes to its surrounding tissue.

It makes up the organs. Okay. So cell cells have to live and then they have to contribute functions. Now when we take these two basic concepts, when cells are living and contributing by whatever job they have to do, what happens to cells?

Two things they produce as a natural byproduct of living, a natural byproduct of making that ATP. They produce oxidants and these oxidants. We have a delicate balance. And look at the beauty of this.

I consider it beautiful. Some might consider it morbid, but the very things that make us live also are the things that will slowly kill us by breathing and consuming, by creating ATP to live right.

The law of physics is we cannot create energy except we create a byproduct. In this case, oxidants is that byproduct. And by the way, those oxidants are the things when in surplus.

Because by the way, a certain amount of oxidants is necessary for cellular behavior. But in surplus, oxidants are the things that will slowly erode the functional of a cell.

So this is the first thing that we've got to understand. Okay. And again very quickly the other thing that we said is as the sun is doing this function, some cells more than others, it's exposed to environmental toxins.

Genetics, Longevity, and Chronic Inflammation

Because when we breathe in we don't just breathe in oxygen, do we? We breathe in the pollutants that are in the air, organic and organic. We produce. We breathe in mold, toxins that might be in the air, for example.

Certainly the chemical toxins that are in the air. We eat things that have preservatives, pesticides. And so the other things that cells have to do is they've got to have the ability, they've got to cells have to recognize that they're going to be exposed to things that are beyond that which they needed for healthy, functional living.

They're going to be some nasties that come in. What does this set the stage for Sanjiv? It sets the stage that cells. And obviously based on the individual their cells have differing capacities.

You need you John Paula Lauren will have different capacities to do what two two capacities that we have to look at the efficiency with which we produce energy, i.e.

absorb the nutrients, take care of the mitochondrial functions. So we may have different innate capacities to make the energy so that we can live. That's an innate capacity, and we have differing image capacities toward us to deal

with the oxidant.

Toxic exposures, which combined together creates a unified equation of inflammation. So here when we summarize these two things in quick summary, we have if the goal of longevity is to keep our cells producing energy that we can live and keep our cells doing the functions they need to do all of the different functions as optimally as possible.

We must know that on the flip side of that equation, our cells have to accomplish two fundamental truths which are embedded much of it in their genetic code.

And therefore, to answer your question, those are the things we need to go study. What are the two fundamental truths in your genetic code? That's the fundamental of how efficient are you at keeping your doing the jobs, the mitochondrial energy producing jobs that they need to do?

And how efficient are you at getting rid and handling the inflammatory, byproduct of living? Right. This creates a seesaw balance. Sanjiv and in conclusion, the individual who has the genetic capacity for efficient energy production and efficient toxic inflammatory clearance when their balance is in favor, that they're always producing energy better and keeping their inflammatory load lower.

This, this, this dynamic system equals optimal health. They're not there. Not each other though, right. Like these are not against each other. They're not.

You can have both high that are both low. It doesn't. Absolutely. And so of course the individual who because of their lifestyle choices, environmental exposures like, nutrition choices is that for what is unique to them genetically predetermined, if they find themselves because of their choices and because of their innate genetic capacities, that for periods of time in their life, their inflammatory load exceeds their anti inflammatory potential.

So now they're existing in a surplus of inflammation, a person that stays in that modality, that's the person that's walking the path for chronic disease.

That's the person that is going down a path that is not equal to optimal longevity versus quickly. The person who is in their inflammatory load is in a deficit, meaning their cellular capacity, their ability to create that energy to cure if the inflammation is a byproduct intentionally, unintentionally, innately, and that which comes in external, if their capacity, everything is working well, they're using the right fuel invisibly, the foods they eat, the right environment.

They're making the right lifestyle choices. They're not exercising in a way that is exceeding their oxidant capacity. Not all exercises are created equally.

They are feeding their unique microbiome in a way that their unique, their unique microbiome is living in harmony with their body so as not to put additional strain on the body.

Because the microbiome, Sanjiv, is one of the primary sources of the inflammatory, potentially primary sources of the inflammatory challenge that the human being faces from within.

If such a human being has all of these things in their favor, such that the their capacity exceeds the load of inflammation that they are faced with, that's optimal living.

That's the person who can go on having cellular functions, cellular repair, cellular replacement, and living, 70s and 80s and 90s and beyond. You think that that's some like some amount of damage, metabolic damage or inflammation is occurring in everyone?

I mean, that's why aging is absolutely, absolutely right. And that's what you. Absolutely. Yes, absolutely. We all nobody can be at zero. Like I'm not reducing it to the level that can I mean whatever you can do to have it to the minimum level possible.

So there are three factors here. And you've nailed it again. Unsurprisingly Sanjeev, the factors were never zeroed out. The fact is our capacity. So let's let's let's get this concept.

There is a capacity cellular innate capacity through all of these amazing mechanisms of antioxidant detoxification, mitochondrial function, nutrients, absorption, cellular membrane performance, hormonal performance.

Through all the summary of all of these things, our cells have the capacity to handle inflammatory load okay, inflammatory load. The higher the capacity to handle that inflammatory load, the more we can tolerate.

We have one person who has smoked all their lives and drunk cognac from since they were 14. And here they are at 18 with a cigar at 80 with a cigar in their mouth, a cosmic in hand.

And they're healthier than you and me put together. Okay, so we have that innate capacity to handle inflammation, and we have the inflammation that we're exposed to.

Now, the inflammation we're exposed to is number zero because that inflammation is a necessary byproduct of our cells living. As we said earlier. So with time there's always a background erosion.

There's always a background diminishing of our total cellular performance. But the higher the delta, the greater the delta between cellular capacity and cellular challenge, the slower is the recruitment or the accrual of that erosion.

Okay. So this is the first concept that we have to focus on. The next two concepts are also as important. And that is well actually can be put into one concept.

And that is the state of the stem cell population in our bodies. You see, as this erosion is our cells are doing the jobs capacity versus challenge, and they're going along throughout our bodies.

Cells have two options. Well, they have three options actually. The first option is they do their jobs and there's a half life to the cells. So cells are doing their jobs.

And of the billions and trillions of cells in our bodies, different cells have different half lives. So skin epithelial cells have different half lives within cardiac muscle cells or central neural cells or kidney cells or liver cells and so on and so forth.

So different cells are able to do their jobs. Red blood cells, you know, famously have a half life. What is a doctor? 40 days. What is it in 90 days. Something that is that the half life of the life on your right.

Maybe 40 days. Yeah. Complete turn out, but 40 days for half life. Right. And so they they turn over. So they're doing the jobs hemoglobin in them taking an oxygen, transporting the oxygen to the body.

And those red blood cells have a half life. They're not the red blood cells circulating in my body. In your body right now are not the same red blood cells that were there when this pandemic began last year or October of last year, or even maybe December of last year.

They're different red blood cells. So the point is this when our cells are facing the situation of capacity versus load, they've got to make they're doing the job and the body has to decide, am I going to keep that cell doing its job as it starts to erode, as that cell becomes weaker and weaker at doing its job over the period of the life of that cell?

Am I going to attempt to fix that cell? Just like going to attempt to repair? Or am I going to attempt, or am I just going to kill it off and replace it?

Right. This is the second and third, components of longevity. So the first is where are you on the equation of cellular capacity and cellular load in terms of inflammation?

Number one, how good is your body at making the decision. Should we keep that cell going even though it's now

running at 80% of its original? You know, I'm trying to keep a past capacity now.

It's 70% efficient at what it's doing now. It's 40% efficient. Okay. Hold on. Time for you to either be replace or whatever it you like. What do you think when your mind what are the things that the factors that influence how the body makes a decision like?

And what could what could one do? Like, for example, could keto or fasting make an impact here? I assume potentially brilliant, right? So the what the of one of the first things we need to do optimize one of the first things and this is something if you asked me this question a year ago and this is this is the this is the great part of medicine that we should always be evolving and trying to learn better, isn't it?

So if you asked me this question two years ago, I wouldn't have been. I wouldn't give you the answer I'm about to give you. And that is the first thing that I'm now utterly convinced of that we need to do to accomplish what we just said is optimize our hormonal systems.

Why? And by hormonal system, say, I mean cirrhotic function. I mean insulin function. I mean sex hormone function. These primary these precedent setting hormonal systems in the body. Why?

The answer lies. And all of this, by the way, Sanjiv, is based on our genetics, which we look at. These are the systems that we look up at the DNA company.

What did I mean to you? Asked you asked the question of anti-aging. You asked me. And so now that we know we've got to make we've got to optimize where we are.

I know what capacity versus loads, of course, load, inflammatory load. Everyone can address this by the decisions they're making, right? I mean, the person who is smoking a pack of cigarets a day is adding inflammatory load to their body way more than the person who's not smoking, the person who's made a conscious decision to eat healthy, unprocessed foods, foods not laden with added sugars and and food colorings, and so on, has made a decision to reduce the inflammatory load of what they're putting into their bodies.

Okay, so we can do that. But then we have this other thing that is hard for us to figure out. How do we help our bodies to make the better decision between replace and or repair?

How do we keep the cells that we have, which are turning over at different rates depending on the cell type? How do we keep those cells? Pick a number working at 75% efficiency.

Because here's the thing, Sanjiv, do I want in any given organelle type, do I want most of my cells at 75% or more capacity, or do I want cells still sticking around doing the job at 40% or 30%?

I think not a former. Yes. Yeah.

Hormones, Epigenetics, and Gene Expression

So because we want our bodies to have the ability go you drop below 60% of you go put back in the new guy who's back up to 100% or 90%, as the case may be.

We want this. And to do that, we need adequate stem cell populations in the body, right. Because those stem cells will give they're the primordial cells roaming the body, either contributing cellular factors that are repair cellular factors.

So stem cells reach the parts of the body. They contribute the environment to help keep those cells rejuvenated, or they flat out repair the existing.

They flood or replace the existing cells. So now you ask the questions mature, what can you do about this? And I said the first thing that I would say now is optimize your hormonal systems. Why?

Because, Sanjeev, the answer to this lies in and so many clinicians. And this is what got me on this path. Don't even recognize how hormones work. How do hormones work?

Hormones, any hormone, whether it be insulin, whether it be thyroid hormones, whether it be sex hormones, testosterone, progesterone, estrogens, hormones, work vitamin D, i.e.

the 125 day dynamic drug sequence, different hormonal, which is as important and more important than most other hormones systems. Hormones work by binding to the receptors, so for every hormone in the body, souls produce the receptor to that hormone.

We have the insulin receptor for insulin, the estrogen receptor for estrogen, testosterone androgen receptor for androgens, vitamin D receptor for vitamin D, and so on and so forth.

Now, what is the key here? When a hormone binds to its receptor what does it do. And I've asked this question and I want to put you on the spot because I know you thought you'd get this up.

But I'll ask the number of clinicians. I said, how does testosterone do what testosterone spike? How is testosterone actually, you know, under denies the body.

How does it do that? Well, it's it's a hormone. It's an androgen hormone. And it impacts the cells by. No, no. How does it do it? Well, what do you mean that to answer.

It's it's a hormone. It gets absorbed and it creates this kind of reactions. No, that's not how a hormone works. The hormone works by binding its receptor.

And one stop receptor is bound by its ligand. It is now called an activated receptor. And this receptor, with its ligand, with its hormone together and sometimes other factors, now transposes and becomes what a DNA transcriptional modifier.

That's what a hormone does, a hormone buying such receptor. And then creates a complex that goes into the nucleus of the soul and initiates gene expression.

I think of the cell membrane actually comes from the cell membrane actually comes in this hormone crosses crosses over the hormone comes in through the external.

The the extra cellular space comes into the intracellular cytoplasm. We're in. And hormone receptors are either on the actual outward facing cell membrane.

They are sometimes in the cytoplasm. And sometimes they're in there already waiting in the nucleus. So the hormone comes in from the extracellular space having been produced 125 day drug cycle, several having been activated through the liver in the kidney.

So now the kidney, which is where the actual final activation of vitamin D occurs, releases 125 day drug cycle of the hormone. I hate when people call vitamin D a pro hormone.

D3 is the pro hormone. 125 hydroxy called Cipro is a hormone of hormones. It trumps many of the hormones in the body. It's a hormone. The hormone is released from the kidney into the bloodstream, carried by a hormone.

Vitamin D transports are known as the vitamin D binding protein. It's circulates in the blood, it enters into the cells. All of the cells of the body.

And different sounds of the body have more or less vitamin D receptors. The vitamin D will bind to the vitamin D receptor, the vitamin D receptor coupled with the retinoid receptor, the Rexall receptor.

This vitamin D retinoid receptor, coupled with vitamin with the 125 day drugs equals the full destroy complex now

enters into the nucleus. And of the 22,000 odd genes that make up our operating manual, a minimum of one tenth of this gene, we think maybe even more, but at least one tenth of all of the instructions in your body.

Those genes only get turned on when this vitamin D receptor complex binds to the gene, signaling to the gene a T genetically that it's ready to be turned on.

Which brings me to another important point, which is my pet peeve how many people talk about epigenetics. Oh, well, document genes don't matter. It's the epigenetics of it.

And they can't even tell me how epigenetics works. Because, by the way, the epigenetic control of one tenth of the human genome is dependent on the binding of the vitamin D receptor complex.

Each one of those things are genetically controlled. So our actual genes, the gene version of the vitamin D receptor, that you have, the gene version of the vitamin D binding protein, that you have, the gene version of the two are one enzyme that activates your vitamin D that you have.

Each of those genotypes defines an outcome vitamin D, getting into the cell, binding its receptor, forming this complex, which then defines the epigenetics of one tenth of your genome.

So epigenetics and genotype are not some independent things that hold. The genotype is an important. But the epigenetics is. This is complete ignorance on the part of people who try to make themselves, you know, experts in the field. Now, every single one of those hormones have their what is called transcriptome.

So we've got 22,000. Our genes and different subsets of genes are optimally expressed and only optimally expressed when initiated optimally by the hormone receptor complex.

So if we want our genes optimally being expressed on, why do we want genes optimally being expressed? Because the optimal expression of genes signals the optimal functionality of the.

So so that's why we think about that's why you're saying about it's important about how the body will make a hormone systems. There you go okay. Right.

So when you ask that question all of that to say yes, we optimize the exposure that we have. Yes, the Cajun rhythm exposure we have to vitamin D to order testosterone and estrogens and thyroid hormones.

And we could pick the main ones and insulin, i.e. reducing insulin. We we want to reduce the transcriptome. And the transcriptome here refers to the the portion of the genome, the x hundred, the x thousand genes who are under the influence of which of these hormonal systems.

In the case of insulin, we want to limit how many genes are in the insulin transcriptome. We want to limit the number of times and the duration that the insulin transcriptome is being expressed.

Right. So sometimes we want to improve the transcriptome of that hormone. Other times we want to limit the transcriptome of that hormone. That's how this is done.

Oh no. No. And when this is done. Yeah. This Sanjiv this doctor glowed. When we the when here meaning the if and when. Because I'm not trying to make it seem as though we've got some mathematical formula that says this is the exact optimal thing.

But once we conceptually understand that when we optimize these transcriptomes, these rhythmic expressions and suppressions of the human genome, in the right cells at the right times, that equals optimal health, that equals up to something, they're just being tied together.

So I don't know if you're familiar with this. It's an epigenetic test looking at, methylation of particular genes. And they have one for looking at a diet for a particular diabetes gene.

And then they can tell you your risk. Yeah. I guess of, you know, how much methylation, which I think might be happening by insulin, perhaps. I'm not even sure by this little one is up into insulin.

Absolutely. And so, of course, the what does the insulin receptor do. And there's a brilliant, brilliant study coming out of the Netherlands. And in that study they showed that when the insulin receptor has been activated by insulin secondary to the exposure of elevated blood glucose.

So of course we eat. Yeah. And by the way, it's not just eating sugars that can do that okay. And by the way there are internal fat storages that when we exercise can trigger a glycemic event.

So sometimes people go I didn't eat, I tested my blood sugar before I exercised. And then I still don't eat. And I tested my blood sugar an hour after exercise.

How the hell did my blood sugar go up on a well? Because your body needed to generate glucose secondary in time if you exercise. So the body has its internal to a limit to whatever that fatty liver and other things that you have.

Point is this what these researchers so beautifully showed? They showed that when the insulin receptor was bound by the insulin produced, because of the elevation of glucose in the bloodstream, the insulin receptor transcriptome, the subtotal of all of those genes that would be expressed in relation to insulin.

Unsurprisingly, many of those genes were the genes required to make the enzymes for the Krebs cycle. What makes sense that when insulin shut down this receptor and that insulin receptor complex went into the nucleus, it turned on the genes.

Unsurprisingly, there are needed for glucose metabolism and the Krebs cycle within the mitochondria. That makes all the sense in the world. But when that insulin receptor insulin complex stayed activated for more than, frankly, short periods of time, other genes under its control were exceedingly pro and created a pro-inflammatory outcome in the cell such that, quote the study in quotes the amazing clinicians that had this study, they said cellular behavior under the influence of chronic insulin receptor activation, meaning cellular gene expression secondary to prolonged insulin, insulin receptor activation mimicked what a body is like when there's a viral infection.

Oh my G. It's literally mimic so that you start feeling flushed. You start feeling fatigued. You start having this surplus of oxidative stress. They literally study gene expression of these individuals in prolonged insulin receptor instant activation.

And that gene expression looked just like if the person was going through a viral infection. People complain like brain fog after they eat a high carb meal or something.

Do you think these things are these are like related effects of effects, potentially 100%, 100% because we are transiently creating this. We are trans secondary to the insulin receptor activation, elevating the inflammatory load within the cell.

And know that inflammatory load may or may not exceed the inflammatory capacity that we each individually have. And so, of course, yes, they're completely no.

One of my favorite, food vloggers is a gentleman called Mikey. So he's one of these famous food vloggers. And, you know, he travels the world eating food, something they vicariously live through.

I don't eat like he does. But the point is this this gentleman eats more carbs. If I were to even slow the amount of carbs that he's eating in terms of rice and noodles and sweets and desserts, I would be dead within a year and few years.

You know, as ostensibly fit as a fiddle. And he's been doing this for several years now. In other words, his innate inflammatory capacity secondary to the initiation of his insane Olympic, transcriptome.

But because of his genetic, his genotype for the factors that lead to that is way beyond mine and that gene that you're referring to. The TCF 702 Thursday, Charlie Friday 702 gene is a gene that is fundamentally related to the activation of insulin sensitivity or insulin resistance.

It's there's a there's an AA and a glial. Right I believe. Right. Yep. Yeah. And so and all otherwise the county of Leo okay. And so when you carry the t a Leo which is the suboptimal version of the Julie a Leo, which I believe that we both carry, I certainly carry the t Leo.

We are what we are innate Lee more insulin resistant. Right. Okay. And what happens with insulin resistance? Which which is a precursor. It does not mean you have type two diabetes and not all people with type two diabetes unnecessarily insulin resistant.

But when the unis of the world literally our insulin receptor, that very receptor that has to be activated to go into the genome when it is less sensitive to the presence of insulin, what happens?

What happens is when we eat a high carb based meal, or at least a meal, whatever that meal might be, that overly elevates our post primordial sugar, right?

Initially, our bodies do not listen to the insulin signal that is appropriate for that level of sugar. So initially we go into a hypoglycemic mode and a hypo insulin mode.

But as the body continues to detect that that blood sugar isn't coming down very efficiently, guess what it does in insulin resistant individuals. It now sends a signal to the islets of longhorns and the pancreas, and we just start pumping up more and more insulin.

We then at about the two hour three hour mark, we now become hyper instantly make our blood sugars plummet. But we're still activating the insulin receptor after the blood sugar has come down. Wow.

And we're still telling the cells turn on the insulin transcriptome, the insulin receptor transcriptome. And now we put our bodies in for a number of hours.

Postprandial our bodies are in that modality as defined by those Dutch researchers and clinicians, where we are in a pro inflammatory mode. All because we ate one too many questions for breakfast.

Well, let me just check here. Hang on. We're going to start with out. Our time is like, how are we doing with time? Yeah, yeah. You really owe to it. Okay?

You gotta you gotta go see. Right. I've got. Because, you know, I told you guys that I was running 15 minutes late, so I have ten minutes. Okay, let's try to wrap it up.

So, just just jumping in there. Just want to finish this topic, because what do you think? Do you think that people's response to the GL, GLP, one inhibitors is different for, for different people because you're saying that now we have different responses to how insulin and this, this type of medication apparently somehow regulates how insulin is, is being used.

And then some people have good weight loss response and some people don't. I'm just wondering because because the pathway upon which these cluster drugs work assumes a normative responsiveness to insulin, that of course, as we go down to type two, the diabetic model, let's see how we got there.

Putting that aside, we still have to come back and realize what is the innate receptivity of that receptor. And if a person is innately less receptive, i.e.

innately insulin resistant, which literally means it literally means that receptor isn't responding, isn't being initiated initially as as efficiently, but then thereafter there is hyper insulin receptor activation creating chronic hyper insulin transcriptomic expression.

Personalized Medicine and Closing Remarks

We can see now that the the stratification of course, that essentially this comes right back to a fundamental of pharmacogenetics, isn't it, that when we when we define therapeutic pathways, we define therapeutic pathways, often in a very generic sense, and we're not considering the individual stratification of the population that we're about to treat the same very quickly.

So that can be talked about. Well, if you've understood and if our audience understands everything that I said, we might say, well, good. This is why hormone replacement as we go into menopause or menopause is such an important and it is an important thing.

Maintenance of hormone, levels in an intelligent way, in an intelligent way, and in a circadian appropriate way is definitely one of the things that should be in the conversation.

For someone looking at anti-aging and longevity. However, if you didn't know the receptiveness of your androgen receptor, if you didn't know that you have a weak androgen binding capacity, that literally your androgen receptor is sparser.

It cannot handle as much testosterone. And of course, we would have seen the manifestation of this in you as a young man in your youth, where you could be slim, you could be, but you'll never have that muscle striation indicative of androgen ization of the body, because mind you, your receptor is just not getting activated as.

And this is different from the conversion like I know. And in the functional genomics you look at indeed this is the only issue is that the receptors in addition to them, this is the receptor.

This is why I say this is why I think, you know, we look we look at our functional pathway to tell you how much testosterone I'm making, how efficiently you're making it, how much of your testosterone are you converting to DHT?

Why do you want to know that? Because DHT, dihydrotestosterone binds with a greater, with a greater attractiveness. With a greater. What's the term? Its binding capacity.

Affinity with a greater affinity to the androgen receptor. One molecule of DHT can out place six molecules of testosterone. We can define all of that genetically.

And then once we determine where you are in your cascade, now we ask it comes back to the fundamental, how are you activating that androgen receptor? How are you activating that estrogen receptor.

Because to the degree that you activated the androgen receptor, the estrogen receptor at the right time with the right intensity, for the right duration, that is the degree to which your cells express the genes, which is the only way that the testosterone impacting yourself to the right transcriptome for the right time, for the right duration.

This is where it comes back to Sanjiv. And this is this is where the DNA company, this concept of the circadian timing and knowing what are you genetic predicates.

So we are becoming the first in the world. If I want to summarize this, and we really becoming the first in the world to dovetail genotype with epigenetics, we're actually showing how your genotype radically influences your epigenetics.

Think hormonal receptor activation of your genome. And it is this confluence, this coming together of a not a sound and intelligent knowledge of genotype with a sound and intelligent knowledge of what what is gene expression that equals optimal cellular performance?

When do I want those cytokine genes expressed? When do I want these circadian genes being expressed? Remember Sanjiv, we had this amazing time together, the particular results of the course two years ago.

And we're sitting there having lunch. And one of our colleagues was talking about taking magnesium three and eight late in the evening, and that she started not being able to sleep.

And I kind of just looked at I said, well, why would you take magnesium three? And it late in the evening it activates Bdnf. And why are you activating BD enough at 10:00 at night?

Yeah, right. So this this knowledge in conclusion singes this knowledge that there is your genotype, your gene makeup and a lot of your genotype influences your epigenetics which then influences gene expression, optimal health, in conclusion, is the appropriate expression of the appropriate genes at the appropriate time for the appropriate duration.

That's optimal health okay. That's awesome. Thank you so much. More. So the more I talk to you more I realize how little I know, but, we keep learning more and more about the human body.

I think this is an exciting time. The cool part. And so I think just a nutshell, I think what I want our listeners to know is that really the the test looks at, I think, what, 30 or 30 or so, by the way, all of these new pathways, we've added them now.

Oh wow. I've got a new that I need to talk again. I need to get up. So the the test looks at a whole bunch of these genes. And then, I can help in a functional pathway perspective.

And so they can understand their own personal eyes, makeup so they can, I guess, optimize the, you know, the strategies need to listen to, reduce the rate.

They they can optimize this strategy even if we get the 5% of it, then the 10% meaning as our knowledge grows, they can optimize the goal of expressing the genes, the right genes at the right time, for the right durations, in the right cell types, at the right times in their aging cycle.

Yeah, for optimal health, that's true. That that it's of course it's a big vision, but that's the real that's where the magic happens. Yeah. That's awesome.

Thank you so much. So have a great day Sanjay. Same to you okay. Time to.