All the Ways Inflammation and DNA Affect Cardiovascular Health – Mansoor Mohammed, Ph.D. with Dave Asprey – #742

Announcer:

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Dave Asprey:

You're listening to Bulletproof Radio with Dave Asprey. Today's guest is a guy you have already met, if you're a longtime listener. If you're a new listener, thank you. And he's my favorite functional genetics expert out there, an incredible guy who understands how DNA interacts with what you can actually do, what you should do, how your lifestyle interacts with it.

Dave:

As you know, I have spent a lot of time, since the very early days. I've had my whole genome sequenced, I've done 23andMe in the early days. And I was overwhelmed to learn I that had 3% higher risk of something that I only had a 1% risk of anyways. Now I have a 1.03% chance risk of something that didn't do anything for me. But it turns out there are some really important things in your genes that do help to determine how you behave, how you act, even who you're going to be attracted to, and things that can be enlightening and useful and actionable to know.

Dave:

And the guy who's my top go-to guy, who's a pioneer in medical genomics, is Dr. Mansoor Mohammed. He's president chief scientific officer of The DNA Company, which is a Canada-based company. He's got several patents in the field and, full disclosure, I am an investor and advocate and advisor to the company. I think these guys are in a position to change the world and I want you to know about them. And I want you to hear what Mansoor has to say because it's amazing. And today we are going to be talking about cardiovascular risk and what your genes tell you about it. Mansoor, welcome back to the show.

Mansoor Mohammed, Ph.D.:

Cool, Dave, thanks for having me back. Always, always a pleasure.

Dave:

So this is your fourth appearance, even though you've only really done two interviews with me before. You talked about women, hormones, and DNA, a really popular episode, back in January. And then in April, we did the ripple effects of COVID pandemic that we're ignoring and how your DNA changes your risk of getting any virus including COVID. So there's a couple of episodes about that in April. And now we're going to talk about cardiovascular system, multigenetic, multifactorial context. So it's one of the four killers that are in Super Human. Most people listening to this, if you don't get killed by cancer or Alzheimer's diabetes, it's probably going to be your cardiovascular system, but not necessarily in that order, because if you get diabetes or prediabetes, which is actually just diabetes, that increases your risk for cardiovascular. So what do genetics have to do with cardiovascular risk? Let's just go straight into that.

Mansoor:

And I think the one word, if you ever want to try to sound remotely intelligent, pretty much now in any disease state that you can think of or dysfunctional state that you can think of is inflammation. If we can understand the foundational concept of inflammation, and we've moved from thinking that inflammation was this amorphous concept that was too vague to recognizing that it is a real concept and it is a real construct and it actually follows a pretty predictable pathway, mind you, multifactorial pathway, within cells. We've understood now that inflammation is pretty much the bedrock of most dysfunction in the body. How that inflammation affects, quote unquote, the cardiovascular system. This is the topic for today, and we're going to have some fun at it.

Dave:

All right. Inflammation is what it comes down to. And I know for instance, because of my experience with toxic mold and Lyme Disease and fibromyalgia and chronic fatigue syndrome, by the way, in my opinion, those are all the same thing. They're all toxic mold. And it's not just an opinion, I've had enough guests on the show. And what underlies all of those things? Give me the I word, what is it?

Mansoor:

Inflammation, every go.

Dave:

Amazing. And the same thing with COVID and everything else. Don't have too much inflammation? Maybe you don't have too much and things are good, but okay, genetics and inflammation. I know my genetic risk for being susceptible to toxic mold is high because of something called HLA-DR. Is that all we're talking about or is there a whole bunch of other stuff I had? How big of an influence is genetics versus, "Hey, you're smoking and you're drinking and you're eating French fries. You're inflamed." How big of it is genetic? How much of it is lifestyle?

Mansoor:

So there are other stuff, but remarkably and you know, Dave, I've started riffing on this concept and I'll throw it out there to the audience and throw it out there to you very, very quickly. When we first discovered DNA, oh, how many ever odd years ago now, probably getting onto 60 something, now 70 something years. When you thought of how complex living things were such that there was an encoding system for those living things, no scientist worth his or her salt thought that DNA was going to be as simple as four building blocks. If you thought of, "Hey, this is going to be the code that encodes the complexity of life as we know it," we would have thought the code was going to be much more complex. And it turns out to be this four letter, four nucleotide code.

Mansoor:

Where am I going with this? I have found, Dave, and this is just coming from the years and years, and this is something that I'm quite excited to share. What I've found is there's a remarkable analogy, the analogy of the simplicity within complexity or complexity within simplicity of the DNA code, as it pertains to cellular behavior, as it pertains to cellular function, i.e., you would think that there was this unimaginable multifactorial complexity that describes all of the nuances and the minutia of cell function. It turns out that there are really some fundamental building blocks and those fundamental building blocks of cellular function are simply recombined and matched and reused in different ways to accomplish, much like the four building blocks of DNA, there are building blocks of cellular function.

Mansoor:

Once we start appreciating that, per your question, how complex is this going to be? It's definitely complex, but it's complexity born out of simple building blocks. Let's apply that phenomena. So this is the concept of complexity within simplicity, simplicity within complexity. Let's apply this to cardiovascular disease and inflammation. There's a triangulation and the triangulation goes like this. The first thing that you need to know about cardiovascular disease is that most cardiovascular disease does not start with the heart. It starts with the lining of the blood vessels. It starts with something called the endothelial glycocalyx, otherwise known as the Teflon coating of the blood vessels, the inner luminal side. So the side of the blood vessel that is constantly exposed to the flowing blood, that endothelial glycocalyx, that's where most of the genesis of the initial formation of cardiovascular disease starts. So we've got to look at factors, and in this case, genetic factors that contribute to that blood vessel lining. Number one of the triangle.

Mansoor:

Second thing we've got to ask, what is the things in the blood that give rise to inflammation of that endothelial lining and damage to that endothelial glycocalyx? Because the blood is the living, breathing representation of you, your diet, your lifestyle. What are we talking about here? The surplus insulin, secondary to the surplus sugars in the bloodstream. The toxins, things like even the mold toxins that you breathe in and creates cascade reactions that end up in the bloodstream. Microbiome changes. Dave, would you believe that 80%, eight zero, of the metabolites floating around in a human's bloodstream? You'd like to think that 80% of what's floating around in your bloodstream came from your endogenous cells. They actually come from the microbiome cells lining the GI track.

Dave:

It's basically bacteria poop in your blood.

Mansoor:

There you go. And so the health of the microbiome equals, and traditional medicines have known this for ages, health of the guts equals health of the body.

Mansoor:

Okay. So point being we, A, look at the health, the resilience of the endothelial lining. B, we look at what is getting into the bloodstream to quantify the... or qualify that blood as being, is it pro-inflammatory? Yes, no. And all of the things in the bloodstream that can be pro-inflammatory. And then the third, to finish the triangle, we ask how good is the body at controlling those things that get into the bloodstream that would otherwise be inflammatory? How good is your insulimic control, i.e., your carb metabolism? Much of what we think of in macronutrients, it's not about the fats. You've pioneered this. You've forgotten more about this than I know, Dave. It's the-

Dave:

I don't know. You're a pretty knowledgeable guy, but thank you.

Mansoor:

No, it's true. Okay.

Mansoor:

And so what we're going to do is we're going to ask how good, and the genetics of how resilient your lining is, one. Two, less genetics, more environmental lifestyle, nutrition. What is getting into the bloodstream, such that it will impact the lining of the blood vessel? And then three, how are we controlling those things? How good is our detox system? Because of course, we talk about toxins and medications as well. And then how good is our anti-inflammatory genes such that if the blood became toxified, i.e., inflammatory, and therefore if, when we combine that to an endothelial lining, the endothelial lining is less resilient than we would like, we then get an inflammatory cascade. And that inflammatory cascade equals the start of cardiovascular disease. That's the slippery slope.

Mansoor:

Once we get there, we're going down a path that we don't like and, case in point, very pertinent to the discussion at hand, turns out that this nasty SARS-CoV-2 virus, it's much more than just a respiratory disease, so to speak. It has profound vascular inflammatory consequences. So the things that can inflame the endothelial lining of, the vascular endothelial lining of the body, they are toxins, they are medications, they are biologic, I.e., organic inorganic, and they're even microbial. And they're products of the microbia, i.e., a dysfunctional microbiome. So that's the structure. That's the triangulation. Once you understand, you see, no, we've gone from complexity, we've broken it down to simplicity, three moving parts. Now we're going to ask an intelligent question. What are the genetics that influences these three moving parts? And then you get this beautiful spectrum. You get to see, how are you in terms of risk? Not the silly, you've got a 10% or 20% increased risk of something that you already had a 10% increased risk of, but you get functional insights that are meaningful, actionable, and oftentimes reversible.

Dave:

All right. I want to recap that for people. We've got, you want to get exposed to less pollution because this is causing inflammation. You want to do something about the gut bacteria pollution, which is separate from environmental pollution or eating dirty food. You want to be able to detox things better based on your genetics or whatever else you do. And you don't want to be over-inflamed based on your genetics or what else you do. Okay.

Dave:

So what I just told you is basically the secret to being Bulletproof. My whole path to go from weighing 300 pounds with arthritis and cognitive dysfunction and all the other nasty diseases of aging you could say that I have. I eat a low inflammation diet that is free of toxins, and I have air filters and stuff like that. I bind my gut toxins and have fixed my gut bacteria, so I have fewer of them. I take supplements to upgrade my detox systems, and I talk a lot about that and saunas and all that kind of stuff. And then I use anti-inflammatory herbs and other nutrients to control my body's genetic inflammatory response.

Dave:

And that's taken me from basically a shit show from a health perspective in my twenties to where I am now. But I did it, until we connected, without some knowledge. Some specific knowledge about which of these was genetic. And I learned over the years, a little bit of this, a little bit of that. But I think you and I would agree those are the things to do, and you've actually kind of boiled it down. But you came to that from a looking at genetics and how the body behaves, which I love. Can you break down what you

would do as a practitioner, or to advise someone, for each of these different things that you've just spoken about? And then tell me the genetic components of each.

Mansoor:

So let's start where we started the triangle. The first thing, if anyone wants to begin a journey into their cardiovascular health, or on the flip side of the coin their risk for cardiovascular dysfunction, the very first thing I tell them to do is understand your individual resilience, i.e., your endothelial results. Understand whether you inherited the Williams Sonoma Teflon coated frying pan, or you inherited the dollar store Teflon coated frying pan, i.e., the resilience of that endothelial glycocalyx. And these genes, they're actually not even genes, Dave, they're genetic markers. They're named after the chromosome and the chromosomal location that they were first discovered, i.e., they're called the nine P, as in picnic, nine P two one. Standing for chromosome nine, the P arm, as opposed to the Q arm, and the band two one.

Mansoor:

What is famous about the chromosome nine P two one region? This region was loaded with genetic associations of all types of cardiovascular disease. So much so that this region of your genome, this part in volume nine of your human genome, gained the famous name of being called the heart of the human genome. That's how important these markers were. So you've got a number of these nine P two one markers, three of them in particular are extremely well studied. And here's how it goes, Dave. So each person has three of these nine P two one markers, three of them. When you and I first spoke Dave, we were looking at two of them. We've yet since added a third.

Mansoor:

And the beautiful thing of these three markers is this. Each of these three markers, you have two copies, one from mom, from dad. Which means you're going to have six copies of these markers. Each of these six copies of the nine P two one markers come in, either an A allele, A as in apple, allele, or a G as in George allele. Of course, because they are randomly inherited, that means that any one human being might have six As zero Gs, 5 As 1 G, 4 As 2 Gs, 3 As 3 Gs, all the way up to and including six Gs for these six markers.

Mansoor:

The more the number of Gs you have for these six associated points, the weaker is your endothelial glycocalyx. The more the number of Gs on the six G spectrum, the weaker your vascular endothelium, full-stop. So if you happen to be that rare, less than sevenish percent of the human population that has all As, you are the blessed individual. Individuals with all As zero Gs at these three nine P two one markers, study after study shows that even with a modicum of healthy lifestyle and diet at 80, 90, their blood vessels still look like a teenager.

Dave:

Right. Does this change based on your race? If you're-

Mansoor:

Excellent question.

Dave:

So if you're from Italy versus Africa versus India, is there any variance or is it 7% of all people?

Mansoor:

The 7% there, that's at a generic ancestral. So taking a generic population there does, as with many other things, there does seem to be some minor ethnic geographic ancestral drift in the people that might have the preferential, all As or the undesirable more Gs. However, it's not one of those things that is as standoutish as other genes that we can trace that have real preponderance per ethnicity. So in this, it's a pretty mixed bag. And there's some pretty interesting phenomena that can be raised with that point. However, to answer your point, to answer your question, we're not seeing huge genetic preponderance based on ancestry for these markers.

Dave:

Got it. So in the U.S. for instance, we know that the population with the highest cardiovascular and highest diabetes problem is black people as a percentage of population. However, those diseases are a hundred percent correlated with poverty, right? So basically the more money you have, the better food you can afford, and the less likely you have a problem, and it's not genetic. And so you're saying that there's no higher genetic risk, at least in these categories-

Mansoor:

In these categories.

Dave:

Based on race, but in other categories, there are all sorts of different races have strengths and weaknesses, right?

Mansoor:

100%. So the upshot is when we start this triangle, it's a fairly equal lottery grab. Where are you going to be on the six G spectrum? Zero Gs, you want to be. Six Gs, you don't want to be, not that you have much control of the matter. And what does that establish if you're a six G or let's say four G and above?

Dave:

Hold on a second. Are you saying that 5G is going to kill you?

Mansoor:

You just have to go there.

Dave:

Oh man, now the show's going to get banned. What are we going to do?

Mansoor:

There you go. You've just been flagged, my friend.

Dave:

Sorry. Keep going.

Mansoor:

So that's not the Gs that we're speaking of, listeners, we're speaking of these allelic variations, okay? Now here's the thing. You're a six G, you're a five G, you're a four G. Oh my gosh, I just found out that my vascular lining, my endothelial calyx, might not be as robust as I would otherwise want it.

Dave:

Do you have my results in front of you by any chance?

Mansoor:

I should. I always should remember to make sure and pull them up. And I usually memorize these, but for this particular let's go here.

Dave:

I remember we went through it a while ago. I don't think I was in a particularly strong situation here.

Mansoor:

We'll remind you.

Dave:

You can expose my weaknesses. I'm practicing vulnerability.

Mansoor:

Which guru have you been listening to get in touch with those inner feelings?

Dave:

I think that I've came across those in ancient Indian and Tibetan texts.

Mansoor:

So there we go. So Dave, drum roll, Dave Asprey. Okay. Dave Asprey-

Dave:

By the way, The DNA Company results. When we first went through mine, before we got to be friends, before I became a supporter of the company, I was really blown away. Because you were able to go through this and I have my full human genome and it just wasn't useful. And your interpretation here was really good, so.

Mansoor:

Well and you hinted at it and you hinted to your audience, and this is what makes Dave who he is. I mean, really the thing that tickles me whenever I got a chance to speak to him is, so there's a six G, yep. He's the guy on that part of the spectrum that didn't win the lottery in regard to this.

Dave:

Dude, I didn't win the lottery on all sorts of stuff, man. I tell you, all this Lyme disease and crap like that, and being prone to inflammation on top of this stuff. So...

Mansoor:

And by the way, Dave, so as much as the initial data and the initial studies focused on this being relevant to the inner luminal lining of the vascular, i.e., the vascular endothelium, my studies is as you know, thousands upon thousands of patients that we look at one on one with their clinicians, it is as equally important. Why should it not be? Because that endothelium and the cell migration that leads to the lining, whether it's the lining of the blood vessels or the lining of the organs or the myelin sheath linings, turns out to be equally as informative. So in other words, people with six Gs don't want to get Lyme disease or don't want to be exposed to black mold.

Dave:

Oh, yeah. I kind of noticed that. So you're saying the lining of the nerves is also controlled by these same six Gs?

Mansoor:

It's influenced. We're still looking at the actual structural influence at the endothelial glycocalyx. We have a much better idea of it, but as you know well, many of the somewhat of course important, but frankly, archaic ways of measuring neural pulse waves and neural connectivity are missing much of the inflammation and chronic inflammation that can occur at the myelin sheath, which is essentially the insulation that allows neural pulses to travel within the axonic lengths of the neurons. And we find that people that are the nine P two one six Gs, again, that inflammation, in this case the inflammation of the myelin sheath, in this case secondary to, what's the culprit? As you've pointed out, many people are missing the fact that they're exposed to mold.

Dave:

In fact, let me just tell people what it's like to have problems with myelin formation. I figured this out quite a while ago. Like wow, myelin is the insulation on the nerves that allows them to carry more current. When you practice, you grow more myelination. So a long time...

Dave:

... more current. And when you practice, you grow more myelination. So a long time ago, I said, "Well, apparently, I've had all this nerve pain. My back hurts all the time," always mediated by toxins. I tell people I'm a canary. Really, these are the genes that make me a canary. That's one of the reasons I'm a good biohacker. And I'll tell you if it works for a 6G, it's going to work for a 4G or a no G. I am the worst you can get. So the deal is, if I can lose a hundred pounds and keep it off and get younger, seriously, you're in the 93% of people, if we're going to play the odds, where you can handle it.

Dave:

But when you don't have the myelin stuff, and you have any sort of biotoxin accumulation that's fat-soluble like lime or toxic mold, very similar things, it gets into your myelin and myelination diseases are some of the things that kill you when you're old, a lot of the neurological stuff.

Dave:

Notice how the whole book Head Strong, the New York Times science bestseller, tells all about myelination and mitochondria in the brain because I had to hack it, right? And it's chronic pain. Even in

my twenties, I'd get this weird problems with nerves in my arms and my upper back and the cognitive things.

Dave:

And some of the things like the “Get Some” Ice Cream, which is a famous recipe on the Dave Asprey webpage. This is full of, get this, raw egg yolks and sunflower or soy lecithin. Why would you put those things in there? Because if you want to myelinate things, you need lots and lots of lecithin to form myelin. And when I started doing that, magically, good stuff happened, but this is not obvious, right?

Dave:

It's just like when you are in pain, because your nerves don't line themselves right, it makes you do stuff. And when you have the external things where I was like, "Wow, a black mold house and active Lyme disease, like I win." So that's what it's like. Anyway, keep going. But you're shining light on my path, which I think is illuminating things for a lot of other people.

Mansoor:

So you start there. You start with exactly what you have, through the years, your brilliance, biohacked. Biohacked because, and I think, I don't mean to put you on the spot, but sometimes we need the individuals who, despite being shortchanged in some ways, when coupled with their brilliance, they're able to say, "Look, I went and I fixed this." And as bold as your statement about beef, you can fix it. What about the guy who has the zero G's or the 2G's or the average 3G's, they should be able to fare much better or as well as you did.

Mansoor:

So now that we establish a really simple building block, just that one thing, and you can line up how that equals increased risk of all forms of cardiovascular disease, including forms of neural inflammatory presentations, as you said, whether you want to call it fibromyalgia, chronic fatigue, Lyme disease, mold, poor response to mold toxicity, at the functional level, we're speaking about the same thing.

Mansoor:

And actually there's a brilliant, brilliant MD, PhD, in other words, a medical clinical scientist, Dr. Amir Minerbi. I have no association with him, but I have no qualms in calling out his brilliance. He's at McGill University and what he showed was that conclusively to the extent that, I think it's in its either last stages or it's already been approved, the very first patented and approved diagnostic test for fibromyalgia.

Mansoor:

And what is it? It is a test that looks at microbiome imbalances. Because what is the connection? He showed that certain microbiome over preponderances and under preponderances of certain species, I cannot remember exactly which ones he looked at. What does that mean? That microbiome imbalance translates into you. Remember that 80% of stuff floating around in your blood that came from the mind, translates into the over preponderance of inflammates, what we might call inflammagens and overly inflammatory blood physiology, which then when they, through the circulation of the body, gets to those myelin sheaths contributes radically to the myelin sheath inflammation.

Mansoor:

So he's literally been able to show when he has conclusively, based on gold standards, determined that someone has these i.e. fibromyalgia, here is the microbiome profile that it matches to. You know what's the other link there, Dave? Why do more females suffer from fibromyalgia? Because one of the myelin sheath disruptors is estrogen metabolites. 4-hydroxy estrogen exerts profound, and here's the point, exerts profound vascular endothelial inflammation and myelin sheath inflammation, which is one of those missing links of why we have a preponderance of females with fibromyalgia. Okay. So we go back-

Dave:

Wow. That's cool. Oh, that's because the reason you know this is because you have a whole panel from the DNA company that's all about women and inflammation, which was our last episode, but we didn't cover that in the last episode. That's actually really interesting.

Mansoor:

It is. Okay. So now we go back to that triangle. We've established a baseline of what's your lining? What were you dealt? How sensitive or not is that vascular lining? Then we're going to layer in a few things. We're going to look into the blood, look into the blood, and we're going to start asking what is contributing to this person's blood profile, i.e. the inflammatory index.

Mansoor:

And we're not speaking here, for heaven sakes, this is not just about homocysteine as though that's the end and be-all inflammatory marker. We're speaking of intelligently. And I want to draw the work of a couple of incredible, and you will know them, again, this been a thesis of what you've been discussing throughout your career.

Mansoor:

This is the work of Dr. Ingerid Arbo and Hans-Richard Brattbakk from Norway. And what did they show? So here comes the point. They looked at various diets. And, in fact, I'm just going to fast forward to a statement directly from the study, and I'm just going to read it. They looked at diets. What's the thesis? What's the summary? Sugars kill. That's your summary. And how are they doing this? And how is it doing this? Via the medium of inducing insulin and the effect that insulin is going to have on the body.

Mansoor:

So let's read just two major direct quotes from the study. What they showed was when a person eats a higher carb diet, we're going to have to qualify that based on individual genetics, but let's keep it general for the time being. A higher carb diet or we can say it this way. A higher than tolerable carb diet for that person's genetics and lifestyle, the body then needs to produce a surplus of insulin. And what does that surplus of insulin do? Insulin is a hormone, and this is another topic that I've been focusing on, Dave.

Mansoor:

It turns out that when I speak to more and more doctors, they have no clue what hormones are. They don't understand that hormones are hormones because they do the job of what? Changing gene expression. The job of the hormone is to get into the cell via the receptor of that hormone, whether it's insulin to the insulin receptor, whether it's estrogen to the estrogen receptor, whether it's vitamin D to the vitamin D receptor because yes, it's a hormone. And then when a hormone mines its receptor on the outside surface of the cell, that docking station motif of hormone plus receptor, disassociates from the

cell membrane, floats across the cytoplasm, goes into the nucleus and does what hormones do, change gene expression.

Mansoor:

And what these researchers could beautifully in this study show when that insulin receptor enters the nucleus, the gene expression profile, in other words, which genes were turned on, which genes were turned off, mirrors, mirrors inflammation, such that the comment and here's the direct quote, "This affects not only the genes that cause inflammation in the body, which was what we originally wanted to study, but also genes," this here is the insulin and the insulin receptor, "This affects not only the genes that cause inflammation in the body, which was what we originally wanted to study, but also genes associated with the development of cardiovascular disease, cancers, dementia, type 2 diabetes," you literally listed it without any prompt. By the way, the audience out there, Dave and I did not rehearse this one bit.

Dave:

No, no, I never do that.

Mansoor:

And so the wonderful thing, there's just applying the intelligence that Dave has brought to his community, he listed quite frankly, everything here that these researchers listed in association with inflammation, in association with excess insulin, in association with a poor diet, i.e. one that was predominant or preponderant of carbs.

Mansoor:

And just one more last thing that they went on to say, and this I thought was really telling, what did she, Dr. Ingerid say, the primary author. She said, "This is not the kind of inflammation that you would necessarily experience as pain or illness," just FYI, "but instead it is as if you are battling a chronic light flu-like condition. Your skin is slightly redder, your body stores more water," I'm quoting directly from the study. "You feel warmer and you're not on top mentality. Scientists call this metabolic inflammation."

Mansoor:

And when you day in, day out, meal in, meal out, when you overproduce that insulin, which is really an adaptive response, it's something that was trying to, you should not be overproducing insulin. You have just, putting aside the microbiome toxins and the mold toxins and the medication toxins and the toxin toxins, just that choice, that one choice in your diet, made without thought, can introduce a type of chronic inflammation as was just described.

Mansoor:

Now, here is this. I want to end this on a positive note. They did something else, though. They then altered the diet of the participants. This was what made this study so beautiful. It was a large study. This is not your typical 10 people, 20 people. This was a large study conducted in Norway. Mind you, the Nordics have it on us when it comes to an understanding of [inaudible 00:33:02].

Dave:

And let's just face it. Norway has a trillion dollars in their sovereign fund. They can afford whatever research they want, and then they make fun of their friends in Sweden. My wife is Swedish, by the way, I just pissed off both sides, sorry, family.

Mansoor:

But here was the thing. And ending this on a positive note before we go to the next point in the triangle, what was brilliant about this, quote, "It took," and Dave, you're going to love this. And this is what makes some of the programs and your Bulletproof program so ridiculously effective. "It took just six days to change the gene expression of each of the volunteers." Six days. In six days, from a high carb, high sugar diet, that led to the gene, and in fact, even when they terminated the diet, such that it was just kind of we might call the run of the mill American diet, with a bit of bread here, a bit a pasta there, and so on and so forth.

Mansoor:

Please, I'm not shooting at all carbs. I'm not shooting at every little consumption of carbohydrates. I'm saying that we're doing things that we don't give thought to and none of our clinicians are talking about what does insulin actually do in the body. And so she goes on to conclude. "So it's easy to get started, but if you want to reduce your likelihood of lifestyle diseases," the ones that we've mentioned, "this new diet has to be a permanent change." You have to commit to not subjecting your genome to the insultery effects of insulin, turning on and off genes that are not in your best interests to be turned on and off in the way that insulin does.

Dave:

To be really clear, this does not mean keto.

Mansoor:

No, absolutely.

Dave:

For a lot of listeners, a lot of people took the Bulletproof Diet and said, "Oh, that's just keto." I'm like, "No, it helped make keto popular, but it is very clearly, there's times you're in ketosis, but it is a diet where you eat carbs for your gut bacteria. You cycle in and out of ketosis and you manage that stuff." And like this whole dirty keto movement is not good.

Dave:

And I would ask you a question and I know it's bad, but I don't know all the reasons it's bad. But when I was writing Super Human, low insulin was actually more dangerous than high insulin. Why is low insulin so bad? And this can happen on unending dirty keto diets, especially. What's the deal with genetic signaling and not having enough insulin?

Mansoor:

So first and foremost, to all of the listeners, I always am proud of being able to say when something is beyond my pay grade, this is one of those examples. But here's the little that I know of it. You see, because the adaption between insulin and glucose, there is a partnership there, there is a relationship that in order for glucose to be shuffled and where do we find glucose in the body? Well, we find this in

the bloodstream after a meal, for example, and we certainly want to channel some of that glucose into the cells of the body to enter into energy ATP reactions. Okay. But there's no place in the body for glucose to be stored. There are places in the body for fats to be stored. And of course we store quote, unquote "proteins" right?

Dave:

What about glycogen? You've got your liver and your muscles. Isn't that where you store glucose?

Mansoor:

There we go, but not as glucose.

Dave:

Okay. It's not stored as glucose because glucose is pretty toxic. Okay. I hear ya.

Mansoor:

There you go. That actually, so you jumped right ahead. So, what I'm saying is you literally can't go peek into something and go, "Oh look, that's the extra sugar there," as glucose, because what Dave just said. Because there's a toxicity, there's a love-hate relationship. Think of this, the love-hate relationships of the two things that literally keep us alive as human beings, oxygen and glucose, from that perspective, the two things that kill us.

Mansoor:

We breathe oxygen, but it's actually over time creating oxidative stress and oxidants. And to the extent that we need glucose or some preamble to it, is the very thing that we're going to need for energy but it also has this love-hate relationship in the body.

Mansoor:

So to the point, Dave, when there's not enough insulin to control the trafficking of that, otherwise, let's use the term, otherwise toxic source of energy, we now run into because we run into the glucose damaging the cardiovascular glycocalyx. Glucose is anathema. When that glucose stays elevated in the bloodstream, it literally acts and we can use the analogy, the visual, as sandpaper to that endothelial glycocalyx.

Dave:

People who are hearing you talk about that, if you've read Super Human, there's something I write about. It's called glycation or advanced glycation end products. This is what we're talking about. This is ` which is why spiking your blood sugar after a meal is probably not a good idea. In fact, if you're going to do it, you can block glycation with a whole bunch of stuff I write about.

Dave:

So I do eat sugar on occasion. I know it makes me a bad human being and all that stuff. But when I do, I'm pounding the cinnamon or extracts of cinnamon. I'm taking my chromium, I'm taking my vanadium and I'm taking carnosine, which is an amino acid that blocks glycation. That doesn't stop the bad insulin stuff. The cinnamon will help with the insulin spike. So I can have a few carbs, a little bit of sugar on occasion, but I'm blunting that because I'm actually in the weak bucket.

Mansoor:

And what Dave has just done there is he's simply, his weakness put aside now for the time being, he's protecting himself from the ultimate bad guy or the consequence of it, which are these gaps, these glycated end products. And when you, and Dave, I'm going to borrow that from you. I've never heard it said that way, but visually it just snaps. When you brown, with that sugar browning effect on the lining of the vascular endothelium, that's exactly the image that you want your listener to bring about.

Mansoor:

So there we have it, what the point here is not talking about ketosis and staying in it. What we're trying to bring to the audience is understand. Don't just think, "Okay, I'm going to eat that slice of bread." Fine. Think of, visualize what is happening in the cascade of events when you elevate your blood sugar. What are the blood sugars doing?

Mansoor:

Is your liver health at the level that you can store that temporary glucose as glycogen? Or is that liver already fatty? Has it already given up the ghost because of over alcohol consumption or other reasons for fatty liver disease, which by the way, has strong genetic correlates such as poor detox genes. Because of course, if the liver it's other primary function or its primary function of detoxification, if it is struggling because of deletions in the GST family and so on and so forth, they are strong predictors of increased risk of things like NAFLD and NASH. And when that liver becomes fatty, do you think it's going to have the proper control mechanism of temporary storing excess glucose in the bloodstream as glycogen? We now create a cascade.

Mansoor:

So we go from what's the lining of the blood vessel or the resilience of it, to asking what are these factors? And so when we speak about the insulin, now we get into, Dave, you've asked TCF7L2, that wonderfully well studied gene that controls the body's response mechanism to insulin, i.e, this gene has a famous SNP variation within it. Comes in a G version and a T version, T as in Thursday. And if you are a T carrier of the TCF7L2, Thursday, Charlie Friday seven left to two. If you carry the T alleles, you are at a considerably increased risk of insulin resistance.

Mansoor:

Now it's remarkable to me, Dave, how many people, including clinicians, either misrepresent or simply get insulin resistance wrong or you're not understanding quite what it means. When you're insulin resistance, you get a very vying post-meal phenomenon. Here's what happens. And that's why you have to look at your numbers, your testing, and what you're looking at to determine where you are in the genesis and in the consequences of insulin resistance. You've got to look out for this.

Mansoor:

When a person is insulin resistant, what does it mean? It means that the insulin signaling on the insulin receptor to cause the cellular changes that we previously described is not happening efficiently. So when that person's blood sugar goes up, here are the two counter things that happen with insulin resistance. A person that is insulin resistant shortly, shortly here means usually 30 to 45 minutes, after a high sugar meal, they become hyperglycemic while being [hypoinsulinemic 00:20:13]. So they're in a hyperglycemic mode and hypoinsulinemic mode.

Dave:

What that means in just plain English is you have a lot of blood sugar, but you don't make insulin to pull it out. So the blood sugar just sits there and cooks you, marinades you.

Mansoor:

Browning the lining, as Dave previously said. Then what happens is as the body goes through that phase, and is now going into [inaudible 00:42:31] inflammation, inflammatory signaling going on, secondary to burning the lining as Dave has beautifully pointed out. Now the body switches, and it sends an overly urgent signal to the islets of Langerhans, that part of the pancreas that's going to produce the insulin. And now it shoots up the insulin levels.

Mansoor:

So now you quickly become, quickly here in that phase, 45 minutes into the game, you become hyperinsulinemic. And what happens with your blood sugar? It plummets, which of course, in that 45 minute, one hour off for that individual's meal, now they've got the munchies. Now they've gone from the hyperglycemia of woo-hoo, and now their blood sugars plummet. They're in a hypoglycemic mode, hyperinsulinemic mode, which of course is really getting that gene expression profile that we're talking about.

Dave:

This is great. You can get the damaging effects of insulin or the damaging effects of sugar. You're actually going to get both if you have the T gene. How am I doing on that gene? My DNA Company results.

Mansoor:

Let's look it up.

Dave:

I love it. I feel like I'm walking around with my pants off here.

Mansoor:

You asked for it. We're going to [crosstalk 00:43:50]-

Dave:

Actually, come to think of it. I'm just filming from the waist up.

Mansoor:

Please, don't. No, no, no, no, no. We don't need that. So, Dave, thankfully, he's got the double G, so you're a double G on that. So for the listeners out-

Mansoor:

See, you're a double G on that. So for the listeners out there, you've got a T and this is the most studied snip for those who are really note taking. This is the rs12255372.

Dave:

What percentage of people? We said 7% are in the really garbage bucket for the other one. What percentage of people have the TT on this one?

Mansoor:

No, this is hugely ethnically biased. Very, very ethnically biased, okay? And so for this, you have huge skews in the preponderance of this, the T-allele of this gene, and actually for the Western Saharan Africans, which make up ... the Maghreb ... the Western hall, what would you call that? West Africa. And then you start going into the Southern, along the Western coast, which makes up most of the individuals that were ancestrally brought to the U.S. The preponderance of the T-allele is significantly higher.

Dave:

How much, significantly higher?

Mansoor:

As many as 40% of that population carries at least one T-allele. And here's the thing, just even carrying, in other words, being a GT, the T-allele, [inaudible 00:45:24] Dave, again, your mind, you might be able to riff on why this is the case, the T-allele, which is the at risk allele, at risk for insulin resistance, is dominant. So even if you're just a GT, you are going to be at risk as much as a TT for insulin resistance. Now why in God's green earth would the gene version that leads to insulin resistance be the dominant version of the gene? Well, that enters all sorts of thoughts as to what we were really designed for eating wise and dietary wise, which I know you've talked about in the past.

Dave:

So whether you have TT or GT your risks of essentially diabetes then, and all of the side effects of diabetes like cancer and heart disease, are substantially higher. How substantially higher?

Mansoor:

Well, of course now it gets to the diet. Are you feeding? So that's the point and the silver lining is, okay, so you've got your insulin cascade control is not as equilibriumed as you would like, but what was the trigger for it? The trigger for it was the glycemic content of the food that you're eating. And again, please, this is not to argue for ketosis or not. It's just to say that you need to know, are you a T carrier of this TCF7L2? And if you are, if you are going to assume as a T carrier of TCF7L2 you can follow a, you know, eat all carbs, but eat no fats type diet because some new guru said, "Eat all carbs is the best thing since sliced bread," pun intended-

Dave:

Oh, you mean like the vegan diet, that one?

Mansoor:

You called it. I was trying to be diplomatic.

Dave:

I was too, the vegan diet is bad for you. Isn't truth diplomatic?

Mansoor:

Potatoes-

Dave:

Look at you dodge that, that was so elegant, the way you just danced around that. I admire you.

Mansoor:

Potatoes are vegan and beer is vegan. I saw this article, that [inaudible 00:47:28] advertisement somewhere that says, "Vegan diets are healthy, but potatoes? We're not jumping in potatoes. We're not killing... ". You know, but look, what does it mean? What is the vegan diet? And please I'm not-

Dave:

It's carbs! Okay, it's carbs. That's what a vegan diet is.

Mansoor:

You're going to have to be full somehow. Okay? And unfortunately-

Dave:

What are beans? Are beans high in protein? No, actually beans are high in carbs, and they have tiny bits of protein that's inflammatory. Oh, there's that.

Mansoor:

And let's not get into the skin of beans and [crosstalk 00:48:00]

Dave:

And the proper preparation and all that. Okay. But so what you're arguing here then is ... what you're saying is 40%, at least of the most common African heritage people in the U.S. have at least a T here, 60% of them don't, right? Okay. And what about say if you're from Asia or you're from India, or if you're from Europe, what's the percentage?

Mansoor:

The T-allele, much less prevalent amongst, for example, the Han Chinese population, okay? Fair enough. We're not going to comment on dietary preferences.

Dave:

20%?

Mansoor:

It's less than 20%.

Dave:

Wow.

Mansoor:

[inaudible 00:48:46] the T-allele in the Han. We're not speaking of the Uyghurs, we're speaking of the Han population. [crosstalk 00:48:54]

Dave:

There's many different races within China, right?

Mansoor:

Now, remarkably in the ethnic ancestral South American, and we're speaking of the indigenous South Americans, the T-allele, South Americans that actually I can just say Indigenous Americans period, because across many of the indigenous, both in North America and through central and South America, the T-allele is again, at varying degrees, higher than the average population. And this is interesting because these were people that when they ate carbs in their indigenous ancestral diets, okay, fair enough, they ate carbs, but what were they? They were super fiber, high complex carbs. Super. And by the way, the corn that they ate is not the yellow, white corn that we eat today. And it's not the yellow, white potato that we eat today. So you take a people that, yes, they had carbs, ie starches, i.e. high complex fiber starches in their indigenous ancestral diets, and they're T carriers. Fair enough. You convert those people on to cornstarch sugars, you know, Monsanto corn and potatoes. Oops, I said the bad word there, but fair enough. Okay? What are you going to expect? And what do you see is the incident rate of type two diabetes of that population?

Dave:

Where I grew up in New Mexico, man, I mean, diabetes was rampant with all the indigenous people. I remember my friends across the street growing up, it's just like the whole family, obesity was there. And you're saying a lot of this has to do, because it's basically carbohydrate tolerance is actually harder for people if you're indigenous, or if you're from those parts of Africa, right? Okay, what about, I guess we haven't talked about like Japanese, Koreans, and we haven't talked about Europeans. What are the differences there?

Mansoor:

Significantly also lower preponderance of the T-allele amongst Japanese. When we say Koreans, we could just say Koreans, okay? Not the same when we get into the Malay, Philippine, Indonesian, that's different, okay? So there are ancestrally different divergences there. And interestingly, but not surprisingly, you then start to see another spike in the T-allele, though not to the level of the West Africans, nor of the Indigenous Americans. You start to see a spike, certainly above those Asian ethnicities of the Japanese, and the Koreans, and the Han Chinese, amongst Nordic people, speaking to the fact that Nordic and North Europeans, as opposed to Southern Europeans. So Western and other Europeans, you do start seeing a higher prevalence of the T, again, indicative of an indigenous diet that was very likely, not very high in simple carbs.

Dave:

Pretty much seal blubber and fish is what, going back to Norway and Sweden, when you get pretty far up there, there's not a lot of corn growing there.

Mansoor:

Indeed.

Dave:

Okay, and then Southern Europeans, I'm guessing, are little bit more carbohydrate tolerant. So they would have a lower incidence of T?

Mansoor:

Exactly. But then you get what we'd call the meeting of the seas, right? Because you start getting now an induction of, for example ... well, it's not the best term, but the Khaleeji Arabs. So the Indigenous Arabs of the Arabian peninsula, they rival the West Africans with the T allele.

Dave:

Really?

Mansoor:

Yes.

Dave:

Well, okay. I was just over in Oman and UAE, I'm in Dubai. I did not see a lot of obesity.

Mansoor:

Where were you in that part of it? Because you know-

Dave:

Granted, I was in Dubai. It's one of the poorer countries in the world, and it's also highly international. I'll give you that. But even in Oman, I didn't see a lot of it.

Mansoor:

So two quick things there, the Omani population amongst the Arabs has a much, much higher South Asian genetic group, and much, much, much higher. And you see it in their features and so on and so forth. Here, when we're speaking of the Kuwaitis, the Saudis, the UAE, but remember you go to UAE-

Dave:

Yeah. It's a problem in Saudi. Okay, I got you.

Mansoor:

Hugely, hugely. In fact, I met with the minister of health from the United Arab Emirates many years ago. I was his guest many, many moons ago, and he said, "Dr. Mansoor, there are five things that kill our ..." and we're speaking here of the Indigenous UA's. Remember of every 10 people you see in UAE, one is an actual indigenous UAE person. Nine are some other international [inaudible 00:09:34]. So we're speaking of the Indigenous UAE, the pearl fishermen of the day, and women. And he said, "Out of the five things that kill us, Dr. Mansoor, you might be able to help us with four of them. You're not going to be able to help us with one of them." I said, "What's the one of them you don't think I can help you with?" He said, "Car accidents." I said, "Okay, we'll put the car accidents aside." The other four things at the top of the list, cardiovascular disease, he separated it from obesity. So cardiovascular disease, obesity, diabetes, and cancers.

Dave:

They all go together. Like if you get diabetes, you're going to get one of the other three. It's just a matter of which one you get first.

Mansoor:

Indeed. And that's why I said, he separates them. Keep in mind, this is many, many years ago. We understand, again, this is what why started the conversation today. Inflammation, inflammation, inflammation.

Dave:

Wow. I actually didn't know this about the incidence of the different T and the CF7L2 gene, about how incredibly based on your history it is. Now we can say that, but anyone who's done a genetic analysis, like the stuff that you guys do at the DNA company, you can determine, "Oh, I thought I was mostly British, Welsh, and Irish and stuff," but it's like, "Oh, there's a surprising amount of Basque. And you know, a tiny bit of indigenous people were going back to the 1700s. And I've got a little bit of this and 0.1% some weird nomadic tribe in Siberia," right? So you can pick up genes, because we'll say there are very few people listening to the show who are one ethnic heritage, even more than 90%. It is pretty rare unless you're ... certain Jewish sects are like that. Like where you have your 99.5% or something because of practices there.

Dave:

So let's assume that we have more in us than we think we are. When you have parents and one of their parents has the G and the T and the other one has a G and a T what are the likelihood that you're going to end up-

Mansoor:

So then now we come down there, that is Mendelian genetics at that point in time. If both parents are heterozygote GT/GT, you have a 50% chance of being GT like them. A 25% chance of being a TT homozygote for the bad critter, and mind you, you have a 25% chance of being better than both of them, better here being a GG. So 25% GG 25% TT, 50% GT, but mind you, remember the 50% that's a GT, the T-allele seems to be dominant. So really what you have is a 75% chance of inheriting that naughty phenomenon of insulin resistance.

Dave:

Okay. Why are there any GGs left? You'd think after 25 generations of 75%, that we'd end up where pretty much everyone has a T.

Mansoor:

That's a brilliant question. Well, you'll always have that 20 ... in an equally mixed population, which, it's still not a milieu as yet, we're getting closer and closer to that mixed milieu, but there's still pop ... and even in the mixed milieu, we'll continuously keep generating a 25% group that will be GGs in a mixed milieu, which of course, you know, that GG population feeds back into. Now, we start to get into all those GGs carrying a better adaptive advantage in terms of not developing-

Dave:

There you go, they live longer. They have more kids. All right, that balances things out.

Mansoor:

Here, we really cannot overstate this. And again, you've beautifully clarified things. We're not talking here about extremes of any dietary choice. In fact, I'm not even talking about dietary choices. What I am talking about is understanding, when you make dietary choices, you are making a choice of what's going to happen to the lining of your blood vessels for the next odd number of minutes into hours. Think of it that way. And you are creating a cascade of genetic ... You are changing the way your genetic manual is being read, i.e. expressed, over several days.

Mansoor:

When we don't think about things this way ... and David, here's something. I'm sorry, I was really trying to control myself and I was not wanting to go here, but you've encouraged me to do this along the way. I can't hold my tongue anymore regarding vitamin D. Isn't it amazing, David, all of the gurus, of the medical gurus, who'd been hoodwinking people, telling them vitamin D is not a big deal. You don't need vitamin D. There's no clinical evidence that vitamin D is really-

Dave:

They're crazy pants. If you can look at anyone and say there's no clinical evidence, it's because you have the unique, it's probably genetic, the unique ability to not see things you don't like. Is there a gene for that? Have you guys discovered it yet? I call it the douche bag gene.

Mansoor:

I was going to use a choicer word. Let's keep it a douche bag. Now those same medical experts are now coming out, telling the world post Covid, "You know what? Maybe you might want to get your vitamin D. Maybe you might want to take a couple of thousand and not the recommended four to 600 IUs." Because, by the way, we find out what? Here comes the point. Everything I just said about insulin, the expressive cadence of the human genome, that when it's exposed to that insulin, insulin receptor gene expression, it's as though vitamin D does the exact opposite. Vitamin D and its receptor, when it enters the nucleus, creates a gene expression that is incredibly anti-inflammatory, which of course is why we now see that vitamin D is being used clinically to treat cytokine storms, the very epitome of inflammation in its uncontrolled manner.

Mansoor:

I'm sorry, but the hypocrisy of feeding the population, "Vitamins, there's no value to them." We stopped even screening [inaudible 00:00:59:59], at least in Ontario. We no longer even pay, forget universal medicine. We no longer even think of doing vitamin D tests anymore, because, by the way, it's not important. And now it took one virus to come along. And now they're all out of their closets going, "Oh, no, vitamin D is important."

Mansoor:

What does vitamin D do? You know those genes, we're talking about how hormones turn on and off genes, vitamin D turns out to be the promoter, regulator of your anti-microbial peptides. Your AMPs. Alexander Fleming, when he discovers AMPs two years, three years prior to discovering penicillin, he considered AMPS an almost more important discovery than penicillin. But of course you could patent penicillin, and the pharmaceutic companies took penicillin and ran with it, and everybody forgot about

AMPs until recently with the viral challenges that we're facing. Because AMPs, these antimicrobial peptides are awesome first-line defenses against viruses. Lysozymes, by the way, lysozyme is an AMP, okay? And the point here is that all of this, and there's probably something North of 75 different AMP genes, the genes make the antimicrobial peptides. And guess what? The majority, if not all of them, their promoters are turned on by vitamin D, okay?

Dave:

So these, if you have enough antimicrobial peptides, you're less likely to get sick from any cause?

Mansoor:

To begin with your anti-microbial shield is just that much better. I'm not riff. There was a point of the riff. The point of the riff is look at how hormones, vitamin D is a hormone, and its receptor insulin is a hormone, and it's receptor, how do they manifest literally polarized expressions of the human genome? One is pro-inflammatory, the other is anti-inflammatory. And it was just a riff, mind you, I couldn't help myself, as to the ridiculous nature of how many patients and colleagues and clients that I interact with that they go, "Yeah, but vitamins don't work. There's no point in taking it."

Dave:

You know what happened, Mansoor, is back in medical school, they had to take the Hippocratic Oath, but they weren't listening, and they thought it was the hypocrisy oath. And they've been operating on this ever since, because they thought that's what they were supposed to do.

Mansoor:

I'm going to be very careful, but it's a sad reality.

Dave:

I'm just going to say this, and I'm married to an emergency room doctor, and I have many, many functional medicine, other doctors who are friends and followers and all this stuff. But if you are one of the doctors who says vitamins don't work and don't take vitamin D, A) You're probably not listening to the show right now, but B) Seriously, like get off your high horse. You have been proven wrong, and saying it more doesn't make it true. It has nothing to do with reality. So yes, some vitamins like synthetic vitamin E in low doses, short term studies don't work. And it's probably bad for you, but that does not mean that all vitamins don't work. It's just not science to say that. So I'm a hundred percent with you, especially vitamin D3. It is one of the most important ones.

Dave:

And it's important for another reason. So we were just talking about the differences in racial distribution, right? Okay. So, and this is something that's been a part of the bullet proofing, it's in the bullet proof diet, it's been on my blog for 10 years. If you have dark skin, and this can be, if you're Arab, if you're Indian, if you're from Africa, it doesn't really matter. The darker your skin, the less vitamin D you're going to have, unless you're outside in equatorial sun for long periods of time, which pretty much no one is these days. So that means your risk goes up. So you have less antimicrobial peptides. You have less of the anti-inflammatory stuff that vitamin D does. And it's such a problem that even sometimes people with darker skin, their babies get rickets today.

Mansoor:

Today. This was something we thought that was a disease in ancient medical textbooks. Rickets? That's like scurvy, you know, scurvy and rickets were the things that you ... Well, because of this completely backward perspective that no, if it's a vitamin and if it's just easily available, it couldn't be important for the body. Nothing could be further from the truth. And of course, Dave, maybe on a different topic, we can talk about the genetics of vitamin D. It's conversion. It's transportation. It's uptake into cells.

Dave:

Yeah. We don't have time in this episode, but maybe we should do another one just on vitamin D.

Mansoor:

Just on vitamin D.

Dave:

Because you went through mine, you explained a mystery for me, because I test my vitamin D and it is so hard for me to get my levels up. And you went through mine and go, "Dave, yeah, you have the vitamin D receptors of a Pacific Islander, but you have white skin and you live in Canada, you're screwed." And that's why I have to take 15,000 IUs a day in order to get my vitamin D levels up to where they should be. But normally that's too much, but not for my genetics. And I have to take it twice a day and all, and it was the DNA company test that gave me that results. It wasn't 23 and Me, it wasn't any other analysis, even in my full human genome that did it. That's why I'm a fan of the way you think about it and the way you write up your reports.

Dave:

On the cardiovascular risk side of things, you talked about endothelial lining and, you know, I'm in that 7% worst risk profile. What are the things that you can do lifestyle wise to deal with genetic weakness around cardiovascular stuff?

Mansoor:

And really, I'm not saying this because I'm on the Dave Asprey show. All of the listeners out there, I'm a genes guy., Someone like Dave, he's spent his career, his intellect on bio hacking this. So Dave, you're going to know more about this than I do in terms of what you can do, but let's just start. The first thing is, well, don't abrase the thing that you just found out.

PART 3 OF 4 ENDS [01:06:04]

Mansoor:

Well, don't abrase The thing that you just found out. You picked up the dollar store frying pan, okay? You've picked up a dollar store, Teflon-coated frying pan, because that's all you can afford at the time. Prudence dictates that you're going to use that frying pan with a degree of care and a degree of being more aware than you would have been really good quality.

Dave:

You're saying because your lining is an easy to scratch frying pan, don't abuse your lining, okay? What does abusing your lining look like?

Mansoor:

It includes anything, which is the point we're going at, anything that when it gets into the blood, you have changed the inflammatory nature of that blood. Anything. We'd started riffing on, well, of course, anything that gets that glucose-insulin paradox skewed either way. Obviously it includes all of the toxins that you can think about. And of course the things that get into the blood are not just the things that you put into your mouth. It's the things that you breathe as well. It's the things that enter the bloodstream through the skin. So in other words, being not hyper-schizophrenic aware, but being intelligently aware of what you're exposing your body to.

Dave:

All right. I have a thing that you might know about, but that most listeners don't. We all know that smoking is bad for you, but did you know that breathing the air when you are frying stuff is probably more inflammatory than eating the stuff you fried. So the rule in the Bulletproof diet is don't eat fried stuff anymore. It's not food. But don't breathe fry air either because it's that inflammatory systemically. And it's those little things that really matter.

Mansoor:

100%. How many times have I in the past, and I [don't know 00:01:42], manufacturing, if it's gotten better, your mattress choices, something that you're putting your nose on for eight hours, you're breathing in. And of course we all know, but our health agencies and our regulatory agencies, they're looking out after our best interests. And of course they're not going to approve something that is not healthy for the human body. Think again, okay. So be more [crosstalk 00:02:03].

Dave:

Okay, quick riff there. In California, they require something like six pounds of flame retardant to be added to a mattress. And because California controls, just because they have such a big market, what people do, most of the other states, you're going to get huge amounts of flame retardants and toxic foam in there. So you have to go for a flame retardant-free mattress, unless you still smoke in bed, which is no one.

Mansoor:

Right? So beautifully said, and again, these may seem trivial, but you asked me the question. You says, "What can we do, Mansoor?" What we can do is take a list of the things that you have control over and recognize that your... Here's the point that I asked. Your blood. When you get up in the morning after a night's sleep, the quality of your blood is not the same as the quality of your blood after breakfast and sitting two hours in a traffic. Your blood represents all of the things you're being exposed to. So be cognizant a degree. Think, if you carry those Gs, what is my blood and what is in my blood doing to the lining of my blood vessel right now? Okay. So avoidance. An ounce of prevention is worth a pound of cure.

Mansoor:

Now let's flip it to where I mentioned earlier to your audience. You're going to know more about these things that I do. What can we do to protect that glycocalyx? And there are a number of micronutrients that... Please don't put me on the spot, Dave, that's not my subspecialty. You've studied more of these than I can even imagine, but there are a number of amazing micronutrients, wholefood nutrients that you can, A, do two things: use nutrients as a way of, I'm going to use the word very carefully, purifying

the blood. So in other words, use nutrients as a way of bringing down the inflammatory potential of the blood, and also use nutrients as a way of rebuilding the glycocalyx. There's actually a lot of good studies out there on things that can actually rebuild your glycocalyx. In other words, re-Teflon the lining of the blood vessels.

Dave:

Of course, activated charcoal doesn't enter your blood. So the [mechanism, 01:10:17] action, you get less crap in your blood, but it won't pull out the crap in the blood, other than some certain charged molecules that get attracted to it. Certain mycotoxins. And so those are things, then, things like coenzyme Q10 and vitamin E, and there's a bunch of different polyphenols. And the list goes on and on, this isn't a full podcast for what to do.

Dave:

But, okay, when I send my sample, just a spit sample, into the DNA company and you and your team take a look at it, I get my report back and you tell me, "Okay, here's all the things," I can tell you that knowing that I'm in that GG thing, which you explained in the same exact way that you explained it to everyone, when we got on the phone for my test results. This was not a podcast. This was just me as a test customer to see if I wanted to even talk to you guys. I was blown away, because I know that I'm a high-inflammatory genetic type, and I know certain different genes, and I know how to turn off most of the inflammatory stuff, because I feel like crap if I don't. But I always felt like, "Why am I fighting a battle on 10 fronts? And I have these friends who have abs and they just eat shit," right? But you're saying, "Oh, those are the people who are AA, not GG. And they're good to go. Or was it TT?

Mansoor:

TT. So the As for the [inaudible 01:11:37] , they could have the... And then for the TCF7L2, they don't have that [tier LEL 00:01:11:43], their insulin control is... And mind you, there are other... Obviously, it's not just one gene, there are other pathways, and there are other factors that influence.

Mansoor:

But we're speaking here of how you can at least start with building blocks and why, if we control... Here is the point. If our listeners come away with something, it's this, Dave. There are two things before I forget. I want to shout out, I have no association with them whatsoever, Dave, none whatsoever, other than I use their product. It's a new product called Arterosil. Arterosil.

Dave:

How do you spell it?

Mansoor:

A-R-T-E-R-O-S-E-L. Arterosil. In fact, I'll just give you the accurate spelling so that everyone here... I have no... A-R-T-E-R-O-S-I-L. Not E-L. S-I-L. Arterosil. It's only, unfortunately, available in the US for the time being, and it is an incredibly clinically-studied formula. It's a formula of actually...

Dave:

It's a green seaweed thing.

Mansoor:

Yeah, there you go.

Dave:

I have looked at this stuff at the American Academy of Anti-Aging Medicine. It's interesting.

Mansoor:

There you go.

Dave:

There's also a brown seaweed extract that's very, very powerful for this stuff as well.

Mansoor:

This is new to me, and this is about those glycosaminoglycans which are the things that will be rebuilding that almost gel matrix, what we're calling the glycocalyx, i.e. the Teflon coating. This is new to me, but the studies on this blew me away to the extent that I am taking it myself, just FYI.

Dave:

I took this stuff for a year after I went to a forum and then the supplier I ordered from, I'm up in Canada, my supply chain broke. But, yeah, I absolutely was on this stuff. I should probably add that back to my stack. By the way, this is the problem with 150 supplements a day. [inaudible 01:13:39] that's a good one. And then it falls off the shelf and then you realize it a year later. Thank you for the reminder. And I have no financial interest in the company. I just saw their doctor give a talk, and I was like, "Oh."

Mansoor:

Neither do I. And this is one of those where they've really taken the steps to go into clinical trials, well-structured clinical trials. The specific compound is something called a [remnant 01:14:00] sulfate, being that extract. I think it's the monostroma something seaweed. Anyways, it's a seaweed as Dave has pointed, the green seaweed. And there's an extract of that green seaweed that remarkably resembles the building blocks of the glycocalyx. The clinical trials, their publications. This is when the company does it well. So I'm very impressed with it. I have no association with them other than I take the formula myself. Very very...

Dave:

Are you Gigi? I am a 3G.

Mansoor:

3G. Okay. So you're not that risky, but you're kind of average.

Mansoor:

Now my wife is a 3G as well, and so my 18-year-old daughter and my sons became 3Gs, per the statistics, but my 18-year-old daughter, she's a 6G like you, Dave. And I actually got this for her, but I take it as well. So my 6G daughter is taking this.

Dave:

Okay. That is fantastic, and I love it when we talk about some sort of new research from a company that neither of us has a relationship with, but is just doing good work. And I think people listening don't understand. It's really easy to go out there and just throw a bunch of stuff together and launch a new supplement and then buy a bunch of keywords and stuff. The problem is that you're probably not helping anyone if you don't do that. And so it's a lot of work to build a supplement correctly. And I looked at the studies and this one, I was sort of blown away. They treat it like a pharmaceutical drug.

Mansoor:

They do. They do. And they're very, very fastidious in even who they would partner with. So putting that aside, here was that take-home. I know that we're getting to that point of wrapping up for take-home. I want our listeners to take a step back when we think of our cardiovascular system. And we think of it as just this somnambulant plumbing through the body. A, that's not the case. It's living, breathing, constantly in touch with... At every waking, breathing moment of our lives, our blood, and that which is dissolved in the blood, the good, the bad, and the ugly, are swishing past the inner linings of our blood vessel. Think for a moment what that's going to do to the inner lining of your blood vessel, depending on where you are on the 6G spectrum. Number one. Think for a moment that the things in your blood have vastly different inflammatory capacities, and just let that register for a moment.

Mansoor:

And then finally, the main thesis of everything I wanted to accomplish today, Dave, is let's start re-evaluating the way we think of foods. The average person, including the average clinician, we think of what we eat as just combustible energy, creating it. You eat it, you burn it, you metabolize it, you excrete it. Many of the things we eat will create epigenetic gene expression changes that can last for days after eating that thing.

Mansoor:

Start thinking of your foods not just as an energy resource or a building block resource. Start thinking about what your foods are doing to your gene expression signature, and whether you are literally signing, pun intended, an inflammatory few days to come. And then the aches and the muscle aches and the neural aches and the joint aches, which any person who's in touch with their body. We know this, Dave. We know that we can, if we're in touch, we can say, "I just ate this and, boy, am I going to be getting that knee pain, that lower back pain." We think of that pain as though it's just some sort of localized joint inflammation. Not at all. It's not. It's just a manifestation that you can observe as inflammation, but when that knee pain, back pain, pain pain is happening, you are equally destroying the lining of your blood vessels. That's the thing that I wanted our listeners to really be able to understand.

Dave:

So that is the link between chronic inflammation, neurological pain, chronic joint pain, and cardiovascular risk.

Mansoor:

And by the way, we can add cancers to there, Dave.

Dave:

Of course. We can. So I'm a true believer in short-term stuff works, but we didn't mention fasting. So intermittent fasting is something that it's been a core part of Bulletproof. The book came out in 2014. On the blogs since 2011, it's been, "Hey, you should do this, or even longer fasting." And guess why? Because nothing inflammatory goes in when you're fasting. So it's the lack of bad stuff can do as much, or maybe even more, than the good stuff that happens when you fast. And I think it's the sum of the two, but what you're saying there is, "Don't eat crap, because it's such a big deal."

Mansoor:

And here's a little inner secret lining of intermittent fasting. If you understood that when you last ate the crap, you've created a gene expression wave. Some genes that you don't want turned on have been turned on. Some genes you do want turned on have been turned off. This is a wave washing over your genome. What does intermittent fasting do? It allows the wave to just die out. It allows that wave, it does not refuel the kinetics of that wave. So intermittent fasting works to do what? Of the many things it works to do, it works to reset your gene expression signature to a healthier gene expression signature.

Dave:

There you go. So I think we're there. I want to be really clear, too, on The DNA Company analysis of my genetic risk, which really drives for me what's important. It changes your whole perspective, because if you're walking around saying, "Oh, I think that what matters for me most is, oh, blood sugar. Because I'm really going to focus on that," but you find out it's actually the lining of your arteries. Or maybe it's both. Or you find out, "Oh, well, I know because of my genetic background that I might have a much higher risk of this. So I'm going to focus on that." It'll drive the data you do, or maybe it will just drive your behaviors, even if you're not going to go out and buy a gadget to monitor it.

Dave:

So I have this thing on the back of my arm right here. It's a continuous glucose monitor, right? Why? Because I know it's important. I'm an anti-aging guy. How high does your blood sugar go after a meal? And I know if I'm going to eat some carbs, I actually take the stuff so I can blunt it, and then I see if it worked. And if it didn't work, I take more of the stuff that blunts it, because why take the hit? You don't have to take the hit.

Mansoor:

And what it does though, Dave, it's a sense of empowerment, but it's empowerment with actual data. And I think when more and more of the population, the audience, the listeners, when they can see, this is not ephemeral guesswork. These are things that you can make intelligent decisions based on your innate genetic makeup. Make purposeful changes, all within that which is healthy, but individual to you, and actually, as you said, monitor things. You don't have to monitor. Viewers out there, you're not going to get too many, I'm sorry to put them on this, you're not going to get too many, Dave Aspreys. Look, that's just a reality. You don't have to be Dave Asprey every single thing that... He's doing the work so that the rest of us mere mortals can choose which aspects we want to focus on the most. And I think that's the thing that we can be empowered with, Dave. That's...

Dave:

Thank you for that. And I feel like I'm talking to a top functional genomics expert out there. You always teach me stuff when we talk. So I'm just going to tell you if you're listening to the show and it's within your means, The DNA Company, a cardiovascular risk profile analysis of your genetics. And I think you

guys will do it. If someone has an existing genetic data they can give you or you'll do a genetic test for them. So this could be just submit your data from... You can take 23andMe data?

Mansoor:

Yep. We can. The only thing that we don't get proper reads on are the C and V. So any of the genes in which the variation is a snip, most oftentimes 23andMe will have the data, but there are certain important genes in which the variation is not a snip. It's a copy number variation, or it's an indel, meaning it's not a single nucleotide change, but it's a deletion. Those genes and those important variations, which we of course do, you won't get those.

Dave:

I got a lot more from sending my spit to you than I did from any other spit sample. And yes, I filled lots of vials with spit because, hey, that's part of being a biohacker. So just thanks for your work and you're the brains behind the algorithms there. And I think anyone who's listened to this interview or some of the others, you know what you're talking about. So thank you, Mansoor, thank you for enlightening everyone who's listening about this cardiovascular risk, the interesting dynamics of sugar. And I have never heard a podcast where we just talk really openly about, okay, what does your race have to do with how well you're likely, but not guaranteed, to handle blood sugar. And knowing that can be really empowering. And it doesn't cost you anything to look at your heritage and say, "Based on that, my risk is probably this, and it's higher than that other person over there or lower than another person over there."

Dave:

And so this is free knowledge about the human condition that really isn't out there. And I think it needs to be out there. So thank you for sharing that as well.

Mansoor:

It's an honor. Thank you so much, Dave.

Dave:

Your website, thednacompany.com. Thanks again, Mansoor. See you on another show.

Mansoor:

Looking forward to it.

Dave:

If you've liked today's episode, you know what to do. You could leave a review that said, "Hey, this was worth my time." You can also, if it's within means for you, go out to thednacompany.com and actually get a test. Send in your spit and get the cardiovascular risk profile or the female hormone profile, or get all of it. That's what I did, except the female hormone profile. I didn't have to get that one. And what you're going to find is you have actionable information instead of just a bunch of genetics that doesn't really do anything for you. This is why I'm such a fan of functional genomics. And I think it's one of those world-changing things. Finally. Have an awesome day.