Introduction to Dr. Seyfried and His Research

All right. Welcome once again, everyone, to the Prostate Cancer Summit. Today I have the great pleasure of speaking with the one and only Dr. Thomas Seyfried who's a professor of biology at Boston College and received his PhD in genetics and biochemistry from the University of Illinois in 1976.

I was a long time Dr. Seyfried For a long time. Well, it doesn't seem that long ago, but but when you put it that way, maybe it is. Not to see. Friede has, done amazing work.

Much of which I have to assume some of you know, about to some degree, on the metabolism of cancer and the work of Otto Warburg, which we're going to talk a little bit about today.

Doctor Siegfried, I just want to thank you for the work you do. What we for? We get into metabolism of cancer. The research you've done. I have a simple question for you. Why?

Why this research? Why did you choose to do this over 30 years ago? And you took really the road less traveled. Doctor Roberts work was put away. It's, you know, it's nonsense is quackery or whatever it is put away.

You rose it from the dead, and you've devoted your life to this kind of work for a long time. Why? Well, let's let's go back a little bit here. I, I came into this, without any plan.

This was not my plan. My plan was to investigate the the structure and function of complex glucose, single lipids called ganglia sides in the brain. Looking at the origin and, how what they, what role they played in nervous, tissue in the brain basically I had no plan to do cancer research.

I was like everybody else. I thought it was a genetic disease. All the textbooks said it was okay, great. Who cares? It's a genetic disease. But when we when I was at Yale University for many years in my postdoc, we were doing a lot of work on epilepsy.

And, you know, we heard that the ketogenic diet might, might be effective in managing managing epilepsy. But when I wrote a grant at Illinois, oh, they said, no, no, wait, don't waste your time on these ketogenic diet stuff.

It's all it's all crap. It doesn't work. And all this, I said, okay, I continue to work on ganglia sites and map genes for epileptic seizures. And, so I wasn't doing much except we do it.

We were doing brain cancer, tissue to compare and contrast ganglia side composition in human, brain tissue with mouse brain tissue. To see if we could get a better understanding of of the of the structure and function of ganglia sites using using, nervous tissue, cancerous nervous tissue and normal tissue for comparative analysis.

And that was a real mess. Couldn't figure out anything. The patterns were all screwed up, and both the mouse and the human.

From Epilepsy and Ketogenic Diets to Brain Tumors

There didn't seem to be a rhyme or reason to anything, but. But the bottom line is that, when we started to do the ganglia site biochemistry, then when I came to Boston College, I continued the research on ganglia sites and epilepsy.

And then one of my students went out to the the the meeting in Seattle, Washington, with, Jim Abrams, the movie producer who produced the airplane movies.

His son had epilepsy and found out that the ketogenic diet, saved him from death, working with the group at Johns Hopkins University, late John Freeman.

And one of my students came back and told me, oh, the hottest thing now is ketogenic diet for epilepsy. And and having had the Yale experience with the leaders in the field, I said, I don't worry about it.

It's not that that's just nobody cares about that. So she actually convinced me. No, no, there's a got to get. I said, all right, all right. You know, I sent her out to the meeting.

She comes back with all this enthusiasm. They paid her way, and it wasn't going to even pay her way because I, I listened to what the the neurologist told me at Yale.

Right. These guys were the they told us that we got it, we got better drugs and all this kind of stuff. So then we started putting the the ketogenic diets on our on our models of epilepsy.

And it turned out that, wow, this stuff, really, this thing really, really did stop the epileptic seizures in the mice. So then we started to look at it and, it turned out that that you lower the blood sugar and elevate the ketones and you get into the special zone, and you were able to control the seizures, but you couldn't do that even with regular diet restricted to form a calorie restricted diet that would lower the sugar and elevate to keep tones to the same degree.

And then we started realizing that it was a combination of lower glucose and elevated ketones that would block electrical excitability in the brains of of of kids and animals with epilepsy.

The actual mechanisms of action are still under intense investigation. But at the same time, I had all these mice with brain tumors like we were looking at ganglia site biochemistry.

And, you know, one thing leads to another, all of a sudden we were putting, ketogenic, a calorie restriction on the mice with the brain tumors and, Whoa, what's going on with that?

So that was a reversal. Yeah. We said whoa, what's going on? Some and some group gave me a drug, to study ganglia side biochemistry and was to block ganglia side biochemistry.

We were going to look at it for a Tay-Sachs disease and this kind of thing. And then, it was in and b DNA, no drugs in neuro myosin, small molecule. And I said, why don't we throw this drug on the, on the, on the mice with the brain tumors and it shrunk the brain tumor.

Well, the drug company got so excited. So they started they gave me $200,000 in the year. And in in the year 2000, it was 1999, which was a lot of money.

Figure it out. What's going on with this drug? Because this was a new company and they just had this little molecule, very small molecule. So it was very interesting.

The executives came up to my lab, they wanted to talk to me, and I said, yeah, look, we gave the gave the mice with the brain tumors, this drug. And it shrunk. It shrunk the tumor.

And also reduced the synthesis of the ganglia side. So whoa, know this is like a beat us thing. So, we noticed that the body weights of these mice were were being reduced, from this drug, but they were still eating, which was the remarkable thing.

The mice were eating the food, but losing body weight. And, we decided at that time, do you think we should put a body weight control in there? Because we had the we had the mice with, with with no drug in the food, just eating the powder chow and the mice eating the powder chow.

The tumors shrunk, compared to the control, but so did the body weight. So then we put a body weight control and just regular mice eating the same food without the drug, just restricting the amount of food they ate to match the

body weight of the guys getting the drug.

It turned out that the tumor shrunk to the exact same design. The tumor shrunk with the calorie restriction just as much as it did with the drug. Yes.

So we figured out that the drug work by blocking suitcases in the gut. So the food they ate was not being metabolized, right, was kind of accumulating in their gut.

So they were getting an indirect calorie restriction from the effect of the drug on the ability to digest the food. So I had nothing to do. And but besides the the drug did inhibit ganglia site biosynthesis, which had nothing to do with why the tumor shrunk.

So of course, when I explained the whole thing to the company, they dropped me like a hot potato. They didn't want any part of this. Right. Exactly. Yeah.

All all of the thunder went away from their special drug. It was all due to indirect calorie restriction, right. Well, so there's this also seen with that enclosed drug Imclone drug with the Martha Stewart fiasco, several years ago where they were doing insider trading and all that, I went back and looked at that drug and it was working the same way.

It was working by calorie restriction. So so you got to be careful if you don't understand the biology and biochemistry of what you're doing and the hosts and all this other stuff, you can actually make big mistakes.

So, so then I said, what's going on? And we looked at the mice that were under calorie restriction and their blood glucose was down and their ketones were up just like the epilepsy.

So I said, what's going on with that? And then I said, I saw I heard this guy, oh, there was a group at Case Western Reserve. Linda Nibbling Group, and she had two little kids.

Hope what we call in the in the field hopeless cases, they both had, high grade, childhood gliomas. And they were both, considered terminal. They were.

We're so Linda, as a PhD nursing student, treated them with a ketogenic diet, according to the concepts of Otto Warburg. So I said, who the hell is Otto Warburg?

Okay, that's, Yeah. So Linda's paper, was published in a peer reviewed journal in 1995, and she rescued two of these kids. One kid, lived twice, three times longer than was supposed to, and the other kid was did so well, they were lost to follow up.

Warburg Metabolism and the Role of Glucose

And in both cases, there was a lowering of glucose and an elevating of ketones, just as we saw for the children with epilepsy. But we had no clue how this the mechanism for working, stopping seizures.

But Warburg had clearly said long ago, these tumors can't grow without sufficient amounts of glucose. And then I you know, I had done a lot of work on ketogenic diets with, lowing what ketones do.

And I work with the late, Bud Veach, who was Hans Krebs, his last PhD student, and learned a normal enormous amount from him. He was an MD, PhD, a, a guru in the field of energy metabolism, the late George Cahill, president and director of the Joslin Diabetes Center here in Boston.

So I was in those guys offices. They were in my office. I was I was a sponge for getting information from whom I considered the smartest and most knowledgeable people on the planet who understood ketone biochemistry, brain biochemistry and energy metabolism and bioenergetics.

And I was trying to figure out what was going on. With these tumors when we lowered the, the blood sugar and elevated the ketones. And it became clear that Warburg's argument that, fos ability to generate energy through oxidative phosphorylation in the mitochondria was compromised in some way, forcing the cell into a fermentation metabolism.

So we knew ketones and fatty acids could not be fermented. Therefore, I immediately knew they couldn't be used as fuel for the tumor cells. So they were dependent on on glucose.

So you lower the glucose and you elevate the ketones. The purpose of the ketones is to allow the body to get really, really low blood sugar, because that ketones can replace glucose, especially for the brain.

And then we also realized effectively, it can ketones can replace glucose in the brain effectively without compromising anything. Yes. And there was a magnificent study by drink, a shockingly clear study where he took morbidly obese men, and fasted them for three weeks, lowering their blood glucose and elevating their ketones.

Right. And then he shot them up with insulin. Can you believe this? And and it and it made the blood sugars go down to a 0.5 million molar or nine milligrams, but.

Well, any knowledgeable physician would know that should be that patient should be dead. There'd be almost no glucose in their body and wrote a beautiful article showing how all the patients were cognitively normal.

There was no cognitive decline. Supporting what Cahill said, that the brain can almost completely transition from glucose to ketone bodies without cognitive impairment.

This is an evolutionarily conserved adaptation. So I said, well, damn, if we do this to the tumors, these tumors are going to get hammered like there's no tomorrow.

And that's exactly what we saw. And then, more and more result. We published the first paper showing a direct relationship in preclinical mouse brain tumor models that the higher the sugar, the faster the tumor would grow.

The lower the sugar, the lower the tumor, the slower the tumor would grow. And then later, groups at Johns Hopkins and other other universities in humans with glioblastoma showed the same relationships.

And then it was shown for breast cancer, colon cancer, all the cancers, the lower the blood sugar, the slower the tumor grows. The higher the blood sugar, the faster the tumor grows.

So it became clear that, all of these cancers were behaving in a similar way when put under metabolic stress, according to the principles of Otto Warburg.

What Otto Warburg did not know is there was a second fermentable fuel which we have discovered, everybody knew glutamine could drive cancer, but they all thought it was respire, not fermented.

Our big thing with our work with Christos Gianopulos at Semmelweis University, the world leader on understanding bioenergetics of mitochondrial metabolism, knew that there was a second form of a fermentation that the cell could do, which was inside the mitochondria.

Everybody thinks the mitochondria gets energy through oxidative phosphorylation, which it does in normal cells, but in cancer cells that it's very minimal.

They're getting their energy from a fermentation of an amino acid, which is a totally different mechanism of getting energy. So we were realizing that the cancer field was mistaken, thinking that the oxygen consumption in cancer cells was used, or oxidative phosphorylation, which is the way most of our normal cells get energy because they're taking in oxygen and throwing out ATP must be ox US.

No, they're cancer cells are taking an oxygen producing reactive oxygen species. And the ATP coming out is from

the amino acid fermentation. And the fermentation is from glutamine.

And everybody no other amino acid involved in that process? No, not directly. And we and we looked into this because we interrogated all the tumor cells to ask if there was another amino acid, a little bit of glutamate could do it, but and a little bit of asparagine.

But they were very minimal. To do that mainly because you have to reconfigure the carbon skeleton to get to alpha key to glutamate. So it's an energy requiring process to make to make them be used for, for energy.

Glutamine is pure energetic gold. There's no that's that's necessary. There's no necessity of reconfiguring the carbon backbone. And that's why glutamine now becomes a real source of maintain.

Because they have the nitrogen for DNA and RNA synthesis. They have the nitrogen needed for amino acid synthesis. The glucose carbons are needed for a bio molecule synthesis as well as energy through the cytoplasm.

So those two fuels together are powerfully synergistic. On driving all major cancers. We have looked into all major cancers prostate cancer, colon cancer, breast bladder, brain, every kind of a cancer, every kind of a cancer cell, whether it's a stem cell, a mesenchymal cell, they're all locked into the same limited ability to get energy.

And that's a fermentation metabolism. Consequently, the solution to the cancer problem, all cancers, all major, all major cancers is the simultaneous restriction of the two fermentable fuels while transitioning the body over to fatty acids and ketone bodies, which cannot be metabolized effectively because the mitochondria of the tumor cells are inefficient and defective.

Exactly as Warburg said, and exactly as we have seen under electron microscopy that we have looked at, fatty acids, they say, oh, no, prostate cancer can use fatty acids.

No it can't. We have looked at that very clearly. Every major cancer that we have looked at has cytoplasmic lipid drops, lipid drop pools in the cytoplasm.

And that's due to ineffective oxidative phosphorylation. So lipids accumulate as lipid drops in the cytoplasm. Experiments were done. If you disturb oxidative phosphorylation there's a first thing that happens is lipids accumulate in the cytoplasm because the mitochondria are needed for lipid metabolism.

So they can't be used to they to protect the cell from death. They they store triglycerides in lipid drops. So we go through all the electron microscopy of all major human cancers.

And in every one of them they have lipid dropped storing in the in the cytoplasm clearly. And every one of them has abnormalities in number of structure and function in mitochondria.

Clear. Absolute clear. So that means they will be susceptible to simultaneous targeting targeting of fermentation fuels, glucose and glutamine while transitioning the body over to nutritional ketosis.

And what we do is we use drugs that push the glutamine a little bit more. And we've developed the pulse therapeutic strategy, which when adopted to adapt it to the clinic, will be the way most cancer patients will have their cancers managed.

And it's very effective without toxicity. And it works really well. Hold that thought, because now we have to go back and ask you some very basic questions before, because I definitely want you to talk more and so that we can all learn more about the press pulse.

Yeah. I just summarized 100 years of of experiments and biochemistry in that short blurb that I just gave you. Yeah, you sure did. And really got us to be right and got us to be, to learn more about how this all happened.

Glutamine, Mitochondria, and the Press-Pulse Strategy

And so thank you for that, for that information. It's I'm always curious as to why we do what we do. I'm asked all the time, why are you not sure why urology.

Why prostate cancer. So. You are essentially saying in simple words, hey man, you look at you guys are looking at it all wrong. You're looking at the genetic aspects of cancer and prostate cancer.

It has nothing to do with genetics. It has all to do with mitochondrial dysfunction. Is that what it is? Yeah, pretty much as an, It's terrible to say that, but it's really, it's really probably one of the greatest embarrassments of the I always say it's the greatest tragedy in the history of medicine, but it could also be one of the greatest embarrassments. Yes.

So somatic mutation. So the way we look at prostate cancer today, and there is I do look at because I'm not ready to give that up, I think that could be a combination and or at least a predisposition.

There is germline genetics that we look at. So if you have a mutation of BRCA two let's say that's a predisposition to not only prostate cancer but advanced prostate cancer.

These are papers that been written on major journals that this connection exists. There's other mutations, but certainly BRCA two okay. So it's one of them. And this is just for that.

Our audience can follow. Yeah. Germline mutations is, the mutations that are, that are transferred from parents to, to their offsprings. So go. Yeah. So Bob Kaplan and I, myself and Bob. Bob.

Bob is someone who works with us. We did a deep dive on all the germline mutations, that we know about. And you're right about the Brca1. The p53 Lee from many.

The retinoblastoma. Is that all these? Every one of them. Every one of those mutations just causes some level of dysfunction in mitochondrial, oxidative phosphorylation, leading to compensatory fermentation, which leads to dysregulated cell growth.

Not one single germline mutation is 100% penetrant. What that means it's a secondary risk factor. It's not a primary risk factor. It it causes cancer only if the product of the gene.

This causes disruption of oxidative phosphorylation, if that we have 5045 to 50% of women with the Brac of one mutation never develop breast cancer or any cancer for that matter.

How is that possible? Because it's not penetrant. So what makes some women have who have the mutation have cancer, and other women who have the mutation do not have cancer.

This is called the penetrance issue. And the answer to that is we don't we don't know, what epigenetics. Well, that's a term that people like to use, but it's a vast world of complexity.

When you when you say that, and the answer is, we're really not what we call tumor suppressor mutations where you have the mutation, but the effect of the mutation is suppressed by the action of another gene in the genome.

There's, this has been studied in the field of genetics for, for for decades, suppressor mutations in this gun. But when you have a primary cause, primary cause is always associated with the phenotype.

For example, Huntington's disease is always associated with brain degeneration, neurodegeneration. 100% of the people who have the Huntington mutation, the mutation in the gene huntingtin, leading to an abnormal expanding protein, will develop neurodegeneration at some point in their life, 100% related to that gene.

Tay-Sachs disease, various inborn errors of metabolism. 100% of the people that are homozygous for that mutation will develop the phenotype. Those are primary causes of the phenotype.

In no case is a germline mutation in any cancer gene 100% penetrant. The closest comes as the lead from any, which is about 80% penetrant. So that means 20% of the people that have that mutation, for whatever reason, don't develop cancer.

Therefore, it's not a prime cause. Everyone who has cancer from the leaf, from any mutation, what does that mutation do? It can code cytochrome c oxidase in the electron transport chain.

So essentially it's the disturbing electron transport chain mitochondrial oxidative phosphorylation function. And therefore that's the prime cause of all cancers.

We have never found a tumor that can survive without fermentation fuels and has and has a normal oxidative phosphorylation system. Everything comes back to the one organelle that is ultimately responsible for maintaining the quiescent, undifferentiated state of ourselves.

And when that organelle becomes chronically disrupted over time, can replace ox fos with fermentation. Whether it's from a germline mutation, whether it's from smoking, whether it's from intermittent hypoxia, whether it's from hepatitis virus, papilloma viruses, we went back out and checked the oncogenic parent.

We have not found any cancer that has a normal respiratory capacity, meaning that all cancers are similar because they all have an abnormality somewhere, somehow in the ability of that cell to generate energy through oxidative phosphorylation, requiring a compensatory fermentation, which then leads to dysregulated cell growth.

How is that possible? The mitochondria controls the cell cycle in the cell. So when those cells divide normally, like liver regeneration, gut regeneration, it's all controlled by the mitochondria.

The mitochondria energy metabolism allows our cells to be homeostatic and bioenergetics and grow when they're supposed to grow and stop growing when they're supposed to stop growing.

When that organelle becomes corrupted, the cell is fermenting, the default state is proliferation, and they fall back on the ancient pathways of fermentation where there's no regulation, everything is explainable.

When you understand evolutionary biology and the role of the mitochondria in controlling medicine, development. So the future of medicine relies on mitochondrial health and mitochondrial medicine.

We can say, if you want to stop chronic diseases for sure, it's not going to help you with understanding a hip replacement or a shoulder replacement, like for, but for chronic disease.

Absolutely. You because you can't it's hard to get cancer when your mitochondria remain healthy. It's hard to get diabetes, type two diabetes. It's hard to get any of these chronic diseases when your body is fit and healthy.

Are you suggesting that, a state of ketosis is. And let me let me step back here for a second, because I have to assume that the listener, many listeners are not they don't have a training in science or medicine.

The body uses different energy sources, and much of what the body uses for energy is glucose. What we're suggesting here is that glucose might be, or if too much glucose might be contributory to cancer development and progression, another way of getting energy into the body through the mitochondria, that's that is the energy source is ketosis.

Where the body takes fat in the liver, converts it, it converts it into a different chemical called ketones, and that those ketones are used for energy.

And so my question to you, Doctor Siegfried, is should we be in a state of ketosis for a couple of days a week, a couple of days a month, forever? What are you, zero sugar at all times? What?

What are you suggesting? Well, I don't want to suggest anything along those lines. Well, I think that comes to a

personal decision. I think if we want to beat cancer, if we have a cancer diet, prostate cancer, any cancer diagnosis, and we're saying and prevent it because maybe I'm in a higher predisposition.

Yeah. For some reason. Do we want is that the approach we want? Well, I think I think the knowledge of, of the situation allows people to make the decision as if they if, what would it.

Because we have two separate prevention from treatment. Yeah. How do we prevent cancer? Cancer is difficult. It's a difficult to elicit dysregulated cell growth in a population of cells in a given organ if mitochondria healthy.

So what? Whatever happened to, the the prostate, the bladder, the colon, the breast or brain? Something happened to the mitochondria in a particular population of cells, leading to a dysregulated cell growth.

How would we prevent that? Avoid the risk factors that would be provocative to damaging oxidative phosphorylation. Exercise, prevents, now, when you said, should we be in a state of ketosis?

You have to realize, we're a species. Oh, they say maybe 1.5 million years. 750,000 years of of being who we are. We were all we were always in some level of ketosis, mainly because there was no highly processed carbohydrates in sufficient amounts in our environment.

Our bodies, evolved to be in a carbohydrate poor environment. We ate and killed animals. We ate tubers. But none of these things, berries when they were ripe.

Any fruit? None of these things were there in abundance, at the fingertips. Most of them would be seasonal. So we would fluctuate in and out of ketosis as part of our natural, environment, as part of our evolutionary past.

So, in the in, in looking back, we can't say we know for sure that cancer would have been extremely rare, in the paleo in our Paleolithic ancestors, just as it is, is extremely rare in human populations today that live according to their traditional ways.

And this has been documented by physicians like Albert Schweitzer, the humanitarian physician who studied and looked for cancer in primitive populations that live according to their traditional ways and strikingly different from Western people living in Western societies where cancer was quite well known and seen, it was rare.

So why is that exercise the right natural foods? What was what was killing our Paleolithic lithic ancestors? Infections and injuries, not chronic diseases and weapons and then killing themselves? Yes.

I mean, of course, you know, you didn't have somebody that can do a hip replacement or a knee replacement or these the chronic pain and it from injury and those kinds of thing.

It wasn't it wasn't type two diabetes, obesity or any of the things that are killing us today. What we have done as a species, is that our technology of knowing how to produce the things that we like and convenient has created an environment that puts us now at risk for a whole slug of different chronic diseases, cancer being a major one.

Cancer Prevention, Ketosis, and Lifestyle

And that's because, as I said many times, our technology has evolved far faster than our biology. We are still Paleolithic in biochemistry and biology, living in the modern age of highly processed carbohydrate foods at the ready all the time.

Consequently, obesity, type two diabetes, chronic cardiovascular disease, dementia and cancer not complicated and sedentary lifestyle. Oh, absolutely.

We're not chasing down an elk to get our dinner for tonight. I mean, and, we open the refrigerator, we pull out and microwave something, right? That's right.

I mean, I mean, let's be honest. We have an obesity epidemic. Where did that come from? And obesity is replacing. Smoking is the number one risk factor for cancer.

And, and, and the younger people are getting are getting obese and cancer is coming earlier and earlier among people. I can't tell you how many people in the in their 20s and 30s are emailing me with all kinds of cancers and I'm saying, what the hell is going on?

It's diet and lifestyle. It's not genetics, the genetics that are, we evolved to starve and store energy. All of a sudden we're living in environment, massive amounts of of easily excess energy.

We get fat, we get sick. That's the that's the bottom line. What is the press pulse? Metabolic therapy for cancer. What is that about? And how who who is doing this kind of treatment.

And is it done after after a cancer treatment not for prevention. How does it work. Yeah, that's mostly for treatment. And that's the recognition that cancer cells cannot live without glucose and glutamine and cannot, transition their fuels from, from the, from these two fuels to fatty acids or ketone bodies.

But, we know we can press glucose. And this, this concept of press pulse came from the field of paleo biology. When they started to ask what was responsible in the past, a history of the Earth leading to mass extinctions of organisms.

We had some incredible mass extinctions of organisms over the history of the Earth, and they claim these paleo biologists said there was a chronic stress on populations in various continents, leading to the death of significant numbers of species, but not all species.

And then this was coupled to like a massive burst of volcano activity, a meteor strike or something, leading to mass extinctions of most of the organisms on the planet.

All I did was take that concept and say, we can press down glucose because it's a non-essential metabolite, but when we talk about glutamine, glutamine is essential for the function of the gut, for the function of our immune system, for the urea cycle, all of these different kinds of things, we cannot rest down glutamine without harming the body.

So we decided to use the concept of pulsing. So if we know how to periodically interrupt glutamine metabolism and then pull off the interruption, it's a pulse, pulse, pulse, not a press.

You can choke, hold the glucose and pulse the glutamine, degrading the tumor slowly without harming the immune system, the gut, or the urea cycle in doing so.

And that's what we're working on. The issue here is dosage, timing and scheduling. What is optimal doses, timing and scheduling for a person with a malignant metastatic cancer so that we can degrade that tumor, successfully over time without causing any, inadvertent toxicity to that individual.

And pulse therapeutic strategy can do that. Once we have perfected the dosage, timing and scheduling of the glutamine pulsing part, because we can we can clearly reduce glucose through diet.

And there's no question about that. As I've said, I've I said also, you'll notice in the press pulse, we also introduced stress management and exercise as absolutely essential components for the overall therapeutic package.

People get freaked out. Nothing freaks out people more most people than being diagnosed with a stage four cancer that you didn't know you had. All of a sudden, you have impending doom.

Your your stress hormones go up, your corticosteroids go up, leading to elevated glucose in the bloodstream. The thing you definitely don't want. So how do you manage stress in a patient diagnosed, with with a cancer?

And that has to be recognized to lower the blood sugar by stress management. You can do massage therapy. I don't know, okay, you know, exercise, all these different kinds of things.

And that's part of the of the price pulse. So we can press glucose, we can press, or at least manage, emotional stress. And then we go and we use a cocktail of various kinds of drugs that play a role in further reducing glucose and glutamine, like.

And then there's all the parasite medication. We and others have found that that targets the glycolysis and glutamine analysis pathways to subtly but enough to force the cell when it's glucose is not a stranglehold.

So so you have the knowledge now to know that there is so many drugs that are already out there available that could be part of the president's, strategy if people knew how to use them in the appropriate time and place.

Right now, we have all the tools. We just don't know how to use them all in the right way. And it's called the the oppressed pulse. So you pulse dosage, timing and scheduling degrading the tumor gradually, gradually, gradually.

And then people say, oh, you'll never get the glucose down as much as you need it. You don't have to eliminate all glucose, because what little glucose remains in your body through gluconeogenesis is sucked up by the brain.

And the muscle, muscles suck it up. Made glycogen. The brain will always take the most. The tumor absolutely needs the glucose. The other cells will be more apt to take it up.

So you have competition between the tumor cell and the normal cells in your body. For a fleeting a fuel that everyone, all the cells would like. But, but, but, but so only the tumor cell requires it.

So again you play the different populations of cells off in your body against the tumor. And you get auto lytic cannibalism, which I wrote about, where the body actually turns on the tumor and uses the damn tumor for fuel for the rest of the body.

But once you're in this level of ketone, it's unbelievable. Your body has so many ways to naturally heal itself, if given the opportunity, and poisoning and irradiating people is not doing the best way to help that person out.

So once you understand how to how to kill cancer cells using press pulsed metabolic therapy, it's going to be enjoyable not only to the physician but also to the cancer patient, because as you're degrading his tumor, he's starting to get healthier and healthier.

And that's just how do you how do you how do you pulse glutamine naturally would often vendors or any drug. And the reason I'm saying that is because part of the paleo right.

Yeah. Healthy. You know, and there's a lot of carnivores and paleolithic diet promoters and people who do such things. There's a lot of glutamine. All these things.

So they eat a healthy diet in air quotes because I'm sure many would disagree. But that's not the point of this story here. Right now they eat a paleo type of diet.

They want to keep the carbs down so there's no glucose scenario. But now there's a lot of glutamine in that diet. How do you limit the amount of glutamine that that feeds the cancer?

The glutamine is always the number one and highest, amino acid in our bloodstream. It dwarfs all the other amino acids naturally, because glutamine could be made from from from other, other sources.

That's why they call it a non-essential amino acid. Right. So it's always going to be there, but you always have an excess amount of glutamine, but that the cancer will be in the food.

What you eat is going to have very little impact, on that. So, well, one way to lower glutamine naturally is through

exercise. And Cahill's group, showed that.

So, a significant exercise, every day, either long walking or whatever, people do lower blood glutamine.

Clinical Use, Patient Involvement, and Research Support

But then with the drug you coming in, don't forget that becomes absolutely essential for this tumor cell to grow when you're under stress from glucose, and you and you're using the available glutamine, you just interrupt that just a little bit and boom, that guy collapses.

It's again a one man action effect. So I, I tell people, don't worry about the food, the glutamine. Your body already has more than enough glutamine in there circulating, but the tumor cell only needs a small amount of that, and we don't.

All we do is interrupt it in the tumor so we're not interrupting it long term. And any of the immune cells, we know that the tumor cell is dependent and the immune system gets healthier.

When when we pulse the glutamine, targeting, because the immune system needs glutamine itself to pick up the dead corpses of the tumor cells that you use.

So, consequently, when we throw in that pulse of glutamine, we're killing tumor cells. But we're just stunning or, interfering a little bit with the function of the immune system.

So, the immune system is not hurt. It's just stunned. You pull the damn glutamine targeting off the immune system gets active very quickly, starts gobbling up the dead, the dead tumor cells.

So that's why we cannot chronically target glutamine for the management of cancer. Because we need those cells to also facilitate the clearance of the cells that we kill, with the glutamine targeting.

So again, that's what I'm talking about. The, the, dosage timing and scheduling. How much glutamine targeting, how long and what are the schedules for optimizing killing tumors without, to, without, paralyzing our immune system for picking up the dead corpses again?

It's a knowledge of systems physiology. It's a knowledge of not only the biochemistry and bioenergetics of what the tumor cells need to survive, but your knowledge of how to play normal physiological systems together with the, the vulnerability of the cells employing the strength and power of the body, using it in the correct way. Now.

So again, every physician will come back and tell me I found all the I found that if we do it this long for that dosage, we're getting better effects.

In fact, we got a really good effect on this person, but we had to change it on another person. And every person is a different bio energetic of themselves.

So again, knowing the strategy, should allow the practicing physician to know this and adapt it for each person individually. And they'll be the ones and the patient and the physician together will be the, the ones to say what we need to do when and how, because that we can't right now we don't have A1A1 size fits all patients.

We have to look at sex, age, body weight, all these different kinds of things before we decide to come in with these targeting glutamine, targeting drugs.

So, I have the general concepts, the framework of what we need to do. We just have to flesh it out in the clinics, and they will come to tell us what the best strategy is for the particular patient they're dealing with.

Is there anybody, any clinics, any practitioner, utilizing this approach and figuring that out? Mostly it's going to be small clinics where we have some folks that understand what I'm talking about, and they start to adapt.

The patients, what we have found, the people who do the best using ketogenic metabolic therapy, are those that are invested heavily into their into their outcome.

Those are the ones who want to know as much about this as we do. They seem to do a deep dive on this. Those folks that say, I don't, I'm, Maybe this is too hard.

Forget it. They're finished. I'm sorry to say that, but we see that when the patient is involved and the family of the patient is involved, the outcomes are so much better than if they.

They're not the physician works as a team member with the family and with the patient. It's a it's a it's a it's a group effort. And they're all collecting data, recording, all of their blood glucose, their ketones of the glucose ketone index.

What foods, you know, how did this work out for you? We're going to give you a little bit of this drug at this time. How does it work? And we use noninvasive imaging to know whether or not this stuff is working.

You look under, MRI, Pet scan, Cat scan, whatever it is, whatever you're looking at. Oh, wow. That thing is not as big as it used to be. Or where did it go?

I don't see it there anymore. What happens? Yeah. You know, but but but that's, So it's a it's a work in progress. I'm not saying we have a cure for cancer.

All I'm saying is we have a strategy, a framework that we can allow people to live much, much longer with a higher quality of life and potentially an outcome that will be maximum compared to what we're doing today.

Doctor C3, thank you. So much for sharing so much knowledge on the metabolic approach to cancer. Your, your work in that regard is, is just, unbelievable.

And you're you're a thought leader in that area. And we thank you so much. How can people find you or be in touch with you or any organizations or any any nonprofits that can support your research?

Well, right now it comes through my university. People make donations to me through my biology website. There's a donation tag there. And the university then supports our cancer research through the university itself.

I don't I don't get any money personally from anyone. I can't accept it. But I do recommend that they give money to Travis Kristofferson's foundation for Metabolic Cancer Therapies.

That's a private foundation that supports our research. A503 foundation. Travis Kristofferson, he wrote the book. I have it here. What's the name? But tripping over the trip.

Tripping over the truth. Yeah, yeah, the the rise of the mind. The metabolic theory of cancer. So it's happening. It's just happening very slowly. What I said to you today is unfortunately not either known, or even heard by the majority of people in the majority of top medical schools.

They all think cancer is a genetic disease. And that's another thing that's extremely important. People need to realize there are two theories that define cancer.

What is the somatic mutation theory and the other is the mitochondrial metabolic theory. And when the and we have enough evidence to eviscerate the somatic mutation theory, it's not the correct theory.

And that what that does is it continues we have over 1700 people a day in this country dying from cancer. That's about 70 an hour. And that number will not go down any time until people realize that cancer is a mitochondrial metabolic disorder that can be managed, as long as they think they base it on the somatic mutation theory.

I see no major advances any time in the future. Professor C3, thank you so much. I appreciate you being on the summit. Oh, thank you much to you. It's very nice to be here.

Thank you.