P5P - Podcast - Travis Christofferson - 11.21.17.mp3

Introduction: Over the last four plus months, I have gotten to know **Travis Christofferson**, the author of Tripping Over The Truth. Over the last several years, he has become probably the leading documentarian of the evolution of cancer therapies. His book does not state but rather questions whether genetic anomalies are in fact the start of cancer, or are more downstream. Agree with him or not, if you look at the field of epigenetics, and the concept of nature vs. nurture across all aspects of our bodies, be it physiological or psychological, it is imperative that we, as a society, look at all theories and then test them. For those of you that follow me, you will know that my opinion, at least my current opinion, is that only using the genetic approach to treating cancer is at best inefficient. If it were me, I would want every edge I can get, and considering my long-dated gut issues, which put me in a higher cancer risk pool, I am focused on using preventive methods on myself; so even selfishly speaking, I want to know.

But what gets lost by some people who complain about the champions of metabolic theories and therapies is that the vast majority of its proponents believe in using these [metabolic] therapies as adjunctive therapies to standard of care, at least until there is sufficient evidence that genetics are irrelevant, which, by the way, none of them think.

Travis may not have his MD or PhD, but he has an encyclopedic mind, and a knack for synthesizing every bit of information on every drug, treatment, clinical trial, etc., so that he can raise the important questions. In my many years of researching all sorts of disease states and treatments, he may be the best value-adding documentarian I have come across. As important, doctors from around the world who are using metabolic therapies are having outstanding results and [almost] all of them are reporting back to Travis. Thus, for those patients or practitioners or loved ones out there who need to find additional help in their journey through the cancer world, Travis is the man to follow. His humility coupled with hard data and a desire for finding the truth should be applauded. I hope that over time the medical establishment in general will adopt some of that earnestness. When they lose their curiosity and lose sight of their limits, brilliant people cease to be brilliant. At least that is my humble opinion. And with that, here is Travis Christofferson.

[Note: I have again ***bold and italicized the background information*** and underlined treatment related information. I have **bold and underlined names and things that I think should stand out in your thinking**. Please do not hesitate to comment back to me at protocols@p5hv.com]

[00:02:49] [DE] I'm fortunate to be here today with **Travis Christofferson** who amongst other things is the author of a book called **Tripping over the Truth**, which has become famous or should I say infamous in the cancer treatment world as it focuses on the history and evolution of looking at cancer as a metabolic disease. He has his hands in a lot of things including sponsoring a recent conference on cancer as a metabolic disease and is involved in a new clinic that started in the U.K. and is the man bringing it into the U.S. that is in oncology care. And with that. Travis thank you for being here.

[00:03:35] [TC] Thanks for having me there.

[00:03:39] [DE] So what I'd love to do is start with you just giving your background and what we're going to do today that's a little different because you wrote the book and it's not you know and it is involved in but the book itself isn't specific to patient care. I do want to talk a little bit about your background and the book and then we'll get into some of the treatment things that you're doing and the way you see cancer being patients being treated now and in the future. So if you just start with your background would be great.

[00:04:05] [TC] My background may be woefully underwhelming but it's non-linear to say the least so I was on the med school track as an undergrad and I actually was accepted to the state school and dragged my feet for about two weeks before I was supposed to move there because I knew you

know I had eight more years of school and I wasn't sure if I wanted to be an M.D. or not. So I ended up jumping into a master's degree here in Rapid City at the school of mines in bioremediation. And so I… you know and then life sort of intervened. I met a girl from Texas in class and we got married and had two kids and I left grad school with three classes left and jumped into a business. And I remember telling my wife don't let me do this more than five years because I love science and I wanted that to be my you know my career. And so 17 years later I jumped back into to finish my master's degree and I only had three credits left so they kind of gave me some latitude and allow me to do this class on cancer theory and I'd stayed current you know with most of them with molecular biology so I knew you know was current with cancer research and I dove into this book called Cancer is a metabolic disease by this guy named Tom Seyfried. I just happened to click it on Kindle. It was more chance than anything. And it was just dumbstruck by it. Here's this guy out in Boston college that was claiming cancer was not a genetic disease. Right.

[00:05:50] [TC] That I had learned all throughout my undergrad. Everybody had learned there was through and through a genetic disease a somatic mutation theory answer contends that cancer is caused and driven by this sort of sequential series of key mutations to oncogenes or cancer causing genes and he had this beautiful textbook that had hundreds, a hundred years of evidence that a kind that you know was out there but never really put into this comprehensive over-arching look this comprehensive theory of cancer and so it was you know it's it was so amazing to me from a scientific perspective that I'd always dabbled in writing and he had written the textbook but within there was this beautiful story almost a novel like story because the main character his name was Otto Warburg who in 1924 claimed that cancer was a metabolic disease and he was a brilliant German scientist. He won a Nobel Prize winner, was nominated on multiple occasions you know highly regarded as probably the premier biochemist of the 20th century and he had claim cancer was caused by defective metabolism. But then in the 50s of course Watson and Crick discovered DNA it was known that there are mutations in cancer cells and all of a sudden the genetic, the genetic theory became entrenched and dogmatic and Otto Warburg, when he died in 1970, that was looked at like the scar in his career that he had this overly simplistic view of cancer and everyone kind of kind of laughed about it almost. So that was a story; it was sort of the scientific redemption story of this brilliant scientist that his theory got swept aside and then it made this incredible remarkable comeback.

[00:07:34] [TC] And so I kind of you know followed the history of all this research and summarize it in a book that was not by any means a textbook. It was more just a history and it over you know kind of a layperson written book of the theories. So that's you know it is nutshell that's so and talk.

[00:07:56] [DE] Talk a little bit more about the history in cancer and how it all came together. You know in terms of the you know what I would call the destruction of the of the metabolic theory.

[00:08:11] [TC] ***The history***: ***So what Otto Warburg noticed in 1910 in the early 20s what he looked at, he was a biochemist and so he looked at energy metabolism. He was looking actually at Sea Urchin eggs and he noticed that when Sea urchin eggs divide, they undergo this to the process embryogenesis there's this furious energy consumption and it fuels us through what's called oxidative metabolism which is the use of oxygen to create energy. So, he kind of likened this fearless growth to cancer and he thought that when he turned to cancer cells, he thought that's what he would see this like this burst of oxidative respiration that was fueling this uncontrolled growth. And that's all that was known at the time that cancer was pathological cell growth. So, when he looked at the cancer cell he didn't see that what he saw was this generation of lactic acid. Right. And so there's another energy producing pathway called glycolysis or fermentation that bypasses the oxidative respiration so it does not use oxygen and it creates this toxic product called lactic acid and cancer cells had switched their metabolism to this different form of energy creation and now the question was why and he didn't know why; he supposed later on in his career in the night in 1955, he did an experiment where he expose he took normal cells and just put him in a hypoxic environment, just took away oxygen.***

[00:09:43] [TC] ***And it was enough to cause what he called injury to the cells respiratory mechanism so it injured these organelles we call mitochondria. He called Gran at the time but this injury was sufficient to cause the cells to revert to this ancient form of energy creation and turn them into this cancerous phenotype. So, he contended that cancer was caused by injure to injury to the respirative apparatus. Right. And then as soon as we saw that that was one of the contending theories at the time there's really three theories: Otto Warburg theory, a guy named Peyton Rouse, an American scientist; he had a viral theory of cancer. We'd notice that there were viruses that cause cancer in chickens and other animals. And so that was kind of this pesky theory that would never go away but they never found a virus in man yet in humans. And then the other theory was a chromosomal theory; they'd noticed the chromosomes were broken defective duplicated in cancer cells and they instantly of course tied this to you know a cause. But nobody knew really what, those three theories kind of jousted back and forth until a famous series of experiments in 1976 where they discovered that the mutations in cancer cells were to key to key cell cycle genes. So in other words the genes that control cell division that's where we saw these mutations. And then this really snapped down what what they call the somatic mutation theory of cancer. So the race was off at this point this was 1976 and Varmus bishop where the scientists discovered this they won a Nobel Prize. Everybody thought we knew what cancer was at this point. And this ushered in this era of what we called targeted medicine, right.***

[00:11:21] [TC] ***We were going to develop these, these drugs that targeted these mutations and it was widely believed. I mean you can go through the history; you see the quotes. Nixon declared the war on cancer already and it was widely believed we'd have this thing figured out within five you know five to 10 years at the latest. And so those cures never came. We developed targeted drugs. They were very marginal efficacy and everybody became frustrated. And this led to what's called the Cancer Genome Atlas project, which is this massive governmental effort to sequence the genomes of cancer cells and if cancer is truly a genetic disease, this would be the right. The Manhattan Project cancer. This would be the end we would find all of the mutations that were causative for every type of cancer and then we could work off these sort of mutational fingerprints to come up with new cures, the exact cures. And so this began in 2006 and right away within the first couple of years the data was way different than most expected. What the data you know what we thought we would see the sort of mutational fingerprint that define each type of cancer and mechanistically to find it we'd say OK Gene A B C D is mutated. This causes this type of cancer. We didn't see that at all. We saw this what we call this huge degree of intertie moral heterogeneity So between diff from each patient's tumor there were wildly different mutations.***

[00:12:51] [TC] ***So it was very, very hard to draw a line between cause and effect and this held true for every type of cancer they sequenced; they went, you know they sequenced most types of cancer all the solid tumors breast lung brain prostate and so forth. And in some of these you know in some of these tumors, you'd find a single driving mutation. There was zero so based on this data it was impossible to reconcile a true a complete genetic cause to any type of cancer. And you can see this in the journal articles in top guys in the field like Bert Vogelstein at Johns Hopkins for example. He was he's you know regarded as one of the best cancer biologists in the world and he's wrote this paper that he says there's this dark matter in cancer. There's some other cause that we have yet to figure out. And this is we're sort of the guys like Tom Seyfried stepped into the fray and said: Well we've been missing this all along. There is these other causes and their metabolic. And so that's it. You know when I wrote my book it was kind of in the wake of this confusion that genetic theory was under attack. These scientists are coming up with these new theories to try to explain this confusing data. And so that's where we are now that's basically 100 years of cancer research and leaves us where we are now. And you know dogmatic theories like this don't topple overnight. You just look at the history of science in general and this is this is typical even though there's very strong evidence to show that it's not entirely genetic disease. Nobody's going to go out***

and say that the textbooks have been written. You know it's just this very slow kind of moving process

[00:14:29] [DE] So what in your research, because you know I know a lot of people liketo say you don't have a Ph.D. or you don't. And people have this or they don't know that and to me it's totally irrelevant. People either have a knack and impassioned or some things so you know I know at the beginning you kind of pooh-poohed not out you know, only having a master's and not being a doctor but to me it's you know you you, to me you wax poetic. So, in your opinion you know it's a metabolic disease or the roots of it are. But what, what do you think you know are the likely causes that you know viruses toxins in etcetera that that are that you know. And then we can get into a little bit about ways to either starve it to help the other cells to overpower etc. but **what do you think are the underlying causes** that use almost…?

[00:15:35] [TC] ***All of the above. You mentioned all those things that damage they damage DNA but they also damage mitochondria. So, it's very hard to parse out you can see these series overlap. It was very hard; you could see how one kind of cover the other up and it was easy to become misled. So, the question is which is damage first mitochondria or nuclear DNA. And of course Tom Seyfried, he contends that mitochondria are damaged first in this energy crisis leads to to down a downstream effect which are mutations in nuclear DNA. They have happened secondary to the true cause and then the genetic guys of course claim the other thing that nuclear DNA is damaged first by carcinogens and then you get the sort of shifting of metabolism from this damage. But but regardless the same things are causing cancer. We've always known that which are carcinogens list. Now I think as almost 300 on the NCI Web site we know them pretty well. They're everywhere and viruses and you know HPV is coming kind of. We're hearing more and more about, about that cause of cancer. Sunlight; anything that damages our body is a potential cause of cancer. And then the next thing…***

[[00:16:57] [DE] Do you think sunlight causes damage? Do you think sunblock, which is stopping respiration is…

[00:17:05] [TC] ***You know, I don't know honest. I haven't researched that enough. I just know that U.V. long-term, light by itself if it's intense long, long term will, will damage cellular structures. So but of course there's benefits to that. You know we need sunlight we need vitamin D. But irradiation in general let's say you know ionizing radiation is definitely a carcinogen. And then the question comes: Well you know we're designed to take damage and people we Evergrey everybody know somebody that's 95 and smoked their whole lives and never developed cancer. So what is it about our bodies that can prevent this. And we're now we're learning and this is kind of where they overlap. Learning how important maintaining low information, maintaining metabolic health is to preventing cancer. And there's kind of there's a I just there's a great video of this experiment that was done back in the 50s where they injected rats with tumor cells and then they injected the hepatic vein.***

[00:18:12] [TC] Right. So these tumor cells went into the liver and then they cut these open rats open five months later and their livers ~~were of course riddled with or~~ were clean. There was no cancer. There was no cancer in the liver at all. So, somehow they repress this cancer then they did the next experiment where they did the same thing they injected these rats with into the hepatic vein with cancer cells. But this time they did an incision in their stomach area so nowhere near the liver as it was separate but they did a few incisions they let they sutured it up let it heal did another incision let it heal and then five months later they again opened up the livers and looked at it. This time their livers were riddled with cancer. So, something about this wounding process you know even though it was unrelated. It was just it was in the body causes this cancer to take hold and grow. And so we now know you know that inflammation is intimately tied to and the wound healing process is intimately tied metastatic cancer growth. And so when you look at something like breast cancer, most, most patients will present with primary tumors. That's what clinicians see the majority of time.

And so, you'll do, you'll operate, you'll do a mastectomy or lumpectomy, and then you look at recurrences and when you look at the recurrence rate. It spikes at the one year point and then falls precipitously after that right. You'd expect it to kind of be smooth but it's not. You see this huge spike and then it goes down.

[00:19:38] And so the question is why does it spike like that. And the reason is when you do that you go in and do surgery. You're creating a wound. So, when you do that platelets come to heal the wound to stop the bleeding. They the granules. They release all the cytokines they release all these growth factors that promote inflammation immune cells come in and this fuels cancer. This process of inflammation fuels cancer. So this grad student was kind of looking at all this literature and he said well what if what if you did something as simple as you know what if the patients were injected with an anti-inflammatory just a non-steroidal anti-inflammatory drug. In the peri-operative phase, right before surgery to kind of quell this wound healing, an inflammation process. And a look back through the data and we found out when you did this because they did it for other reasons some of the patients just happened to be injected with a with an I.V. NSAID. Right. And it resulted in a 75 percent reduction in recurrence. So just a ten dollar ($10) shot. The simple intervention of suppressing the inflammatory response results in this huge reduction in recurrence. And so this this kind of experiments they show us that we could do so much better than we're doing. We don't even do things like that which are very, very low risk and have this potentially huge impact on recurrence.

[00:11:01] [TC] And these things that you know all these kind of interventions like that are sitting in front of us that that kind of focus on the whole body this kind of holistic view of metabolism and inflammation and so forth. That's kind of a long winded answer but I hope I hope that was good.

[00:21:21] [DE] Which speaking of less invasive and kinder procedures. Made me think of Kris Smith, who presented at that conference a few weeks ago. Can you actually maybe just take a few minutes before we dig into Care Oncology and I'd love to briefly discuss Dr. Slocum, and as I know you're close to him, and he's doing some amazing work over in Turkey. But just now, if you just give a brief summary of the key highlights from… it was a three day conference, which I am happy for you to give more details, but it was a three-day conference focused on cancers as a metabolic disease. There were some great speakers. So if you just give a little background…

[00:12:02] [TC] ***The two you mentioned Kris Smith, and you mentioned Slocum, Dr. [Abdul] Slocum and what strikes you about their, the sort of the example I gave you before. You know that's their approach is: what are these simple interventions that we could do that might have these dramatic impacts and outcomes!? And in general, where this leads you as to this this idea of combinations you know it's never going to be one thing. We've been stuck in this kind of ideology in pharmacy where we were always looking for this targeted silver bullet and we'll never get there. And the most profound effects we see in biology from a manipulation standpoint, a medicine standpoint is always in combinations and we've seen this over and over.*** **We learned this lesson with chemotherapy back in the 60s that combining agents is so much you know exponentially more efficacious than single agents.** ***And so you know it seems like we have to relearn this lesson over and over again. But what they're what they're doing is simply try to prepare the body in a way they use they use something called the ketogenic diet. And it's a very very simple intervention.***

[00:23:12] [TC] We don't we don't have large scale clinical trials on this but we theoretically if there's any predictive power you know this intervention is going to work and **there's lots of anecdotal evidence that it does work and what it does is you shift your internal metabolism from a carbohydrate burning metabolism the cancer cells love to a fat, ketone burning metabolism the cancer cells have trouble dealing with because they have less mitochondria, mitochondria damage, so they can't burn these [ketones]. These are the molecules that have to be burned through oxidative phosphorylation.** So that's kind of the starting point when you do that you prep the patient's body in the way the cancer cells are rendered much more vulnerable to traditional therapies. And ironically it's a kind of goal in therapy because then normal cells are made

more resistant to chemotherapy and more resistant to radiation and there's very good biochemical reasons for this. And this has **been shown in small clinical trials too when somebody is in ketosis and they go through chemotherapy they have way way fewer objective measurable side effects like the number of times they vomit and so forth**. So that's kind of the starting point of their therapies. And then they'll stack on additional therapies on top of that. And I think **Kris Smith was using something called Op tune which is a helmet that releases this electrical free frequency that disrupts mytotic cycles, so disrupt cell division;** and Slocum is using you know he really what they. Their clinic in Istanbul kind of like I did, they read Tom Seyfried's book. I think the head oncologist's that they're the name of their place is called chemotherapy. He's ***John Hopkins educated; then went back there and started this highly respected clinic in Turkey and then he found Seyfried's work and really kind of retooled his clinic based on the metabolic theory of cancer. So what they do is, is kind of* stacking protocol they do a ketogenic diet*. They'll have the patients fast before they're given chemotherapy. They have to give chemotherapy right.***

[00:25:16] [TC] And they're given a dose range and **they use a lowest dose that they're allowed to use. So they do low dose chemo. They do this thing called insulin potentiation which drops the patient's blood sugar you know into the 50s or even lower right before they get the chemotherapy. So you're trying to keep these you know these cancer cells starved at that point then they do hyperbaric oxygen which is another kind of gentler way to provide stress to cancer cells. They do glycolytic inhibitors, these molecules called two deoxy glucose that prevent sugar from entering the cancer cell. And so they stack all these therapies and they're one of the. They've measured results and they published they published on lung cancer and on pancreatic cancer and the results are all standing there. I think the lung cancer paper they're seeing 400 percent increase in median life expectancy and I think you saw the scans, David; they're just remarkable. They showed before and after one after the other scans; there were complete remissions from you know from massive tumor loads. So this is and this is what's interesting is this is really the first attempt at this. And this can only get better. I mean this is all going to be a matter of timing dosing scheduling and finding the right mix in combination where we can really have a massive impact. But that was a general theme expressly that I got out of the conference was this kind of this resurgence of people looking at combination therapies like that.**

[00:26:55] Yeah. So I was I also Kris Smith who I am going to have on this podcast. You know just less invasive. Using know lasers. You know his focus is on glioblastoma. But also you know again noninvasive, able to use directed lasers and not you know break open the entire skull. The results are then is his data. You know I and back to Dr. Slocum, and I believe it was Dr. Mercola that did the video? [Yes] And that's where I have that link and Tom Seyfried's interview and I will have it linked again at the end of our transcript. Everyone who goes there is stage 4…

[00:27:43] [TC] And if they're just staggering. Yeah there's study and I think everyone that goes there is always worse than the comparative ones they use. You know they compare to the standard of care which was prescribe with three and four level patients so yeah their, makes their data even more non-biased.

[00:27:59] [DE] So I'd love to talk about your current endeavor with bringing the clinic Care Oncology into the U.S.

[00:28:08] [TC] ***Care Oncology came about when I did a talk in London and that's where I met some of the doctors from care oncology and I'd heard about him before at a conference in Tampa and I knew what they were doing they again they also were from the starting point of therapeutic starting point they looked at metabolism and then they asked a simple question of what drugs are in front of us right now that can target this dysfunctional metabolism of a cancer cell.*** And it really is an underutilized research from their starting point when you look at what we have are a pharmacopeia we have about 2000 novel entities that are FDA approved drugs and

many of these are small molecules that effect they're called plyotropic. They affect more than one pathway. They're probably able to modulate more than one disease. But what happens is they get they'll go through they'll be sure through clinical trials which cost about a billion dollars for a single indication and then they're prescribed for that for years and then they go off patent and they become generics and then and then even if you just discover that they're good for a new indication, a different disease. There's absolutely nobody that will usher them through that those clinical trials are going to get approval for that new disease because they'll never win back their investment; they're a generic drug. So they're called financial orphans or just stranded. And everybody agrees that we need to utilize… these are just sitting there.

[00:29:49] [TC] **The Power of Combinations**: And the impetus behind Care Oncology was to take these drugs that we know have this robust data and you can look through the literature on the drugs and their protocol and there is so much data both at the population level and at the cellular level that shows how efficacious these are; and then they also knew about the power of combinations, so they looked at the synergy between certain medications and came up with a cocktail of four drugs that they thought would be the best to treat cancer and they began treating patients about four years ago. And at the same time they were approved for a clinical trial. So, they've been capturing data and looking at it over for four years. I think they've treated about 1,300 patients and they finally have gotten enough data for it to publish on glioblastoma, which is pending they should be publishing very soon. But the data looks outstanding right now. So, you know this this is another one of those things that that has been right in front of us for a long time and nobody has really used. And these drugs, a lot of these drugs like for example one of those metformin which is prescribed for type 2 diabetes. It's been around forever prescribed for decades and clinicians noticed in these retrospective studies because so I mean I think it's number one top prescribed drug in the world now or number two. So there's these massive blocks of data where type 2 diabetics are on these drugs for a long period of time. And clinicians begin noticing that they had far less cancer they had far less cardiovascular events they tended to live longer.

[00:30:30] [TC] **Metformin** So even though they had type 2 diabetes being on **metformin conferred all these you know all these preventative effects**. So then they sort of look back at the cellular level and try to answer the question why. Why is this doing this and metformin from the metabolic standpoint is pretty easy. Even though we still don't have a complete picture of all the ways metformin works. We kind of have a rudimentary understanding from the metabolic level when you take metformin it concentrates in the liver and that it inhibits what's called complex one of the electron transport. And so it's sort of just slows down metabolism. And when you do this you sort of trip all these caloric restriction mechanisms that promote longevity in the liver cells respond to this by producing less sugar blood sugar. So, your blood sugar drops and that's why it's an effective type 2 diabetic drug. But when you do that you also require less insulin so your insulin levels go down. Insulin is associated with aging of course and also with cancer. **Cancer cells have about 16 times the number of insulin receptors in normal cells so they are hyper responsive to insulin**. So now we're kind of getting the picture of how these you know these drugs work in a metabolic level. But yeah that was that was the impetus behind care was to kind of strand these these financial orphans and have the courage to start treating patients right away. One of the benefits of using these repurposed drugs is we have decades of safety data. We know the interactions. We know the side effects we know the dosage.

[00:33:03] [TC] The ***pharmacokinetics, all these things so there's a massive head start to using these***. And when you compare it to you know new drugs that come through the FDA pipeline for cancer; it's so expensive to do this it's you know north of a billion dollars that often times they won't even take these drugs out to measure overall survival. They use surrogate markers, use progression free survival. So, they'll get FDA approval and we won't even know if they're actually, you know to what degree they're affecting overall survival so some of these drugs have gotten approved. And then in the end once they've been on the market for a long period time, you realize they don't affect overall survival at all or if they do it's just you know marginally and they can cost $100,000, $120,000.

And also you don't have a long picture of side effects. For example, there's two I think the papers came out very recently on the new immunotherapies. I mean these are new procedures and we don't have a long tenure with them so we're going to uncover, do toxicology as we go on and they're starting to contend with patients that are becoming Type I diabetics from these new checkpoint inhibitor immuno therapies. So that's you know we contend with that with these new drugs we just don't have a long history. **We don't know what 10 years down the road, we're going to uncover and side effects and even how efficacious they are in most cases. With these repurposed drugs, we do know; we do know the safety profile and so there's no reason to not start treating patients off label with these and that. That's how Care [Oncology] got started**.

[00:34:37] [TC] And they're there and now they've we've expanded to the U.S. and we're starting to treat patients here too

[00:34:52] [DE] So, I know you know and as it stands you are one of the components I noticed in your article last week that there was a study on MRSA and other nasty infections that by adding a Statin was having a profound effect on the efficacy of the antibiotics, which is incremental to this conversation. But from the you know from whatever mechanism it's working on either…

[00:35:17] [TC] **The statins, most people don't realize how you know there really is a market that advances or so plyotropic, and you know we kind of get this kind of myopic vision. OK well there is hmg-CoA reductase inhibitors. They lower the bad form of cholesterol LDL, but they do so many more things and that when you really look at cellular processes, they're tripping multiple inflammation, epigenetic pathways, nuclear factor Kappa Beta, which is intimately connected to inflammation is down-regulated from statins. COX 2 is down-regulated; so all these things we know; clinicians notice that in addition all the effects you see from statins, the beneficial effects could not just be related to lower LDL. And so when you look closer you see all these anti-inflammatory properties and all these things so they're doing you know an incredible amount of things; we're just lucky that they are**. [Pause & technical problem] **Atorvastatin, which is a lipophilic statin**, which is important.

[00:36:27] [TC] **Mebendazol:** And then to the others: And then **Mebendazo**l ***which is actually a very old antifungal drug it was given to kids for pinworms and it's sold over-the-counter and most of the countries in Euro***pe. And **Gregory Riggins** he presented data on a phase 1 trial for Mebendazol alone at our conference for glioblastoma. And by itself they're using dosages that are you know far above that what we use. But they were able to get a double in the phase 1 trial they saw a doubling of overall of a median lifespan when it's added to standard of care. So, there's a very powerful drug it acts on the turbulent formations in it that's how it's an antifungal but it does the same thing; ***cancer cells have to create this sort of cytoskeleton to divide***. And that it inhibit's formation of that cytoskeleton.

[00:37:19] [TC] **Doxycycline:** ***And then the other one that's part of the cocktail is just a simple antibiotic, doxycycline. It's been around forever; usually gets prescribed to kids for acne for long periods of time very safe. And they noticed you know clinicians notice that sort of these long-term remissions in patients that had taken it. And then at the cellular level there's a beautiful paper in Nature that shows that it inhibits the formation of cancer stem cells. So, it just is a side effect, an off-target effect of the antibiotic and it sort of inhibits the mitochondrial formation of cancer stem cells, which are dependent on, more dependent on mitochondria biogenesis, and it inhibits that. And doxycycline is a little more interesting because you know people always get up in arms about the gut bio but it's absorbed higher up in the G.I. track so it disturbs the gut much less than traditional antibiotics***.

[00:38:33] [DE]I actually didn't know that part. So, I'm not going to speak out of turn on the data. I did see some of the data on using this [formula] for glioblastoma and I you know when it comes out. ***I will be sure to publish it on the Web site and tweet it out because*** **the data that I saw was**

nothing short of staggering. So soon enough. So you opened a clinic here in the U.S..

[00:39:06] [TC] Yeah yeah. So, yeah, I this new small town, Rapids City, South Dakota. The idea was just kind of get the logistics down here and then expand to bigger markets and bigger locations. And this is you know we want **the idea of this therapy is to never replace standard of care. It's purely an adjunctive; it goes alongside a standard of care. It's like the ketogenic diet in the sense that it it weakens cancer cells that are so dependent on these energy to grow**. ***And so that's why we're seeing you know that's obviously the largest effect is when it's used as an adjunct to therapy. But we wanted to bring it. We want this option for cancer patients you know everywhere and we're trying to make it as usually friendly as possible. And unfortunately, the way the laws are written that you can't prescribe it across state lines. So, they have to come see us here for us to prescribe, but the telemedicine laws are coming where we'll be able to you know do do when it's indicated we can do Skype we can do consults via telemedicine and then do prescribe across state lines will become much more user friendly once those laws get in effect***.

[00:40:26] [DE] Oh yeah. **You know to be it's you've opened a clinic with you with licensed doctors M.D. is doing a prescribing not you know**. Just being clear. So, what are you've been traveling a little, and what are you seeing in in receptivity in and in what you see in this country where the patterns you're seeing of where it's going and how this that hasn't been embraced in a lot of medical institutions. **But to my knowledge probably a leader of metabolic research has historically been at Hopkins if I'm correct and a few other schools, I know Duke** has done some research on and and…. [Technical difficulty] Actually they started with epilepsy with children.

[00:41:21] [TC] That is where **Pete Pederson…. He's a famous biochemist and really kind of took the baton from Warburg and mapped out a lot of the research that Tom Seyfried used to come up with his theory**. But there's a lot of this **repurposing research M.D. Anderson is doing a lot of it, especially with Metformin**. ***There's some huge nonprofits that are really pushing repurposing research but they're doing it a different way. They're trying to do the trials, the big trials too because they know these drugs will never be you know taken up by pharmaceutical companies so they try to do the big trials that give clinicians give doctors the comfort level to prescribe these things off label. Now whether that'll ever happen or not in a big way, I don't know. What my impression is that oncologists, there's so much information there, they have so many cancer types to deal with, right, that are just different anatomies, different standards of care. So it's an overwhelming amount of information they have to take and to get good to be good at their jobs. Then you put this next layer. OK. Can we do better. What else can we add onto this to do to do better than we're doing. And it just becomes overwhelming. So most doctors that I talk to you don't even know about metformin; they don't know the degree of research out there that shows that it could be this wonderful adjunctive therapy.***

[00:42:49] [TC] So that's what I encounter is it just there's almost too you can have two specialties; you could have an adjunctive specialty, and just a standard of care especially almost out there; and there are…. When you look at some of the more innovative universities like UCLA. If you go to their Web site on glioblastoma on brain cancers, they'll show you what they're doing in addition to standard of care off label, and they're one of the few places that actually show their overall survival statistics and for glioblastoma almost everywhere. The overall survival is the same; it's about fourteen point six months. And at **UCLA they're doing much better than that there I think**. So I want to say 600 some day 630 days. So; they're beating the national average and then you and then you scroll down and you'll see how, and they do tons of off label prescribing. And you know it's all evidence based but it's it's they're going pretty far out there off label in an attempt to try to you know help somebody with a terminal disease, and **they use metformin they use ketogenic diets, they use anything that they that they can**. ***And a lot of doctors in more conservative places won't do that. They won't stray from the standard of care. Now that doesn't help when some who's got a terminal***

illness right. It doesn't. You're locked in that mindset. It's not a good place to be when there's something that is safe that you could potentially change the outcome. Why wouldn't you do that. So there's kind of a disconnect I think between what you know what's a term illness and how far we should what we should do for these patients.

[00:44:24] [DE] We should be willing to take some chances and risk you know hey I had lunch with the woman who actually I'm going to be doing extra podcast with patients and this woman who's at. She was in one of the immunotherapy trials at Penn and she survived and was down to 80 pounds she was gone. And she made a very interesting comment. She said you know I had this mindset that I was supposed to be sick and die and I said I'm here and I'm in perfect health and I'm trying to figure out. And she wasn't talking about workwise and she has children… she's just "I'm trying to figure out what to do with the rest of my life because she didn't know it. And it was a mental energy thing and philosophical thing because she was supposed to be sick and die and she's not and then I said well and I so it's amazing, and how did the other people do in the trial. And you know and almost everyone else died. And it amazes me that you know when we know that the placebo effect alone is so powerful what the body is capable of if you unleash it to find its own state of health and that your approach with Care Oncology by opening up respiration and trying to kind of beat down and weaken the invaders while at the same time giving the host or the home team so to speak a chance to get stronger or at least stay constant long enough to overwhelm. It just amazes me because they'll put you through the nastiest drugs and special trials but they won't change your diet. And that's been a hard part for me having lost my dad to cancer and several relatives and other friends. So on that note I am appreciative of your taking the time. I'm probably going to ask you off line for a few more links to good interviews are there things that you think are great that will add to the show notes. Of all people I know in this area I don't think there's anyone better versed and better connected. And I think a lot of people find you to let to let you know what they're finding in their work. So to those listening I would I would say you need to follow Travis and I will go out. Sure.

[00:47:19] [TC] My pleasure. Thanks David.

[00:47:24] I will make sure Has that information at their fingertips so that they can do so. Thank you for coming today.

Show Notes

Travis' web site
http://www.foundationformetaboliccancertherapies.com/

Care Oncology Clinic - UK
http://careoncologyclinic.com/

Amazon link to Tripping Over The Truth
https://www.amazon.com/s/ref=nb_sb_ss_c_1_13?url=search-alias%3Daps&field-keywords=tripping+over+the+truth+by+travis+christofferson&sprefix=tripping+over%2Caps%2C125&crid=2LJTP7PA6LJA2

Barnes & Noble link to Tripping Over The Truth
https://www.barnesandnoble.com/w/tripping-over-the-truth-travis-christofferson/1125283646?ean=9781603587297#/

Simple name search for Travis Christofferson on YouTube. I would give you the individual

speeches and interviews, but they are endless. I do list a few of my favorites below though:
https://www.youtube.com/results?search_query=travis+christofferson
https://www.youtube.com/watch?v=8SeqDxuhFBU&t=6s - Slocum
https://www.youtube.com/watch?v=WvNaO0Poeqs - lecture

Johns Hopkins Medicine
https://www.hopkinsmedicine.org/index.html

Thomas Seyfried

- https://www.bc.edu/bc-web/schools/mcas/departments/biology/people/faculty-directory/thomas-seyfried.html
- https://www.youtube.com/watch?v=SEE-oU8_NSU

Hyperbaric oxygen
http://www.hbot.com/faq

Google Scholar Searches for the Four Drugs in Care Oncology's Formula

Metformin & Cancer
https://scholar.google.com/scholar?hl=en&as_sdt=0%2C7&q=metformin+cancer&btnG=

Atorvastatin & Cancer
https://scholar.google.com/scholar?hl=en&as_sdt=0,7&q=atorvastatin+cancer

Mebendazole & Cancer
https://scholar.google.com/scholar?hl=en&as_sdt=0%2C7&q=mebendazole+cancer&btnG=

Doxycycline & Cancer
https://scholar.google.com/scholar?hl=en&as_sdt=0%2C7&q=doxycycline+cancer&btnG=