P5P - Tom Seyfried #4 - 06.28.17.mp3

PLEASE NOTE THAT WITHIN THIS TRANSCRIPT, WE HIGHLIGHT CERTAIN THINGS IN DIFFERENT WAYS. ***THINGS THAT PROVIDE CONTEXT AND BACKGROUND GET BOLD AND ITALICS***. THINGS THAT ARE **TREATMENT RELATED GET BOLD AND UNDERLINE**. AND CERTAIN THINGS WE MAY BOLD, ITALICIZE AND UNDERLINE WHERE WE WANT TO BE SURE YOU DON'T MISS IT. WE ALSO PROVIDE HEADERS TO CERTAIN SECTIONS**.**

DAVID EIGEN INTRODUCTION TO MY INTERVIEW WITH TOM SEYFRIED

Welcome to the next episode of P5 Protocols with Tom Seyfried, a cancer researcher at Boston College, who is the world's leading researcher in, well let's use the title of his book: Cancer as a Metabolic Disease. When you meet Tom, who is 70 and widely experienced, you immediately sense that he is a force of nature!

Next week, I will be at a conference, aptly named Tripping over the Truth, which will have the world's leading experts in the research and development of metabolic treatment protocols for cancer as well as Alzheimer's. Tom is one of the organizers and featured speakers, deservedly so. No one with metabolic therapies for cancer does a thing without speaking to Tom!

I recorded this interview back on June 22 of this year, but held it back a bit to coincide with this upcoming conference. As well, since Tom is a researcher and not a practitioner, he does not prescribe treatment protocols, but he does the research needed to create them; and, with other practitioners around the world, he has done just that – and to great effect.

I love contrarian thinkers. As Mark Twain once said: "Whenever you find yourself on the side of the majority, it is time to pause and reflect." However, few do that – especially when their life's work is on the line. But Tom Seyfried did just that. For decades, Tom was a genetics researcher, who kept looking to find answers and found that virtually all of his past work did not make sense; AND he had the guts to turn his work in entirely different direction, risking the pitfalls of heresy. When I see people like that, I'm in!

I believe Tom is on to something. As some say, he thinks that going on a ketogenic diet will kill cancer. Though at times, when he talks, it sounds that way, if pressed, he emphatically states that is not the case. His entire research is based on putting together protocols that include many different tools and even some current tools that have their place and time of use.

As to the current genetics and immunotherapy approach, I hear of occasional miracles and in certain areas there have been and will be. But is it efficient? When will it work for most? Statistics alone tell me that many millions will die before that approach figures it out, if ever. Remember that the war on cancer started during the Nixon Administration. So, I wonder: If in the 1960's, when the world's computing power was equivalent to roughly one iPhone, we were able to put a man on the moon, AND get a lost space capsule back to earth, why, with hundreds of billions spent every year on genetic related approaches including immunotherapy, gene therapy, etc., are we not making any material progress? This should be done by now! Why are cures always around the corner? It makes no sense and for those in need, why not try things now??? Get any edge you can get?

If you are in cancer therapy – as a patient or provider, you have to at least listen to what Tom has to say. If you don't at least consider it, you are by definition not looking to get every edge you can get, and if your or a loved one's life is on the line, why not? And with that question, here is Tom Seyfried.

INTERVIEW WITH TOM SEYFRIED

[00:03:22] (DE): This is David Eigen again at P5 Health and I'm sitting here with Dr. Tom Seyfried who is a researcher in metabolic. He is the author of "Cancer as a Metabolic Disease", which I have my own copy here with me today and is the leader, or one of the leaders, in looking at metabolic therapies as both adjunctive as well as replacement therapies for various modern therapies. And Dr. Seyfried, thank you for being here with me today.

[00:04:01] (TS): And it's a pleasure. Thank you.

[00:04:03] (DE): We've spent quite a bit of time talking and I get to tour your lab and meet some of your cohorts and students and what I would love to do, as I've watched a lot of the lectures you've given online and some of the interviews and I'd love for you to talk briefly about what you're doing here. And then I'd love to go back into kind of your history in **your evolution and how you got here**.

[00:04:32] (TS): Yeah well thank you. We right now are, **we focus on cancer metabolism and we're also focusing on Lipid stores diseases and epilepsy;** we've done a lot of work in all these areas. But, of course, **the cancer problem is our primary, one of our primary areas of research and we've discovered over many years of research**, why or how we came to know that **this is primarily a mitochondrial metabolic disease**.

[00:05:21] (TS): **Tom's evolution in thinking**: Despite the fact that ***we, myself included, we were all convinced originally that this was probably a genetic disease mainly because you read so many papers about it and mainly because when you teach it in the classrooms all the textbooks focus on oncogenes and tumor suppressor genes and the signaling pathways and all this. And as instructors in biology, if you're not working directly in the field you pretty much use a textbook as a guide. So, but when you start working in the field you realize that, you know some of these things in the textbooks may not be correct, especially when your own research begins to challenge some of the fundamental issues that are in the textbooks because today many of the students in medical school or in graduate school, they go by textbooks. They go by what's written in the literature and they pretty much think well if it's in a textbook and it's got to be right that's got to be the correct information and then it started to become clear to us that there's a massive area of cancer metabolism that seriously questions the entire structure upon which the field is based. And that's not discussed it's not mentioned it's not even referred to in a general sense. So and we began to collect more and more data showing that these tumor cells are damaged energetically their mitochondria damaged and therefore the solutions to the problem become much more effective and reasonable and not so mysterious says as the current situation would have us believe***. And you know basically in response to your question you know how did I get to this place...

[00:07:01] (DE): Where did you start?

[00:07:03] (TS): ***So I started, well I started in graduate school and it was in genetics I did my basic research in neurosurgery genetics and at Illinois State University under Herman Brockman who is a classical geneticist in mutagenesis research and at that time we did mutagenesis research causing you know drugs that would make forward and reverse mutations in the bread mold in [indistinguishable]***. And we were using chemical carcinogens and certain drugs that are used to treat cancer but yet we were interested in how these cancer drugs caused mutations in a [neurospora] system. So our focus was more or less on the mutations, not cancer because we figured that if cancers; it was just you know it was a background for all cancers. You know it's got me cancer has a lot of mutations, so if you're in mutagenesis, it's linked to some way to cancer and all this stuff. But it wasn't like clinical issues or anything; it was purely basic research. So then I went to Illinois, University of Illinois to get my Ph.D. with the late Bill Daniel, and I was interested in human genetics and I was interested in lysosomal storage diseases which now represent Tay-Sachs disease and some of these Lipid stores diseases and I was interested in

studying the Lipid stores in the brains of mice. So we did a lot of work with gangliosides which are a class of complex Lipids that store in the brains of some children that have these Lipid store diseases. So we were doing heavy biochemistry into lipid metabolism and looking at certain mutations that affected the development of the brain.

[00:08:49] (TS): So this is very different than what I had done as a master student. However, it was in the field of human genetics. So while I was at Illinois, I took every kind of a genetics class that you could take. I took population genetics, I took human genetics, I took evolutionary genetics. On top of all of the genetics that I took at Illinois State University, at the same time I was studying Lipid biochemistry, so it was basically genetics and biochemistry that I did at the University of Illinois. And my dissertation was on the role of gangliosides and brain development. Then I left Illinois and did post-doctoral work at ***Yale University following up on gangliosides with with Bob Yu. Robert Yu was a professor in neurochemistry. So I got heavy into neurochemistry and because the department I was in at Yale was in the department of neurology and the main thrust was on epilepsy, they said if you want to stay around in the department you better modify your research to be involved with epilepsy***. Sure, gangliosides are great, but the focus of the department is epilepsy. So I started to look at the genes that controlled epileptic seizures in mice and dovetailing that with the biochemistry of gangliosides. And we put everything kind of together. But at the same time my mentor Bob had published an interesting paper on gangliosides and cancer cells and there were some interesting changes that were taking place in the gangliosides of tumor cells.

[00:10:33] (TS): So we said, well you know what kind of ganglioside changes are in brain cancer because that was in neurology and this kind of thing. And it was just a branch of extending you know one study to another, and it was also interesting at the same time with epilepsy I wrote an internal grant to Yale University to study ketogenic diets and epilepsy and they turned it down and they said no one's interested in ketogenic diets because the drugs are doing such a great job. So this was in the 1970s, ***OK probably '78, '79 somewhere in that area***. And I was doing ganglions - I came to Yale in 1976 and I was there for three years as a post-doc, and then I was appointed to the faculty as an assistant professor of neurology. So you know I was pretty ambitious in the projects that I was choosing epilepsy, gangliosides and then certainly some brain cancer, and then Dennis Spencer who was the Chief of Neurosurgery at the time, knew that we were working on some brain cancer. And he said once you come to the operating room with me I'll show you how it's really done. So he gave me this poor patient that he had with a glioblastoma I was able to get some brain tissue. You can't do that anymore today, too many rules and regulations. So we started isolating gangliosides from the tissue of a human and comparing it to what the mice cancer models. And it was interesting looking at the biochemistry of gangliosides in relationship to brain cancer.

[00:12:26] (TS): And then of course we were at the same time doing all this epilepsy research trying to map genes that controlled epilepsy, so I had I had several projects going simultaneously in the lab. But then I ***took the position at Boston College and extended all of these studies, and we continued evaluating lipid abnormalities in brain tumors as purely a basic research project***. Really there was really little clinical relevance to looking at gangliosides at least from the perspective, can we can we develop a therapy to treat cancer or brain tumors with our understanding of gangliosides. Of course, there were some loose connections but there was nothing really solid. But it turned out that at the same time we were developing the best animal models or natural models of epilepsy. ***And one of my students invited me to a meeting at the University of Washington Seattle about Ketogenic diet. And I said after my experience at Yale, I said one of my students came to me and said this ketogenic diet is really exciting. I had a disinterest from the Yale neurology department on Ketogenic diets. But my student was so excited. So Jim Abrahams, who started the Charlie foundation was there and he was giving an impassioned speech about Ketogenic diets. His son Charlie had experienced a horrific series of treatments drug toxic drugs, went to Johns Hopkins, and was able to get a Ketogenic diet.***

[00:14:16] ***(TS): Jim was just impassioned by the lack of information about this Ketogenic diet, and it was Jim's interest in this that kind of sprang the whole field forward. So my student, Mariana, came back and she said we're going to treat our EL mice with these Ketogenic diets and I said OK great.*** So she did all this stuff and I said OK let's see what happens. And it turned out that the diets were working really well in stopping this natural form of epilepsy in mice and I said this is really interesting. So when I was working with calorie restriction, Perna Mukerji joined my lab to look at anti-Angiogenic therapies for cancer because we found that a drug that we got to stop ganglioside biosynthesis was also effective in slowing the growth of these tumors.

[00:15:12] (TS): So you have to realize this is a hodgepodge of different areas kicking around with no real linkage between any of the areas: We had epilepsy, gangliosides, cancer and all this kind of stuff. But it became clear that the Ketogenic diet was working by calorie restriction. So that was very interesting. Calorie restriction shuts down angiogenesis. Calorie restriction was changing some of the profiles of the gangliosides. This is starting to become interesting and then we started treating the mice with brain tumors with Ketogenic diets. So it's like oh what's going on here?

[00:15:55] (DE): What year was that?

[00:15:57] (TS): Oh it was probably in the late 80s. It started when we started building natural brain tumor models in the early 90s. I felt that I had made a lot of brain cancer models by using a chemical carcinogen and unhip on pathology these guys. ***A great story you must read to get context in the field of diagnostics and specifically brain tumors***: ***They look very interesting, like human tumors. But then I became very very disillusioned with the neuro pathology field, the field of what we call neuro pathology, because I was making these tumors in the brains of mice using chemicals and we're getting beautiful slides. And I remember getting the first tumor's into my lab which started while I was at Yale and then transitioned over to BC from Harry Zimmerman and Harry Zimmerman was a physician at Montefiore Hospital in New York. But he started the first department of neuropathology in the United States at Yale University back in the 1930s. So Harry was old. Now Harry lived to be about 95, but he made all these mouse brain tumors with 20 [indistinguishable]. And he told me, well we have ependymoma, we have gliomas and we have these different names of these different tumors.***

[00:17:25] ***(TS): So I didn't think much about it since I knew nothing about neuropathology. These are all distinguished physicians and scientists telling me what they call these tumors. So I got the slides from Zimmerman and I was making my own tumors. I went down into the neuropathology department at Yale and Professor Kim who claimed to be the best neuropathologist in diagnosing tumors, looked at the slides that Harry Zimmerman made and said none of these are brain tumors, these are all muscle tumors. I said muscle tumors? I said this to Dr. Zimmerman who started the first school of neuropathology told me these were ependymomas, gliomas, brain tumors. So Kim said no. So I called up Zimmerman and I said to you know our neuropathology. "Oh well he doesn't know." I said I can send you back the slides and you can look at them. And he said yeah no no these are gliomas and he said Well Kim says they're sarcomas, muscle tumor. I don't know. They can't be a muscle tumor. So anyway I sent the slides to my good friend, the late Allan Yates from Ohio State. Alan was the chief pathologist there and he said no these are not either of those they're pure poorly differentiated neuroepidermal tumors. Completely different diagnosis than Kim or Zimmerman. And these are the same slides. I'm just passing the same slides around to different people.***

[00:17:55] ***And then I knew Albi Messing from University of Wisconsin who won all the contests for accurately diagnosing tumors. So I sent Albi the slides, and he called them something totally different. Another kind of a tumor that was different than what all the others had said and I said, Are you sure about this? And he says, I'm dead sure about this. And everybody was dead sure. And they all had completely different explanations for the***

same kind of tumor, and all from the same slides; it wasn't like they were. Chapter Three of my book discusses this in great detail, recorded exactly. So then, a kind of tumor that exists that is an oligodendroglioma, which is a tumor made from oligodendrocytes in the brain and a common kind of brain tumor oligodendroglioma, a lot of people who have brain tumors have oligodendrogliomas. And I'm in the neurochemistry and I go and I listen to some of these people talking about the origin of oligodendrocytes. These are small gliocells that make myelin for the brain and the controversy that exists on determining whether and where those oligodendroglioma cells, where those normal oligodendroglioma cells come from and I said man this is a real this is a real difficult field, yet, when you get a brain tumor, these neuro pathologist can look at the slide and in two seconds and say oh this is an oligodendroglioma. And I said how?

[00:20:24] ***(TS): So, Sandy Pelée, a member of the National Academy of Sciences who is considered the greatest neuro pathologist that lived, other than Ramon y Cajal who was the great one from Spain, wrote a beautiful book on an on an origin of cells in the nervous system. So I asked Sandy how these neuro pathologist made a split decision in a period of less than five minutes to call a tumor and be so certain that they were accurate. He says I have no idea. He says you can't make statements like that if you really know the problem. And I said oh my god. So this whole field of neuropathology is like based on a bedrock of sand and which is devastating to a field. If I said that to neuropathologist, they'd probably be quite upset.***

[00:21:10*]* ***(TS): But what my experience clearly indicates that you can get totally different explanations. So I brought that to my close and good friend Allan Yates, and he says, yeah well it's kind of a secret in the field we don't really like to talk about where you make decisions on how you're going to treat people based on the pathology, but that pathology could be most everything and anything.***

[00:21:31] (DE): So what year was that?

[00:21:33] (TS): ***Back in the 90s I guess when I was passing the slides around. It took over a couple of years. It didn't happen all at the same time. I was just passing these slides around, you know one thing leads to another and you start to question. You start to question the very foundations upon which a field is built, and you know I'm not a neuropathologist, but I had very close friends that were neuropathologists and they were all given me different explanations. And then I talked to the leader of neuroanatomy who knows the field, and he says I have no idea how these guys make these decisions because it's not based on the anatomy of the brain and the cellular anatomy of the brain. So I'm getting different explanations from different people around the same kind of subject.***

[00:22:29] (TS): ***So you know as we began to treat tumors with calorie restriction and Ketogenic diets, it became clear that the Ketogenic diet was having a major effect on the growth of the tumor as long as the diet was calorie restricted. So then we questioned what it was doing. So, Perna Mukerji and I showed that calorie restriction was powerfully anti angiogenic. It was such a large part of the pharmaceutical industry was building all these anti angiogenic drugs like Bevis's M-ID which is in in a variety of other drugs that were targeting blood vessels. And I said, wow you can target blood vessels by cutting calories, you don't need this drug. Why are you going to spend all this money on a drug when you can get the results from calories? Well nobody wants calorie restrictions, it's easier to take the drug.***

[00:23:21] (DE): But also I think once someone started down again, was Avastin that the only treatment usually used in a adjunctive therapy?

[00:23:28] (TS): **Yeah Avastin was used for brain cancer and other cancers. It was pulled off for breast cancer because it was causing more harm than good, but it still remains as a procedure for brain cancer**.

[00:23:43] (DE): **But I guess my point is when you start with certain drugs, then the fear is cachexia, the fear is that the patients get weeks to go. So if you don't start with so we're in the process calorie restriction really works at the beginning**.

[00:23:58] (TS): **That's another part of what we began to recognize. Weight loss from calorie restriction is a fear for cancer patients who are already losing weight. You can't treat a patient who's losing weight with a therapy that makes you lose weight. So this was the conundrum, and the problem of course is that calorie restriction is therapeutic weight loss. You get healthy when you do calorie restriction. If you have tumor [00:21:04] cachexia [0.6] or you're being treated with very toxic drugs you're getting poisoned and you're losing weight because you're sick as a dog. So you lose weight that's called pathological weight loss and weight loss in cachexia, insulin is elevated. Insulin and glucose become elevated during cachexia, whereas in calorie restriction insulin and glucose are very low. And that's putting the pressure killing the tumor cells, so this whole idea about weight loss: Where does weight loss come? From cachexia weight loss, the tumor cell is dissolving the muscles using the energy from the muscles to feed the tumor and you lose weight. And you also have weight loss from the sickness induced by radiation and toxic chemicals. So you put [00:21:47] cachexia, toxic chemicals, the patient's going to lose weight. All of these are pathological forms of weight-loss, very different than the healthy benefits of calorie restriction. But if you say I'm going to use standards of care that I get to make the patient very sick, and now you're going to put calorie restriction on top, that makes no sense. Although the work from Longos Group in California showed that if you do calorie restriction you can seem to tolerate chemo, toxic chemo better. So that's another kind of a branch from this whole thing.**

But getting back to the revelation of what we made in looking at anti-angiogenic therapies and seeing how many different targets calorie restriction has, Perna showed that calorie restriction kills tumor cells by a hepatotic mechanism, programmed cell death, and this kind of thing. And a lot of cancer drugs are wishing that they could kill tumor cells the same way, making anti-cancer drugs to stop blood vessels which calorie restriction will do more effectively than any than any anti-angiogenic therapy. And also inflammation is known to drive tumors and calorie restriction is a powerful anti-inflammatory therapy. So calorie restriction is actually doing what a whole different range of drugs would do. But the problem is calorie restriction is calorie restriction. People don't like to do that. So that's when we said when I said that hey let's try these Ketogenic diets maybe because the kids with epilepsy use Ketogenic diets and they seem to be able to tolerate this quite well as a powerful mediator of seizures.

At the same time, I started to run a small group of physicians at the American Epilepsy Society meetings who were interested in Ketogenic diets and calorie restriction for epilepsy management. So I started to lead the discussion groups at the American Epilepsy Society. ***It was a small group at first and then Jim Abrahams got involved and had Beth Zupec-Kania introduced her at the meeting. We used to do this Ketogenic diet in a small room, maybe 10 or 15 scientists and physicians. And then when I turned over the chore to Adam Hartman and Eric Kossoff at Johns Hopkins, I mean we had over 100 more than 100 people now interested in Ketogenic diets and epilepsy, and then it branched into cancer***. So the question is how is it working and what's the bottom line? So we lowered blood sugar and elevated ketones.

[00:27:51] (TS): ***Otto Warburg back in the 1920s and 30s, was showing that cancer cells are powerfully dependent on glucose and they don't have an effective respiration, and therefore they have to ferment glucose***. **And I said well if we lower glucose, we take away the**

fermentable fuels and then we regulate an alternative fuel called ketone bodies. And if the tumor cell doesn't have good respiration, mitochondria can't use the ketone bodies, so that then told us that the ketogenic diet was actually operating through Warburg central theory and it was Warburg's theory that brought us to the idea and ***then I said, how come I never heard of this? This guy Otto Warburg was of the 20th century's leading biochemist! And everybody talks about Warburg's findings related to biochemistry. But very little was talked about Warburg's cancer, it was like kind of "passé". It was a kind of an idea back when nobody's cancer was a genetic disease. And you know the metabolism was all like effects of genes and all this other stuff. And then I said, well geez, if the cancer cells can't use ketones, that's certainly supports Warburg's Central Theory that the reason they're using glucose in fermenting is because they can't respire. And then we and others started to test the cancer cells to see if they could burn ketones and they can't, which supports the idea there's a mitochondrial problem. And then it became more and more clear all these papers showing that the number structure and function of mitochondria are damaged in cancer cells. It locks in the idea that Warburg was right. Otto Warburg was absolutely correct in his understanding of that. And anyone who says that Warburg was wrong is either avoiding the subject or hasn't carefully read what Warburg has shown.*** I was very influenced by this and I had to go back and read all of Warburg papers and everything was ever written about Warburg, and I began to realize that he absolutely was right. But yet people didn't want to believe that it was. So I began to look at every paper saying mitochondria was normal and ***looked at the evidence to say that it was normal and it was mostly done in cell culture using oxygen consumption rather than looking at the structure and function of the mitochondria in many other ways. And when you do that you find mitochondria abnormal in cancer supporting Warburg's theory.***

Now the problem is if Warburg is right, then most of what is being done in the cancer field is probably not correct. And I said what's going and what are oncogenes and tumor suppressor genes? So they are the drivers of the disease. Well the oncogenes and the tumor suppressor genes according to the dogmatic view are causing the problems that Warburg saw. So what I did was bundled up all of the different observations using nuclear transfer experiments, and showed that, when scientists move the cancer nucleus into a normal cytoplasm, the cell recovered its normal growth ability and did not show the neoplastic dysregulated cell growth. So I started plucking these articles out of the literature one after another and put them all together in one big paper in Chapter 13 of my book and then later up another paper, showing the nuclear transfer experiments do not support the gene theory of cancer. They support Warburg's theory of cancer. So this was the first time we bundled up all these papers together and made a very strong statement that cancer cannot be a nuclear driven genetic disease. Period.

Now ***this went over like a lead balloon. You know the immediate response was not to attack me or attack this, but to ignore it. People would just simply not want to know about this. And if they if they knew about it they would not discuss it. So if you are wrong in the field, they're going to they're going to show you in no uncertain terms that you are wrong. The idea that there was complete silence over these nuclear transfer experiments which provided massive evidence to support Warburg's theory over the Gene Theory just was met with very little discussion. No major symposium on this, nothing! Yet, the field continues to pursue the idea that the cancer is a genetic disease despite having all of the evidence that's ignored. Wow, something has to be done. So I put all this together in my book because then I now I had the evidence knowing I'm a geneticist, knowing the biochemistry, knowing what Otto Warburg said, knowing that the mitochondria are in fact damaging cancer that a lot of people say doesn't happen. So I'm seeing a field ignoring all kinds of evidence not consistent with the dogmatic view that cancer is a genetic disease. So now you're touching upon dogma. You're now challenging a dogmatic view of an entire industry and field that's not correct. So how do you think that's going to go over. Not well but they don't they don't attack you, they just ignore you. And that's the way it always is, and that's the whole history of science. When***

something new comes out, most people ignore it because it's too disturbing to actually get into, because if you really get into it, then you have to be confronted with these facts and then it becomes very uncomfortable. Your world view begins to change and you now become uncomfortable with this new realization. So the bottom line is just to keep publishing papers saying Warburg was wrong, Warburg misled us with references that never really devoted.

And the majority of scientists don't read, and it's not that they read intensely in their own field. But if there's something that challenges that worldview, they wait to see what others say. This is what I always found remarkable. What do you think about the nuclear transfer experiments. Well I don't think they can be right. Well why not. Well I just don't think they can be right. Did you read all the- No I didn't but they just can't be right!

[00:34:17] (DE): But I think that in any research if you have grants and you have all your livelihood based on something, you're fully vested. But what about looking at it? So one of the questions I've had as I've looked at ketogenic diets and calorie restricted diets for probably five to seven years now, is if you get a PET scan, and my understanding, and I am being a little facetious, because they give you radioactive glucose, right? Because the cancer cells eat them up and it lights them up. Effectively. I mean in layman's terms.

[00:34:54] (TS): Yes. Yes.

[00:34:55] (DE): And so if that's what cancer cells feed on and they don't feed on fat or on ketones, then what I'm trying to understand is why don't doctors at least do that as an adjunctive therapy? You can still get calories from fat, certain amino acids, or proteins, but still heavily weighted to fat and associated calories.

[00:35:19] (TS): Yeah.

[00:35:21] (DE): But not feed sugar and glutamine, right?

[00:35:23] Right, right. Yes. And. Well you're absolutely right that PET scan is a way to detect cells that are taking in much higher levels of glucose because 2-Deoxy-D-glucose is metabolized to a non metabolizable fuel that gets accumulated and you can then you can see it. Now some organs like the brain also take a lot of glucose and it's harder to PET image brain tumors than it is to say tumors in the breast or some other organ. But you can use other forms of imaging. But yes, that that was one of the key things and we know that if you have damaged or insufficient respiration, in order to maintain the same level of energy coming out of the cell, or being produced within the cell, you have to use a more primitive, which is fermentation. Therefore, the amount of raw material you need has to go up exponentially to make up for the lost efficiency from the organelle that should be making the energy. So then you accumulate this product, then you can see these cancer cells or many of them. **So some people say well that's only because some cancer cells use glucose. There's some tumors that don't light up on PET. And that's true and those are the ones that are using the glutamine. So they're going to be more glutamine dependent and they don't show up on PETs so well. So they say we'll see Warburg is wrong because there's a cancer cell. But the issue is they're fermenting the glutamine, a lot of people don't know that. So, if your mitochondria is damaged you can still generate energy through mitochondrial fermentation. It's different from oxidative phosphorylation, it's mitochondrial fermentation you can actually ferment succinic acid in the mitochondria and you get tremendous energy from that they don't need so much glucose. We're seeing that with most of our metastatic cancer cells; many of the metastatic cancer cells use glutamine more than glucose, but many cancer cells use both. So the idea now becomes, Otto Warburg's theory of cancer is correct. In my mind there's no doubt about this.**

So if that's the case then cancer is a mitochondrial metabolic disease. So what is driving the tumor? And you say, well it's using fermentation. Well, there's only a certain number of fuels

that can be fermented and those fuels have to be abundant in the micro environment to drive the cell if you can't respire effectively. And the tumor cells are growing in a lot of hypoxic areas, so they're obviously using fermentation, because normal cells can only ferment for a very short period of time before they die. So tumor cells don't die in hypoxic environments; they do quite well; they're fermenting. So then you say, well cancer cells even ferment in oxygen because their mitochondria is defective when they switched over to an alternative primitive form of energy which is fermentation. So this is what all cells on the planet had before oxygen came in every living organism on the planet was a fermenter. So these cancer cells are simply falling back on an ancient pathway of fermentation. So if that's the case, they're living on the fuel of fermentation. They're living through fermentation metabolism, and there's a very limited number of fuels that can be fermented and the predominant ones are **glucose and glutamine. OK these are the fuels that are driving the tumor cell.** So, how do we kill cancer or take away their fermentable fuels? And then you have a very easy manageable disease and it's not 100 diseases.

Every cell that's a tumor cell is fermenting either glucose or glutamine or the two of them whether it's a brain cancer, or lung cancer, or bladder cancer, colon cancer, breast cancer, they're all doing the same kind of metabolism. **So the question is: doesn't it make more sense to target the common metabolic malady of all the tumor cells rather than focusing on a mutation that's causing a disruption in one pathway?** And then the other thing that became clear to us is that the mutations are not the cause of the disease. They're the effects of the damage to the respiration. So when you damage the respiration, you form reactive oxygen species which are carcinogens and mutagens. **So the mutations that exist in tumors, the many thousands of mutations, the millions of mutations that have been detected in cancer cells, are all a downstream epi-phenomena of the damage to the respiration. And we know that when we do the nuclear transfer experiments,, we can then eliminate all those. The mutations are not the drivers of the disease, they are an effect of the disease. So this is another challenge to the concept that cancer is a genetic disease.**

So most of the therapies that we use to treat patients in the clinic are all based on the view that cancer is a genetic disease. And this as I said accounts for ***why we have 6500 people a day dying from a disease which is treated as something other than what it actually is.*** So, you know, **immunotherapy and new drug therapies, most of these therapies are based on the idea that cancer is a genetic disease. Consequently, the tragedy of deaths by the millions throughout the world and the toxicity of trying to stop a disease that doesn't need to be, that where patients don't need to be treated**. [7.8] All you need to do is remove the two fermentable fuels and the tumor cells will die. No matter what kind of a tumor it is you remove glucose and glutamine and you transition over to ketones so the normal cells are protected. The ketone bodies protect normal cells. The removal of the fermentable fuels kills the tumor cells. It's a beautifully elegant, nontoxic system that can lead to the resolution of every kind of a cancer.

[00:41:05] (DE): But if you **break down things into a protocol**, which you're trying to develop here...

[00:41:08] (TS): Yes.

[00:41:09] What are the other... So there's the ketogenic diet.

[00:41:12] (TS): Yes.

[00:41:13] (DE): And what are the other components that you add in to help push the system?

[00:41:17] (TS): **Yes so we add we add small amounts of drugs that put additional pressure on glucose and glutamine while under Ketogenic Diet. So we can use drugs that will push glucose down very low. And you know people get all freaked out, oh my god you're going to**

have an epileptic seizure, going have all kinds of problems, you're going to die if you push your blood sugar. Yeah you could if you don't raise your ketones. If you elevate ketones, your brain transitions over to an alternative fuel, and you don't go unconscious when you push your blood sugars down to 30 milligrams per deciliter. And most physicians say, oh you could kill patients doing that. You could if you don't elevate the ketones, if you elevate the ketones you don't hurt the patient. The patient is no hypoglycemia associated with pushing glucose down as long as the ketones are elevated. So that eliminates that concern. So you know targeting glutamine is a little bit more difficult because glutamine is such an important molecule for the normal health of our immune system and our gut lining in all these other things. So you have to be able to carefully tweak the glutamine system so as not to harm the patient. So we put it all together, exercise stress management. We use hyperbaric oxygen, because hyperbaric oxygen in our view can replace radiation therapy. Radiation kills tumor cells by creating reactive oxygen species. Hyperbaric oxygen will kill the tumor cells using the same way, but without the collateral toxicity. But again you have to lower glucose and elevate ketones. And now that the cancer cells become extremely vulnerable to hyperbaric oxygen. So we can use alternative methods to every kind of a toxic drug therapy that's being used, and also radiation therapy by targeting the energy metabolism using drugs and procedures like hyperbaric oxygen and hypothermia. We use a number of glutamine targeting drugs, but all of these don't work individually. They need to work together synergistically with the entire cocktail approach. So the resolution of cancer will not come in my mind from any singular kind of drug used by itself, or even a cocktail of toxic drugs. You have to use drugs that are going to target glucose and glutamine, and protect the health and vitality of all the normal cells in your body which is what the ketones do. So Ketogenic diet is basically the platform by which all of these other drugs and procedures will interact synergistically to resolve the disease. So this is what I think will eventually replace it at some point in the future, once the field comes to understand what we're talking about, and to see the biochemical evidence for what we're talking about, will come to realize this is the strategy that will be most effective.

[00:44:11] (DE): What is the risk of trying hyperbaric oxygen which I've done, stress management, which I've done, although I think my wife would argue I haven't done it well enough, exercise and Ketogenic diet? These are all things which I've done and I've run multiple half marathons barely even needing to sip water and taking in a little bit of exogenous ketones right before and running half marathons with a few sips of water, versus previous years when I had to take tons of water. But I've tried all these and none of them seemed to harm me. So what I want understand from your perspective is what is the harm of any of these?

[00:45:05] (TS): Well I don't think I mean really harm relative to radiation and chemo. Very little. Right. I mean the ketogenic diet is not going to cause your hair to fall out or you know all these other kinds of things that you would experience if you had toxic radiation, or toxic drugs, or even the amino therapies have a whole list of adverse effects. None of the adverse effects on palatability, uh maybe a little digestive issues maybe a little bit. Very minor compared to...

[00:45:38] (DE): Foot cramps, foot cramps which I got.

[00:45:40] 9TS): Yeah. That's from too much exercise or something along these lines, all of these things will work really well. As I mentioned to you earlier, **it's all dosage, timing and scheduling, because none of these what I've spoken about will be effective in managing cancer alone. And in our new paper where we put all this together, the therapeutic strategy for the resolution of cancer was outlined in the Press Pulse paper** and ***the concept the Press Pulse is very interesting because it comes from paleo biology. And in the history of the Earth, we had these catastrophic events that led to the mass extermination of species at various ancient times. From volcanoes to droughts and to meteorite impacts, but massive extinction of species never occurred unless two very unlikely events happened simultaneously. There was a chronic stress on the entire population of organisms called the press, and that could***

have been from a climate issue or something along these lines. But it eliminated a large number of species that were weak. But some of the stronger and hardier species survived. It was only when the pulse, like a massive volcanic reactions, or meteoritic striking of the planet did we chronically press and acutely pulse that lead to the mass extinction of all the organisms. So what we did is we took that concept from paleo biology and applied it to Oncology, and ***what we do is the press is the Ketogenic Diet stress management and exercise and the pulses are hyperbaric oxygen, glutamine targeting, and glucose targeting. So we do that pulsing; we pulse those and we use the press. So it's a press pulse and the goal is for the mass extermination and elimination of cancer cells and has to be done nontoxic. And I believe that we will show that this strategy can be effective.***

In other words, is it possible to resolve cancer without toxicity? Because this is very important, because most people fear the treatment of cancer as much as they fear the disease itself. They know that they have to suffer from hair loss, vomiting, nausea, a litany of health issues. And I think we can achieve the same goal of eliminating cancer cells without any of this. But ***it has to be done strategically, and it has to be done in a very planned way that requires professional physicians that know when and how to apply this particular cocktail approach. And right now we have no training. None of this is discussed in medical schools. The physicians and the oncologists are not trained to know how to use food as medicine, to know that cancer is a metabolic disease, and not a genetic disease. So the very professionals that would be tasked with the new novel approach are not trained. So we have this gap of knowledge. So we know how to do this but we don't have the professionals to do it. And that's where we have to fill in these gaps.***

[00:49:01] (DE): These are these are protocols that I think sounds like you're most of the way there, and it's nuance here. Where are you seeing people, and what are you seeing with people that are being effective at these treatments?

[00:49:15] (TS): Well we're seeing quite as you saw some of the **data coming out of Istanbul, when they've applied a cocktail approach with diet's procedures and drugs, low dose drugs, we're getting tremendous therapeutic response** and this is just the beginning. I think we can streamline, refine, and improve these therapies. I think this is just the beginning. And I think cancer is going to be a very manageable disease. A lot of companies and think, well yeah we want to make cancer a chronic disease, you know chronic disease. **I'm not interested in chronic diseases, I'm interested in eliminating the disease completely**. So why would we make a disease chronic when we can resolve it? You know the bottom line is let's get rid of it. And people say, well you can't do that - but you haven't tried! If you do metabolic therapy you can. And the patient also has to be a participant in that healing process. It can't be, you know, you go to the physician: you have cancer. Ok, you turn your precious soul over to a profession with a less than stellar track record in keeping people alive. When you take charge of your own of your own destiny, you are now participating in your health. So this whole concept that he's battling is cancer. They always use this term. Oh they're battling cancer. How are they battling? They're sitting down there, they're being infused with a toxic drug, radiated a surgically mutilated, and they're battling? What are they doing now? They aren't battling. With metabolic therapy you are actually battling your disease because you are now an active participant in the management of your disease. With the assistance and guidance of a knowledgeable physician and nutritionist to do this, now you become part of battling your disease. When you sit down and have someone else treat you with these toxins, you're not battling the disease. There, by the grace of God, you survive what they're doing to you and I think this metabolic therapy is a completely different strategy and it will be shown to be the most effective way to manage cancer. It's just a matter of time.

[00:51: 16] (DE): I mean having had my own health issues and actually twenty six years ago seeing my mother go through - she was one of the first recipients of a [00:48:03] neupogen which, thank God, kept her out of the hospital, and she's totally fine and thriving and takes care of herself and

can sit on the floor with my kids and pop up without touching anything, but the havoc that it wreaked on her for the four months the chemo and the following several years of trying to rebalance her system. So, for me, what I'm looking at is an industry that will not just make a rapid change. There's just way too much invested from research dollars to big massive buildings and teams of people. And I think the hope in the near term would be to use these as adjunctive therapies and gather data. And in certain parts of the world where they're going to be more open minded, push those there and build the data.

[00:52:17] (TS): That makes sense. But of course it makes perfect sense. But on the other hand, you know people want to live. Do they want to wait another 10 years while they get there while we do this? Well, we're going to give you some chemo I think**. I think what we're seeing now is we can give people chemo, but at a very different dosage which would massively reduce the toxicity. So I think there's a place.**

[00:52:48] (TS): **Now, radiation will never be completely eliminated. There will always be a tumor that will be better serviced by a radiotherapy than any other kind of therapy. But I think the vast majority of metastatic cancers, which are the most deadly, and the ones that are most difficult to manage, will be the target of most of the of the metabolic therapy this is. You want to kill metastatic cancer cells non-toxically. I mean, if you have a benign tumor, a tumor that does not spread, any tumor that doesn't spread should be considered benign, and that can be cured by surgery or radiation. The only problem with radiation is you put yourself at risk for a future cancer. So why would you want to be treated by a treatment that itself causes cancer and could possibly come back at you in some way in the future? Now if you're 95 years old and you're going to irradiate some guy's tumor in a small area, a very benign tumor, fine do it! There's a place for everything, it just has to be logical. What's the line? You've got a guy that's 20 years old and you don't want to irradiate this person, because this therapy could come back at this person at some point the future.**

If you could eliminate that tumor with a nontoxic procedure, then you would want to do that. The problem is there's not enough evidence. People say, I have to have a clinical trial and all this stuff to prove this. We have a lot of it, but all of these are anecdotal. We do pile up a bunch of anecdotal reports. There's got to be something to it. The other thing about clinical trials and metabolic therapy is you've got to have a knowledgeable staff. You can't take people and run a clinical trial when they don't have any knowledge base on how to do this, right? So you're taking guys that do clinical trials and various drugs from the pharmaceutical industry, and now you're going to ask them through a clinical trial in a metabolic therapy about which they know nothing? You have to have the right kind of people running the clinical trials for the medical schools and everybody else has to stand back. We'll show you how to do it. And when you do it the right way, you get very good results. You saw some of the results from this. It's unbelievable.

[00:54:55] (DE): Now the pictures from this doctor in Turkey who I've seen in his podcast, both the video as well as read the transcript several times, that the results are staggering. It has nothing that I have seen here. I mean no one would ever get there. I've had gut issues and I've had that discussion when you start seeing any kind of dysplasia over someone long term with any form of inflammatory bowel disease, they automatically cut your colon out. That's just preventive. And so I asked, what percentage of people when you cut do you find even dysplasia by the time they do the procedure? Only half. Right? Half. They don't even know. And when a doctor who is extremely prominent said to me she just kind of dropped her head and she couldn't even look up when saying it. And I'm here because I'm fascinated by this work and the amount of people that are doing this work seem to be following a lot of your research. You've been the pioneer of this generation.

[00:56:03] (TS): Yeah well it's because we've developed in our own lab here at Boston College. We've developed the best preclinical models for the disease. So it's like a tool. It's a new tool. So if you have a good tool, you can understand and build things from that tool. Most of the animal models that are used in the big studies of the major hospitals are all genetically engineered mice.

All right. So again, if cancer is not a genetic disease, what are you studying these kinds of models for? I mean, every model is valuable in certain respect, but to put too much emphasis on an artificial model as you as I have a little discussion about that in the beginning of chapter 3, the concept of using artificial information to get real you get from artificial information you get...artificial models gives you artificial information. And then of course the hot thing now is patient derives anagrams. So you take a piece of tissue out of a human cancer, and you then put it into an immune compromised mouse. And the biology of the human and the mouse have been separated by 50 million years. The mouse is so much different than the human system. And you select only for those cells that grow in the body of a mouse, and then you treat those cells with a drug and you're expecting to go back to the patient. In some cases the patient does well, and in many cases the patient doesn't do well. So the best thing to do is use model cancers that develop naturally in the host. We develop those. So these models directly replicate human glioblastoma and exactly replicate what we see in the human disease. It's most systemic metabolic metastatic cancer and glioblastoma. And we try to manage those, and it's very difficult for us to resolve. And in fact, we've never been able to resolve a mouse glioblastoma but we're moving in that direction. So we make discoveries in the preclinical model, and then we tell our physician friends who apply it to their patients and the patients invariably do better than the mouse. Which is a mystery to us because where we thought we could be better in the mouse, but the patients do better. So and this is we're seeing it time and time again. So clearly the pre-clinical system having the right tools, knowing the right tumors, having the right strategy, allows us to explore all this: Treat it, test it, and then it goes right to the clinic and the patients do much better.

[00:58:27] (DE): Over the last bunch of years, what are the rough numbers of patients you've seen that have materially benefited? I know they've done their own thing, they're not following your protocols specifically, nothing is in peak performance so to speak. But what number of people have you seen over the years over studies?

[00:58:47] (DE): ***Dozens and dozens of people. They relate this to me and letters and they're still alive. The problem is we're having a tough time... we want to publish these patients, and a lot of them are not part of a study. So we have the data, we piece it together. The interesting thing about Ketogenic Diets if you're doing just Ketogenic diets, maybe a few extra add-ons, we're not saying we cure them... a lot of these people die. They're going to die, all right? Not all of them, but many of them die anyway. But you know it's the process. Quality of life. I've seen GBM patients who reject standard of care of chemo and radiation and steroids and all this stuff. They have a very high quality of life, and they're very good two or three days before they pass. And it's very interesting. A lot of my physician friends have been telling me the same thing. The patient is like, wow this guy, all of a sudden, he didn't have this long lingering horrible existence of suffering from toxic therapies. They live a very good quality of life and then expire in a couple of days, which is actually surprising to the family. Because I've gotten letters back from family members and saying, my loved ones so and so was great up until the past two or three days before he just started going to real quick downhill. Now, the problem.***

[01:00:15] (DE): Is that the tumor starts pressing on...?

[01:00:17] (TS): I don't know what is going on. It's a very surprising thing because a couple of my physician friends have told me the same thing. These people on these metabolic therapies or at least Ketogenic Diets, they're very healthy and they look really good. And then they go downhill very quickly at the end, rather than the slow lingering degrading of the body.

[01:00:37] (DE): But do they live longer?

[01:00:38] (TS): A lot, yes. Because they... there was a French person who was 71 years old, and he had a glioblastoma, and he rejected all chemo radiation. They said, you're going to be dead in three to four months. He lived 17 months with a very high quality of life, and then passed very

shortly very quickly and I've had Dr. LaValley telling me the same thing about several of his GBM patients. Now the question of course is that we have not yet applied the full battery and cocktail of approaches that we could we have, and we are still vetting them in the lab right now, because we're not targeting the glutamine in these tumors yet. We're just using the glucose targeting. And we haven't pulled the whole full cocktail on these hyperbaric oxygen, hyperthermia and all these kinds of things that we can do. So I think that it's just a matter of time. Now when we're doing this in some of our GBM patients, treating them before we do standard of care they do much, much better. So I think it's possible. It depends on your age.

But you know people say, well a year or 14 months might be the average death from GBM, but I think we can triple this at least. 36 to 48 months out which is far, far greater in longevity than we currently have. So you know I'm looking at strategies that are going to enhance the health and vitality of the normal cells while specifically targeting and knocking out the tumor cells without any toxicity and this alone will allow patients to live so much longer. **We're on the path to resolution. I'm not saying we have a cure for cancer. We have a path to resolution and long term management. And I think that's what we have. I'm not going to say anything like that. People sometimes say, well Seyfried says you can cure cancer with a Ketogenic Diet. I never said that. I said a Ketogenic Diet is going to be the important platform by which we're going to move toward resolution. But it's not the resolution itself.**

[01:02:48] (DE): But I think maybe another way of saying that, my own interpretation is, if you can go one path or you keep feeding the cancer. And that's a much tougher battle. I just think of World War II, because I was watching another movie recently, and if the guys up in battle that are fighting the war aren't being refueled, rearmed, and re-fed they're going to break down. And the way I see this is that you're trying to starve the cancer of their fuel, you're trying to support the healthy cells. And over time, first of all you're giving the body a fighting chance.

[01:03:29] (TS): Yes. You battle your disease!

[01:03:31] (DE): You're battling your disease as you said before, but you're giving the body a fighting chance.

[01:03:35] (TS): You're using a fuel that the healthy cells will immediately use that becomes non metabolizable to the tumor cell because they have the defective respiration. So it's a marginalization of the tumor cells to the advantage of the normal cells. This is a totally different strategy than what we're using today. We're using toxic drugs that go in and kill tumor cells but also put the normal cells at risk. So even the immunotherapies can cause massive problems; they can attack your immune system and they can kill you. A Ketogenic diet is not going to kill you. Whereas some of these immunotherapies will actually kill you. So you have to be very careful. And it's the drug working with the diet in a synergistic way. Now will it immunotherapy work together with a Ketogenic diet? As I said, I think the immunotherapies could be very valuable as the last stage in the management of the disease, not the first stage. They could clean up any surviving tumor cells because they were all going to have the same epitope that will be targeted by the immunotherapy.

[01:04:44] (DE): I don't want to go too far off into that, but it's very interesting. The one thing I have seen as I've looked a lot into the microbiome, is diversity. The microbiome tied to immunotherapy. The higher the diversity, the higher the survival rate. But if you go through chemo or you go through antibiotics, the other thing you are killing is diversity.

[01:05:04] (TS): So you don't want to do that. Right. These are things you don't want to do. Right. And you don't want to give patients high dose steroids. This is another thing that's really remarkable. **When you have patients that are getting sick from chemo, you give them high dose steroids to make them make them hungry which makes them burn glucose.**

[01:05:22] (DE): Which makes them burn glucose.

[01:05:23] (TS): **Right. Which is fueling the tough cells...**

[01:05:27] (DE): Seems like you become hyper vigilant, and you can't calm down, you breathe less, you wind up in fight or flight mode...

[01:05:33] (TS): **Oh it's terrible and it drives the tumor. So you would never- When you understand the biology of cancer, really understand the biology of cancer, the treatments that we're giving to this cancer patient...you would never want to do that.** Those are things that you would never want to do, but they do it because they don't understand the biology of the disease. If you understand the biology then you move into a totally different realm, a totally different approach; a strategy that's very, very different. **Do we use drugs? Yes! Do we use procedures? Yes! Do we do diets? Yes we do them all, but it's a very different context. And I think when you put them into the right context the results are spectacular. It's still an emerging field- we're just beginning, but we can see the end, we can see a light. It's a very bright light at the end of the tunnel.**

[01:06:25] (DE): I appreciate your time. I would love to do a follow up interview soon. And my focus is on cancer and several other diseases that are highly prevalent, to find the protocols that will work and that will create the environment for each individual to be healthy going forward because, really, I've seen too many people and too much cancer affecting too many people, I know and it's highly destructive and they become shells of who they were. And I'm fascinated and I'm a believer. I practice a lot of these things preventively. And I want to thank you for all of your contributions. I know it's your life work and your passion. I hear what drives you, which is to save people, to right a wrong so to speak. Even whether it's right or wrong or not, I personally am very much in your camp, but I know I have to say I remain open. It's the old quote that is attributed to Mark Twain which is, '***it ain't what you don't know that gets in trouble it's what you know for sure that just ain't so". And I've had this view that whether you can have a left wing or right wing view or anything, it's this fundamental unwillingness to listen to the other side.*** And I think in this case that with the amount of work that not just you're doing, but others are doing, and with the with the Internet and social media, the word is starting to get out. You've said you get over a thousand people requesting help, you pass them on as you're not a doctor, and you give them information. But this stuff will come. This stuff will happen, it has to happen if not just because it's supportive and you're starving the cancer and you are giving the body nontoxic methods. It should at the very least be an adjunctive therapy and hopefully adjudicative.

[01:08:38] (TS): Oh yeah I hear you and I'm open to this. Let's see the results. Like we said, we're getting very remarkable and astonishing results on some people and I think that could be the trend. This could be not the exception but the norm.

[01:09:01] (DE): I'm thrilled to launch this podcast talking to you. And I want to thank you for your time.

[01:09:09] (TS): Remember to donate money to this Single Cause Single Cure Foundation.

[01:09:13] (DE): Which I will be doing.

[01:09:14] (TS): Because Travis is a good person. He's focused on metabolic therapy for cancer. He knows the cancer is a mitochondrial metabolic disease, and I think his foundation will eventually be one of the leading foundations in the quest to manage this disease.

[01:09:30] (DE): Well I will write down the URL and all the relevant information in the show notes so that people can easily access that.

[01:09:40] (TS): Good. Thank you.

[01:06:19] (DE): And thank you.

Resources & People Mentioned

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

New York Times – May 12, 2016 Sunday Magazine – MUST READ!
https://www.nytimes.com/2016/05/15/magazine/warburg-effect-an-old-idea-revived-starve-cancer-to-death.html

Single Cause, single cure foundation
http://www.foundationformetaboliccancertherapies.com/

The Charlie Foundation
https://www.charliefoundation.org/

Ketogenic Diet

- https://www.healthline.com/nutrition/ketogenic-diet-101
- https://ketonutrition.org

Hyperbaric oxygen

- http://www.hbot.com/faq

Metabolically Supported Therapies for the Improvement of Cancer Treatment

- https://articles.mercola.com/sites/articles/archive/2017/03/19/metabolically-supported-therapies-cancer-treatment.aspx
- https://www.youtube.com/watch?v=yGnJQ2kGB-g#action=share (Tom Seyfried on Dr. Mercola)
- https://www.youtube.com/watch?v=tL8rQ3aNvhs&t=1540s (Dr. Slocum on Dr. Mercola with pictures of recovery!)

Dominic D'Agostino

- https://dominicdagostino.wordpress.com
- https://twitter.com/dominicdagosti2
- https://tim.blog/2015/11/03/dominic-dagostino/
- https://tim.blog/2016/07/06/dom-dagostino-part-2/
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Otto Warburg
https://www.nobelprize.org/nobel_prizes/medicine/laureates/1931/warburg-bio.html

Ramon y Cajal
https://www.nobelprize.org/nobel_prizes/medicine/laureates/1906/cajal-bio.html