Tripping Over the Truth: The Metabolic Theory of Cancer:
A Special Interview with Travis Christofferson

By Dr. Joseph Mercola

**JM:** Dr. Joseph Mercola

**TC:** Travis Christofferson

**JM:** As you’re watching this interview today, 1,600 people will die, in the United States alone, prematurely from cancer. That number goes up ten-fold, up to 21,000 if you include the entire world population. Wouldn’t it be interesting if there was a simple dietary tweak that could not only prevent but treat the vast majority of these cancers?

Hi, this is Dr. Mercola, helping you take care of your health. Today I’m joined by Travis Christofferson who wrote a phenomenal book. I read 150 books in 2015, and this was clearly one of the best health books I’ve read. It was fascinating and absolutely, in my view, a required read for anyone who has cancer or knows someone who has cancer. I can’t say enough good things about this book, and I am just absolutely delighted for Travis to join us today to delve into more details. Welcome, and thank you for joining us today, Travis.

**TC:** Thank you for having me.

**JM:** Alright. As I said, it’s just a magnificent book. It really highlights the work of Dr. Otto Warburg, and his descendants or generations of additional researchers. But I never had any idea. I mean, anyone who’s in natural medicine has heard of Otto Warburg. He got the Nobel Prize in 1931. But you go into… It’s such a fascinating read what you described, and we’ll talk about that in a bit. So why don’t you explain to us what motivated you to write this masterpiece?

**TC:** You know, it was really a convergence of several factors. I was in graduate school, and I was doing a class on cancer theory. I was studying what the textbooks put in front of me, which is the somatic mutation theory of cancer. I was just leafing through my Kindle, looking at the books that were published on cancer theory, and I stumbled on this book...

**JM:** Hang on for a second, what were you a graduate student of?

**TC:** My undergrad was Pre-Medical. My Master’s degree was in Materials Engineering and Science.

**JM:** Okay.

**TC:** I was doing Bioremediation, so I was still stated at kind of at the [inaudible 2:27] of biology. I was doing a class on cancer theory and I stumbled on this book by Thomas N.
Seyfried, called *Cancer as a Metabolic Disease*. It was just an incredibly well-written book. It was incredibly well-backed up by research. It laid out this elegant argument for this non-genetic origin of cancer, the metabolic origin of cancer.

I was just so stunned and taken aback by this, and that more people didn’t know about it, that I compiled a huge list of questions and actually flew out to see Tom in Boston, and sat for like six hours in a conference room with him and a couple of Grad students. He answered all my questions, which really served to kind of pique my interest more.

When I came home, I dove into this data from the Cancer Genome Atlas project, which started in 2006. It was the largest government project ever conceived to sequence the genomes of cancer cells. If you’ve heard of it, the Human Genome project, this was 10,000 times the amount of sequencing as the Human Genome project. The goal was to ferret out all these mutations found within cancer cells.

When I dove into this data, what I found was this incredible amount of confusion right at the top ranks of scientists, as the data was trickling in. It just didn’t look like they suspected it would look. That, in combination with Tom’s book, really just… I thought this story needed to be told. The challenge after that was the writing. A book that had come out recently at that time too was *The Emperor of All Maladies*, which was a New York Times bestseller. The author, Siddhartha Mukherjee, has won the Pulitzer Prize. It’s just an incredibly well-written book, but it was from this genetic angle.

So I really had the desire to tell this story. Tom had written a science book, but the story, like you alluded to, was so compelling, beginning with Otto Warburg and then going through this story of redemption where he was kind of ridiculed. This brilliant scientist, won the Nobel Prize, was nominated for three separate achievements, and then was ridiculed at the end of his career because of his overly simplistic view of cancer. But now, in 2012 – today – he’s made this incredible comeback.

**JM:** Well, first of all, let me provide a warning for the book, because it wasn’t without fault, I’d like to point it out. In my view, I mean, it’s a magnificent book if you want to give fair and equal coverage to both sides. But from my perspective, the genetic theory is beyond fatally flawed. We’ll talk about the reasons why that is the case. But I think you gave him too much coverage. I could have easily… In fact, I skimmed through 30 to 40 percent of it because I didn’t need to know about the genetic theory, I knew that it was wrong.

But the rest of it is just so incredible. I just want to talk about Warburg for a moment. I had no idea. Really, it’s like a novel that describes his growing up in Germany and how he actually got his MD. I didn’t know he has an MD. He has an MD and PhD, but not only that, he was recognized as the most brilliant biochemist in the 19th century. Hands down, no expert will refute that. I didn’t know that. I just thought he was some chemist.

**TC:** Yeah. Fascinating character. He was all that, and he existed in this kind of golden age of science where these guys were given every resource they ever requested. Otto Warburg wrote one request for grant money and he scribbled it on a napkin. Today, the typical scientist spends over half of his time just fighting for grant money. So these guys
were able to operate in this environment that was so conducive to good research and knowledge. And Otto Warburg, unquestionably one of the brightest minds and advanced physiology and biochemistry so far at that time… But he wanted cancer to be his... He was attached to forever.

**JM:** That’s what he wanted to be remembered for.

**TC:** Yeah. So he dove into cancer in 1924 and noticed the striking metabolic anomaly that we’ve all heard of, probably, the Warburg Effect, which is that cancer cells overproduce lactic acid in the presence of oxygen. There’s no reason they should be doing that. It’s very striking. It doesn’t make sense. Why would cancer cells be doing this?

If you remember Biology 101, a cell can produce energy in two ways: it can produce energy aerobically, and it does this in these little organelles called mitochondria, or it can produce energy anaerobically, and it does this in the cytoplasm but it generates a toxic byproduct called lactic acid. So if you ever lift weights or do some sprint-type exercises, you’ll revert to this ancient energy creation called anaerobic energy generation. But then once you’re done, your cell instantly reverts to aerobic energy generation, which is far more efficient. Thirty-two times more adenosine triphosphate (ATP) is generated through this method.

Cancer cells have reverted to this ancient method of energy generation and Warburg wanted to know why. And so, later on in his career, he concluded that the reason there’s this damage to the apparatus of aerobic energy generation – and the word mitochondria had not been developed...

**JM:** He didn’t know about them.

**TC:** Right. He didn’t know about them yet. He called them “grana,” these sort of granular objects that they could detect in the cell. So that was his theory, his conclusion. He was a very succinct guy, he hated excessive words. His conclusion was the prime cause of cancer was reversion of energy generation from aerobic energy generation to fermentation, which is anaerobic energy generation. That, he said, was the prime cause of cancer until he died in 1970. Brilliant man, phenomenal career.

**JM:** He was also good friends with Albert Einstein and many other prominent scientists of the 20th century. I mean, this guy was in the inner circle of the who’s who of the top scientists of the 20th century.

**TC:** Yeah. Good friends with Einstein. In fact, Otto Warburg volunteered to go to war in World War I. He was on the frontlines in Eastern Europe. Albert Einstein was beside himself because here was this guy who’s the world’s best biochemist and he was off fighting a war. Albert Einstein wrote him a letter, pleading him to come back. He was just saying, “Your talents are not in the war. Surely somebody else could take your place. Your talents are best served for humanity here in the laboratory.” He finally listened to Albert Einstein and came back as the war was concluding.
But yeah, the inner circle was incredible. I think two or three Nobel Prize winners came out of Warburg’s lab, including Hans Krebs, who made the Krebs cycle, a very well-known biochemical metabolism. Hans Krebs actually wrote Otto Warburg’s biography. This is one of the most interesting parts of writing the book. There’s a National Institute of Health (NIH) scientist named Richard Veech, who – I’m sure you want to touch on the ketogenic diet – is one of the pioneers of the ketogenic diet. He got his PhD under Hans Krebs, who got his PhD under Otto Warburg. I was delighted to find that the lineage is still alive today.

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**JM:** There’s another interesting aspect of the lineage. I believe one of Krebs’ student, was Albert Lehninger who essentially ran a lab at Johns Hopkins, and then Pete Pedersen at Johns Hopkins, was really I think a first-generation disciple of Warburg. Anyone who’s taken biochemistry… I mean, Lehninger’s textbook, that is a standard textbook for you. So it’s pretty interesting about the lineage.

**TC:** Yeah. Pete Pedersen is another phenomenal scientist. Same lineage as Albert Lehninger, got to work with him in Johns Hopkins. I feel we’ve lost this really hardcore biochemistry, the integrity of that sort of mechanistic science when molecular biology came along. We lost those old masters. Pete Pedersen was one of those old masters of biochemistry, and an incredible amount of metabolism work came out of his lab at Johns Hopkins, which was kind of the continuation of Warburg’s original work. He saw how damaged the mitochondria were in cancer cells and documented the very, very detailed effect of the Warburg Effect, and extrapolated this to theory and treatments, and so forth.

**JM:** Absolutely. So one might wonder, with all these incredible science and the most prominent scientists of the 20th century behind this, what the hell happened? Why hadn’t we adopted this? Why is the whole system focused… Now, I want to spend a little time on that because I think it’s an important point. Part of the answer I think lies with Warburg himself, who I’ve read has been described as relatively eccentric. So that didn’t help, because he couldn’t tell a story. He certainly couldn’t tell a story as eloquently as you did.

Then the other component is he was a German, and he stayed and remained in Germany in World War II, which didn’t help his position in the scientific community. And then you’ve got Watson and Crick in ’54, with the Nobel Prize for figuring out the DNA, structured DNA. So you’ve got this catapult into genetics as the cause, and this whole focus on there, and I think it was competing. At least that’s my perspective. I’m wondering if you can elaborate and fill in the details.

**TC:** No, I think you got it exactly right. I didn’t touch on that much in the book, but you hit a very important point. He stayed in Germany. He was half-Jewish. He stayed in Germany. After the war, there were a lot of scientists who kind of disparaged him for that. It seemed like a conspiratorial effort to discredit him based on non-scientific reasons, just based on some kind of a grudge, with that regard.
So there was that going on. You know, I’ve heard a lot of evidence that… Yeah, he was a difficult person to deal with. Very arrogant. The people who knew him close liked him, but he could rub people the wrong way, so I think he didn’t feed his case the best way he could have.

**JM:** Was it strategic or diplomatic?

**TC:** Right. Exactly.

**JM:** [inaudible 13:25 – 13:26]

**TC:** Yeah. Yeah. The backdrop of that, while this was going on, there was this revolution in genetics happening. Watson and Crick discovered DNA in the middle of the century. From that point on, it really, when you follow history, all eyes turn to DNA. It had been known for some time that there’s mutations within the DNA of cancer cells. Everybody, all the resources were looking at DNA, the genetics. You kind of look at the series of experiments that really lock down the genetic, the somatic mutation theory of cancer.

In 1976 was the big day when Harold Varmus and Michael Bishop won the Nobel Prize for finding viral oncogenes within the DNA of cancer cells. There was a viral theory that was competing with it, that viruses could cause cancer, but nobody knew how. These guys found that the viral gene that was being inserted in the gene was just a copy of the gene we already had, but it was distorted. So now this somatic theory was off to the races that cancer is just a distorted version of normal cellular division checkpoints, mutations in other words.

If you look at all the evidence, if I was there, if we were there, we would be very certain that it was a genetic disease. If you looked at all these evidence that have piled up to make an almost irrefutable case that cancer was genetic in origin. But then you get to 2006 and the Cancer Genome Atlas project, which was, like I said before, to sequence the genomes of cancer cells to ferret out all the mutations that were thought to be causative for cancer. This was to be the final concluding effort to cancer. We’d know every single detail, every aspect of how it operated. But once the sequence was being started up and this data came out, it did not look like anybody thought. It was much more random than people have suspected.

So if you have 10 people in the room with, say, pancreatic cancer and you sequence each of their tumors, what you’ll find is there are a couple of commonly mutated genes, but from one patient to the next, there not much of a pattern. It’s very random. You’ll even find some cases with one single driving mutation. You cannot explain that through a genetic origin of cancer, through the somatic mutation theory. You’ll even find tumors with zero mutations.

Clearly, there’s something else going on, and the people at the top of the field have noticed this. They’ve had to retool the theory, one which is called “dark matter.” There’s something else driving cancer that we don’t know. Many of these guys don’t know of Tom Seyfried’s work, of Otto Warburg’s, all the evidence that is compiled to make this
argument for a different origin, an origin that states that it’s mitochondrial damage followed by an epigenetic response as an origin.

You can see how the genetic theory got locked down. When you look back at how scientific theories evolve and they get taken over over time, it’s so predictable. What’s happening today makes perfect sense. This has happened so many times in history. It happened with Albert Einstein, with his theory. It was not regarded in the beginning, and it took decades for it to really gain traction. There was a quote by Max Planck, who won the Nobel Prize in 1918...

JM: Who was a friend of Warburg’s.

TC: Yeah. Exactly. Yeah. That old theories don’t die until the scientists that have come up with them die first. So it’s this gradual process. It’s going to take a long time. We have to rewrite textbooks and a lot of things, too.

JM: Well, fortunately we are in the 21st century, and we have technology that did not exist back then, which is the Internet, the ability to communicate with large numbers of people. Thanks to your book and my new interest that you’ve catalyzed, I’m seeking to rapidly accelerate that process because it is beyond extraordinary.

If you seriously reflect on the fact that 1,600 people in the United States will die today, my best guess, at this point, 1,500 could have been prevented or treated, and not have to die. Cancer is not a disease we should be dying of. Maybe in 100, 110, or 120, but certainly not before that. If you are dying from cancer, there is something seriously wrong. We haven’t stated discussing what we can do about this, but the treatment is so radically simple, and explains this whole process. We can help accelerate that rapid adoption by the masses, ultimately the people initially I believe that can catalyze that are within the profession, because they are going to be coming kicking and screaming.

But interestingly, even guys like Jim Watson who got the Nobel Prize in ‘54 for discovering the structure of DNA, he’s even come around. Maybe you can share that story. Even Watson, who’s been unfortunately discredited since your book was written.

TC: Yeah. He’s an interesting character, too. He sort of leaves a wake of… He upsets a lot of people with some of his comments. He turned his focus to cancer after he discovered the structure of DNA. He’s been at cancer research for his entire life and was very focused on targeted therapy and the somatic mutation theory of cancer, trying to target the derivatives of the mutations within the DNA, the protein derivatives. These drugs have been very, very disappointing. At one time, we’ve always been told that this was the path to cure. These targeted therapies were going to resolve in cures. It’s clear that’s not the case.

It’s clear because of the data from the Cancer Genome Atlas, there’s just too much random diversity within the genes. It’s hard enough to find a target from one patient to the next. And if you do, there’s another phenomenon called intra-heterogeneity, which is the difference in mutations from cell to cell within the same tumor. So it’s almost like a checkmate, therapeutically from the targeted perspective.
James Watson has noticed this and said "I'm no longer espoused giving money to the Cancer Genome Atlas project, and if we're ever going to cure cancer, we're clearly going to have to go back to the days of Otto Warburg and focus on the metabolism to make any real progress."

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So, yeah, he’s made an abrupt shift in how we should approach cancer therapeutically. When you look at cancer metabolically, the whole paradigm of therapy has changed. You go from this targeted paradigm to all of a sudden you’re targeting the metabolism. You’re trying to restore mitochondria function. You’re trying to increase mitochondrial numbers. You can probably rescue some cells within a tumor and divert them back into living within the collective of the multicellular organism. They will revert to being normal.

Some you can send over this tipping point. You can kill them through these various therapies metabolically. It’s an interesting time. The paradigm of cancer is being turned therapeutically and our understanding of it.

**JM:** Yes, indeed. Let me attempt to summarize the current thought as to people who follow and believe in the Warburg Effect as the primary mechanism of cancer. Starting with Warburg, he’s the first person who noted that there’s a difference in the metabolism [inaudible 21:14]. They’re anaerobically metabolizing glucose inefficiently to create this. They have this relative primitive form of energy generation, and it’s inefficient. So the question becomes, what caused them to revert to this?

I think Pete Pedersen at Johns Hopkins did a magnificent… He took it to the next step and he actually determined morphologically that there’s a radically reduced number of mitochondria, which are typically several thousand in each cell, comprising about a third to 50 percent of the volume of the inside of each cell. These generate the ATP, the energy of our cells. If you’ve got a radical reduction of those, and the ones that are left are mostly dysfunctional, not even working, if they’re working at all, then you’ve got a problem. These cancer cells don’t have a choice. They have to revert to this primitive metabolism.

The mitochondria, when they’re healthy, actually send these signals, which you mentioned, epigenetic communication between itself and the nucleus. This epigenetic signaling from the mitochondria is actually what’s responsible for initiating a significant percentage of the genetic damage that has been identified so well from the DNA sequencing project that you mentioned, which majority of the scientists have been focused on. So that’s where we are. Maybe you can fill in the blanks and elaborate on what I just mentioned.

**TC:** That’s exactly it. It’s all about the timing. There’s mitochondrial damage, that’s irrefutable. We look at cancer cells and the numbers are vastly reduced. When you isolate the mitochondria, you look at them and morphologically, they’re messed up. There’s protein problems, lipid problems, and all kinds of structural abnormalities. There’s always been this debate. Why is the cancer cell doing the Warburg Effect? Why is it reverting to anaerobic energy generation? Nobody really tied that to the terrible
structure of the mitochondria. They didn’t have the retools to see the mitochondria, and now we do.

Tom Seyfried has done such a great job of piecing together these events. Once mitochondria are damaged… This relationship between mitochondria and the nucleus is so important in biology. I think we’re learning this more and more now. They constantly cross-talk, and mitochondrial health is correlated to the health of the entire organism.

The dominant theory of aging explains that you age because your mitochondria age. They take the brunt of metabolism. When you generate energy, you are whipping around free radicals. They’re constantly under stress. They get banged up and beat up, and you look at the antioxidants within mitochondria, there’s decline about 50 percent within age, with advanced age.

**JM:** Excuse me for interrupting, but part of the refinement of what you just mentioned is when they burn fuel, they generate these reactive oxygen species (ROS). But the question is what fuel are they burning?

**TC:** Right.

**JM:** They’re burning glucose. They’re burning dirty fuel, generating tons of reactive oxygen species, as opposed to burning ketones or fats.

**TC:** Yeah. That’s such an exciting time for…

**JM:** It cleans you.

**TC:** Yeah. It’s very exciting with the ketone research that’s going on. As a fuel, they’re incredible. They burn clean, like you said. Much less ROS or free radical generation. The clincher with this theory is that once there’s enough mitochondrial damage – there is, it’s called a retrograde response or epigenetic signal to the nucleus – once this happens, you start to see the genomic instability. You start to see the accumulation of mutations.

So the whole crux of this theory is which is first? The argument is the metabolic theory, this mitochondrial damage, is happening first, and then you see the mutations. The mutations appeared as the cause, but in fact they’re a downstream signal from the true cause. So you can see why researchers were led down this wild goose chase, trying to find what these mutations were and why they were important.

Now that we know this, and it’s being worked out in real time, we can go back to the mitochondria and ask questions, like how do we keep these things healthy and restored.

**JM:** We’ll talk about that at the end. But that is the exciting question to ask, no doubt about it. But I want to elaborate on Tom Seyfried work, because he really is an innovative pioneer. He put together some of the most profoundly convincing collection of studies that, really, I don’t know how any respectable scientist could refute. I’d like you to discuss those, with respect to transplanting the nucleus of a cancer cell into a healthy cell, and transplanting the mitochondria of a cancer cell into a healthy cell.
**TC:** Yeah.

**JM:** That was what happened. I mean, these studies recurred 15 years before he found them. The people who did them [inaudible 26:31]. I don't know, I don't know. And then he put it all together.

**TC:** Yeah. That was so interesting to find all that out. Tom really did dig those studies that were just kind of buried. We just talked about this with Tom and another scientist. We were saying how Google has just transformed research, because you can dig up all these studies, where it used to take weeks in the library to find, all these old treasures that are just buried.

Tom found what are called nuclear transfer studies. They were performed mostly in the '80s, and they were very simple, elegant experiments. There were all these questions going on, and once the technology became available to do this, all these questions were able to be answered. Simply put, the experiments took the nucleus of a cancer cell and put it into a normal cell with its nucleus removed. Then you grow the cells in a petri dish and you can inject them in mice and see what happens.

When you take the nucleus of a cancer cell, put it in a normal cell, and put it in mice, nothing happens. So you have these cells that have all the driving mutations that are purported to cause cancer, but you don't get cancer. What's going on there? There's something in the cytoplasm that's suppressing cancer. That's all they could say. When I interviewed these guys, we were so under the spell of the gene theory that we were trying to reconcile our results with the gene theory of cancer. If we had Tom Seyfried's book at that time, it would've been a different story. We just didn't have it.

They flipped the experiment, and took the nucleus of a normal cell, took out the nucleus of a cancer cell, and put the normal nucleus within the cancer cell. You grow them in a petri dish and inject them in mice, and something like 98 percent of the mice develop cancer. So now you've got this irrefutable evidence that something in the cytoplasm is not only repressing cancer, but is driving cancer. Those guys, when you read their papers, there was one sentence they allowed themselves to say. It was: "This is the first evidence for a cytoplasmic origin of neoplasms." Which it was, and they have no explanation for it. They couldn't elaborate on it further than that. But in light of Tom's theory, this all makes sense, right?

When I interviewed the top guys in the field (I won't say who they are), the very, very top of the field, and asked them about these nuclear transfer experiments, they didn't know about them, for one thing. When I explained it to them, they said, "Well, if those are true, they're going to turn cancer biology on its head." But they just hadn't been exposed to these data yet.

It's incredible. Tom did an incredible job of compiling evidence that builds up. It's almost like you're building a case for a murder mystery. There's just so much evidence here and there, and you connect all these dots, the nuclear transfer experiments provide so much compelling data. When you put it all together, it's impossible to deny that this, if not the origin of cancer, it has to be explored further.
JM: Okay. I just want to give you another standing applause for your book because… I’ve read 150 books for the last year, and almost every health book I’ve read is so annoying because all of these are just reshuffling stuff I’ve read hundreds of times, if not thousands of times before. There’s nothing new, maybe a little twist or something here or there.

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But yours is a completely different approach. Essentially, this has not been written about or compiled in one book before. And you, it’s very, very clear, that you took years – I believe it’s five or six years – to write this book. This book was not compiled in a few months. You did a lot of investigative research and it shows. It was so engaging to read. It’s like a novel. It’s so hard to put down, because you just want to read what happens next. Congratulations on that.

As I said, if you’ve got cancer or you know someone you’re concerned about, you’ve got to read this book. Continuing along those lines, because we’ve got limited time here… We can talk for hours. Literally, I can talk for days. This is my new passion in life, there’s no question. I’ll expand on that in a little bit.

One of the other intriguing stories you’ve discussed is that of a researcher who worked with Pete Pedersen at Johns Hopkins. A brilliant, absolutely brilliant, Korean biochemist, Young Ko, who I believe – and predict, if I may be so bold – that she will receive a Nobel Prize for her work. This woman is just beyond phenomenal. To me, she’s one of the stars in your book. [inaudible 31:17 – 31:20] an interview in the future, if she hasn’t had a challenge with a thick Korean accent. But she is brilliant.

Just like in every area of health, we have these stories where there are these major innovations, and yet they are impaired, stonewalled, through the bureaucracy and just human evil and greed. I believe she’s got the answer to a large number of intractable metastatic cancers. But that’s a separate story. Why don’t you tell us what happened, discuss the story of how she discovered it and what happened to her after that at Johns Hopkins?

TC: Yeah. Everything you said about Young is true. I respect her more and more every day, and I’ve developed a very close friendship with her. She’s brilliant, innovative, and creative. Pete Pedersen, who didn’t miss a day… I think it was 22 years, not missing one day of work. He’s this boot-strapping, Midwestern guy who’s quote to me was, “When I arrived at Johns Hopkins, I wasn’t the smartest guy, but I knew I could outwork everybody.” And he did. He literally didn’t miss one day of work in 22 years.

When Young Ko came to Pete’s lab, he was just taken aback by her, because she was the first person that actually outworked him. When I talked to Young, she said “I forgot to not leave the lab.” She said she had looked at her shoes after a month and realized they were worn out. She actually just immersed herself in this work. She slept in the lab.

Yeah, it was a beautiful story. Kind of like a Lorenzo’s Oil type epiphany moment. Here’s Pete Pedersen doing this continuation of Warburg’s work, and Young comes in and dives into this research.
What they noticed was cancer cells were overproducing lactic acid, so they have to produce more of these pores to let lactic acid out or the cancer cell will die from the inside out. It’s a toxic substance. It’s like leaving the garage door shut in an idling car. These pores are called monocarboxylic acid transfer phosphates, and cancer cells produce a lot of them to get the lactic acid out.

Young’s sitting here, thinking, “Well, you know, this is a functional difference between a normal cell and a cancer cell. How can we exploit that?” Pete was trying to do it through a sort of a backdoor method, from gene expression angle, and that wasn’t working. They were struggling with that. She remembered this compound she had worked with while getting her PhD from Washington, called 3-bromopyruvate (3bp). It’s a very interesting little molecule that looks like lactic acid, but it’s very reactive.

She started wondering, “What if we gave this drug and it could slip in that pore, that opening, that’s allowing lactic acid to get out?” Her first test, it was funny because she tested it... She did saline studies and petri dishes, and tested it against the common chemotherapy drugs, the big hitters, cisplatin and so forth. She did the first test, and 3bp just blew them away. She’s like, “this can’t be right.” She did the test a hundred times over because she just can’t believe her eyes. She continued to develop this drug. It went through animal models, and just blew everybody away. It melts tumors away. The preclinical data is incredible.

But then, Young got mired in this in-fighting at Johns Hopkins. It began with the Vice Dean of Research there. They were arguing about grant applications, and this sort of devolved into this multi-year lawsuit that hung 3-bromopyruvate up. There were sort of patent-fighting about it. That’s how it got derailed, otherwise it would have marched through clinical trials by now. It’s still pending. Young is working very hard. She does have offers on the table for clinical trials. It’s just a matter of getting it done.

**JM:** Yeah. I think maybe I can jump to this now. I’m in discussion with her to try to see to make this therapy available to everyone watching this, actually, who is struggling with cancer. My best strategy to make it widely available is to have Cancer Treatment Centers of America (CTCA), who initially refused her request. But I want to come to that from a different angle, see if I can convince them that this is something that they need to use now, to integrate in their program, or at least offer it as an option.

This 3bp, 3-bromopyruvate, it’s not a magic pill. It’s not a miracle magic bullet. It needs to be done. Every one of these treatments, the foundational aspect of this is to address the metabolic mitochondrial defect, which is to radically reduce the carbohydrates, the non-fiber carbohydrates, and increase to maybe 85 percent of the diet, the dietary calories, as the highest quality fat you can get, and not go overboard on protein.

So that’s the solution. If you don’t do that, 3bp will not work. Possibly, but it probably won’t. It just doesn’t. You need a combination. It’s not 3bp, as I mentioned. But it is, from my view, if you have that tool, you’re doing an effective ketogenic diet, and you’re not going to die in two or three weeks from your malignancy, I think you can turn around most all cancers. That’s my current impression. That certainly could be flawed, and will be revised in the future. But everything I’ve seen points in that direction.
**TC:** Absolutely. I think you’ve described it beautifully, from what we’ve seen, the data that’s come out with the dietary interventions and adding on synergistic metabolic therapies. That’s what it looks like. It looks like the dietary therapies, what Tom calls a restricted ketogenic diet, is sort of a foundation of this therapeutic approach, because it does incredible things to the body where it differentiates between cancer cells and normal cells.

When you switch from glucose metabolism to ketone metabolism, you put energetic pressure on the cancer cells because they have to burn ketones in mitochondria, which is something they don’t have much of. They’re put under this energetic pressure, and they’re put under oxidative stress. Whereas the same time, normal cells are given better fuel, oxidative pressure is reversed, they generate more antioxidants, and so forth.

We’ve noticed that once you put people under this dietary state, everything becomes more effective, even traditional chemotherapy, and even radiation. At the same time, you’re mitigating side effects because healthy tissues are able to withstand the toxic payload from traditional chemotherapy. But the exciting thing is you add on these other metabolic therapies, their synergistic mechanisms overlap.

Exogenous ketones are exciting. How far can we take that metabolic state to this tipping point where cancer cells, not only is their growth stopped but they begin to die? We can bring blood glucose levels way farther than most people think safely. Gluconeogenesis inhibitors, that’s one of the main culprits you contend with, with as far as keeping blood glucose down. You add exogenous ketones and people are put in this different metabolic state. They’re shifting away from carbohydrate metabolism.

You can take this really far, and it’s going to be very interesting to see how far you need to take it, and then add on these pulses of pressure, 3-bromopyruvate. Dichloroacetate (DCA) is exciting. Metformin is exciting. There’s all these new things that are very non-toxic and overlap in mechanism. I think you’re right, Joe. That’s going to be it. This foundational shift to a different ketone-based metabolism followed by these pulses of pressure. I think that will be… It’s possible to say that it’s going to be a cure, but that appears to be the best method.

**JM:** Well, we have it now. It goes to show that we’re winning the war on cancer, if there ever was a war. Hyperbaric oxygen is another one that can be used, too. But when you were referring to gluconeogenic inhibitors, is that metformin, or were there others?

**TC:** There are other stronger ones. Metformin does that as well. Metformin is a very unique drug. Nobody is really sure on the mechanism of how it works. We know it trims gut bacteria in a certain way.

[----- 40:00 -----]

It inhibits what’s called the Complex I of the electron transfer chain. It’s a gluconeogenic inhibitor. But it’s an amazing drug because…

**JM:** Let me interrupt you here because you and I know what that means, but perhaps 90 percent of the audience doesn’t. Gluconeogenesis is the liver’s ability to create
glucose from raw materials and then raise your blood sugar, which is exactly what you don’t want to happen when treating cancer.

**TC:** Right. Right. And it was originally developed as a type 2 diabetes drug because it would lower blood sugar so low, I mean, keep it stable. But then we started noticing all these strange effects. There was a huge epidemiological study where type 2 diabetes patients were on metformin and they noticed that they have these drastically reduced rates of acquiring cancer. So why would that be? What possible mechanism? It’s certainly not protecting DNA, so it’s operating through some metabolic [mechanism]. It’s restoring mitochondria. It’s tuning up the metabolism in a way that you don’t get cancer.

So, yeah, that’s an interesting drug. There are many interesting drugs like that. From a prevention angle, like you said in the beginning, we shouldn’t be getting cancer at these rates. I agree. If we can keep these dietary problems that we have in our society – this type 2 diabetes epidemic – down, which is associated with cancer, then we’re going to see a reduction in those rates.

**JM:** I have known of this work for some time. In fact, one of my mentors is Dr. Ron Rosedale, who first made me more fully aware of the role of insulin and leptin in the process of chronic degenerative diseases. That certainly plays a role with mitochondria. I think the mitochondria has a deeper pattern to it, though. But your book was a real catalytic event for me, and it has really inspired me to do this. I’m writing my own book right now. It’s not just focusing on cancer, because what we didn’t talk about… I mean, this whole talk has been about cancer.

But mitochondrial dysfunction is at the core of almost every single disease. Which ones? Unquestionable, for like 80 or 90 years, there is no respectable expert who will deny that it is the standard of care, the treatment for intractable seizures. No question. Hands down. That’s the best doing it. But since then, we’ve known with other diseases: neurodegenerative diseases, Alzheimer’s, Parkinson’s, obesity (two-thirds of the country is overweight), diabetes, heart failure, heart disease, and arthritis.

When you’re on this ketogenic diet, your inflammation goes down to almost nothing. I’m doing this myself. I just had my high-sensitivity C-reactive protein (hs-CRP) test to a 0.7. That’s about as low as it gets. I mean, maybe you can get it lower, but that is pretty darn low. So that’s what happens. Inflammation disappears. You just can’t have inflammation when you’re on this type of program, for most.

And then, aging. All of us eventually are going to bite the bullet. This does not cure aging, but it certainly allows you to essentially put the brake or take your foot off the gas pedal, which almost everyone’s is on, to accelerate aging. My guess is over 99 percent of the population is not receiving the benefits of this approach. Having 99.99 percent would be my guess, because they don’t understand it.

My book is going to be explaining this whole metabolic theory, tying it all together, and then giving them practical detail, which I want to go into a bit now, just to give them some knowledge. As we’re all apparently excited. Like, how do I do this? How do I do this? So let’s give them some resources that they can use to begin to benefit from this.
Because the purpose of your book, I believe – I could be wrong, but I believe – is to give them the foundation. If you don’t understand why or know the reasons, you're not going to be motivated to pursue a course of action. That’s the purpose of your book: to open up a broad picture for that. It has something about treatment, but it’s not a treatment for it all. It has some decent starting points, but it’s not the main focus. So why don’t we begin there and you take off from that?

TC: Yeah. I just wrote an article with Dominic D’Agostino about the ketogenic diet…

JM: I read it. It was good.

TC: I was fascinated to learn as much as I did about it. It kind of has a similar story with Otto Warburg's story. It was developed in the ‘20s – well, even before that – for the teens as a treatment for epilepsy. It was the standard of care in the ‘20s. It was the best treatment by far. It started when people noticed... When people with epilepsy fasted, their seizures were mitigated. Then, Dr. Russell Wilder of the Mayo Clinic noticed that the ketogenic diet is the maintenance of the fasting state. It’s dietary fasting, I guess you could put it. Nutritional ketosis.

So when you do this, an incredible amount of biochemistry is going on. When you fast-forward, like you said, you connect all these dots, you realize that so much pathology spins out from this metabolic dysfunction at the level of the mitochondria. It just shows you… In my mind, it shows you how far we've stepped out of our natural state of being.

Our natural state of being was to starve occasionally, to have these bouts of periodic caloric restriction where you enter ketosis. When you do that, ketone bodies are not just a fuel, they’re signaling molecules. You completely rearrange the architecture of your DNA. You start expressing new genes. You drop down the inflammatory genes and boost all these restorative genes. There’s mitochondrial biogenesis. All these incredibly restorative things happen.

You look at all these diseases that spin out of this metabolic dysfunction, from not entering the state of ketosis periodically. Like you said, type 2 diabetes and all these neurodegenerative problems. It completely blows me away that just a shift in your macromolecular consumption, from carbohydrates to fats, can stop seizures. That’s incredible. It can stop so much other things as well that we’re really… This dietary research really hasn’t exploded until this century. Now, we’re getting this, kind of reaping the benefits of all these.

But it goes back to Dr. Veech, who you alluded to earlier, who is the lineage of Dr. Otto Warburg. He got his PhD under Hans Krebs. He’s done a lot of this ketone body research, along with Dr. George Cahill. He made that comment back in the late ’70s or ‘80s that so many disease states spin out of function of mitochondrial decay, and the ketone bodies can potentially mitigate this process. We’ve known this for some time, but it just hasn’t been put together by experts like you who have noticed and connected all these dots.
It’s an exciting time for health, for general health. We can do these principles. Now we have exogenous ketones that may be of help to people who aren’t able to do the dietary restrictions to some degree. I think a book along these lines, it’s high time for that.

**JM:** Yeah. There’s no question in my mind, no one… This book does not exist on the planet. No one has put all the dots together about a comprehensive and extensive detailed approach on how to do this. I’m so excited and so passionate about this, I can’t even go to sleep at night. I wake up every two hours. I’m just so excited. It’s the only thing I’m working on. It’s just driving me because there’s just so many lives at stake, and that people are going to die prematurely if they don’t have access to this information. I’ve never been so driven before about this. It’s like a whole new phase of life for me.

I have a question for you. I know you’re not a clinician, but you’ve done a lot of work in this, and I wondering if you have an impression… Because I was (and still am, although relatively less so) a passionate fan of intermittent fasting for the reasons you alluded to. They go through periods of time when you’re not eating. I think there’s still benefit to that, but it seems to me (I could be wrong, I’d probably talk to some of these other experts to confirm it) that you could achieve almost all the benefits of intermittent fasting if you’re doing the ketogenic approach.

By that, I mean maintaining a non-fiber, carbohydrate intake under 50 grams, maybe 30 grams, depending on your size and activity level, and protein levels at one gram per kilogram of body rate, or maybe even lower, and then having the highest quality fat. So if you’re engaging in that type of dietary pattern and you’ve got gluconeogenesis inhibited, it seems to me you’re going to achieve similar benefits as intermittent fasting. Maybe even better, because if you go for extended time [without food], 16 hours, on a regular basis, there’s going to fuel that you’re going to potentially lose some muscle mass, which is a concern of this approach. You don’t want to lose muscle mass. So I’m wondering what your thoughts are and if you’ve reflected on it.

**TC:** Yeah. Those are the big questions of the day. What is the best approach to do this? Nutritional ketosis, we’ve seen all those metabolic markers improve like you mentioned, high-density lipoprotein (HDL), triglycerides, and C-reactive protein. In most people, those are drastically improved on the ketogenic diet. When you look at the mechanism, the ketogenic diet really is just the maintenance of the fasting state nutritionally. Periodic fasting, for a lot of people, is difficult because, like you said, you do transition to this induction phase when you do that. Your body is tooled up to burn carbohydrates, so when you fast, it has to retool.

[----- 50:00 -----]

During that process, you burn muscle as a gap, a bridge, to get you over to fat metabolism. So there’s some stuff going on there that may not be terribly beneficial. I don’t think it’s known exactly what approach is going to be best in the long run. That’s the big question. Is nutritional ketosis…

**JM:** It’s not known, from your perspective. We just don’t know.
TC: We just don’t know. Right. If it’s maintained for decades, is that the best approach or is it best done periodically? I think that’s the big question of the day, as far as that goes.

JM: Yes, because Tom Seyfried -- I’ve listened to him a number of times, and I interviewed him once, and I think I’m going to interview him again next month – he’s a big proponent of doing this water fast, this 5- to 7-day water fast, a few times a year, which I don’t… I have enormous respect for him, for all he’s done to contribute to this field, but I’m not sure that’s the best clinical [approach]. Because he has a PhD as a researcher. He’s not a clinician. My gut feeling speaks against doing that, and that there might be a more effective and less dangerous strategy to pursue.

TC: Yeah. Yeah. Tom, when he first came out with the theory, I think that was his first suggestion, that you can do a 7-day fast. That’s very draconian for most people, and tough. Realistically, most people probably aren’t going to do that. A ketogenic diet, you might get the same effect. It looks like you’ll probably get the same effect. A lot of people can do that, and so many people are doing that now, too. It’s kind of the in vogue diet now. It looks like you’ll probably get the same effect.

JM: It’s still a very small percent. My guess is way less than one percent of the population, probably one-tenth of 100 percent.

TC: It’s probably the circle that I’m in. Yeah.

JM: That’s who you know and you can be able to convince them. I have never given away a book as much as I have given yours away. Number one giveaway, because I think everyone I really care for needs to have this book. It’s just a magnificent, magnificent book.

Let’s get into some of the things that they can do. If you’re convinced, you read it, you’re motivated, there are some really good resources. One is Ketogenic-Diet-Resource.com (There are hyphens between those words). I believe that’s the name. I haven’t had the time to check it through, but it seems like a really good website. It’s free information. Let’s talk about that. Maybe you may even know the owner of that site.

TC: Ketogenic-Diet-Resource.com… Is that Dominic D'Agostino’s website?

JM: No, no, no. It’s a woman’s.

TC: Oh, that’s probably Miriam. Miriam Kalamian, she’s a nutritionist and she’s been kind of working with Tom Seyfried since the very beginning.

JM: Okay.

TC: She has treated… I don’t even know how many cancer patients. She's counseled them with the ketogenic diet. So Miriam has got this down to a science. If you go to her website, if you want help with the ketogenic diet, she’s a wonderful resource for that. [Editor’s note: the author of Ketogenic-Diet-Resource.com is Ellen Davis. Miriam Kalamian’s website is DietaryTherapies.com.]
JM: Then there’s another book you’ve probably heard of, which I’ve read after yours, *Keto Clarity* by Jimmy Moore. I haven’t read the website as much but it really does do a magnificent comprehensive overview of implementing the ketogenic diet. It doesn’t go really as much into the theories at all, but how you do it. He’s interviewed like 30 of the top experts in the site, Seyfried, D’Agostino, and lots of other people, who gave him his viewpoints on. That’s a great resource.

TC: Yeah, *Keto Clarity*. Jimmy Moore, I’ve met him at Paleo f(x). He’s been doing the diet. He’s got a website called Livin’ La Vida Low-Carb.

JM: Something like that. I think he weighed about 400 pounds at one time. He looks like you now.

TC: Yeah. He’s done a good job.

JM: The reason why is this stuff works. I’ve said this before about intermittent fasting, and everybody’s curious when you’re doing this approach. As we’re interviewing this, I went to our Christmas party at the office last week, and been exposed to these relative temptations. You’ve got the dessert bar and all these chocolate stuff all over the place. When you are in a ketotic state, and you have been for a while, it just…

Jimmy calls it “keto clarity” because it does incredible things to your brain. It functions at such a high level, and the normal cravings that you typically would have aren’t, they disappear. They’re not there. It’s not a struggle. It’s not like, “Oh, I’ve got to be so disciplined to do this.” No. There’s no desire to do it. Would it taste good? Yeah, probably. But, you know, it’s not a craving. The cravings are gone. It’s like a really… It’s almost a miracle. I’m wondering perhaps if you could comment on your experience with the mental clarity, which is, to me quite perhaps… I’ve never seen anything do that to my brain.

TC: That’s ground zero, I think, for the ketogenic diet, the brain. That’s the first effect you notice, this sort of… What I noticed is this calming effect. I just feel much more stable and calm. I can get by on much less sleep, which is a huge benefit. I never get those surges throughout the day, those ups and downs. If I get less sleep, I still feel completely fine. Within the brain are the most profound effects.

The Charlie Foundation, which is the foundation that Jim Abrahams – he’s the Hollywood movie producer of the Airplane! series – he had a son, Charlie, who had very severe retractable epilepsy. He went through all the epilepsy drugs there were, and was left hanging there, until he stumbled on this book [on the ketogenic diet]. He put Charlie on the ketogenic diet, and that night, he didn’t have a seizure. He stopped having seizures. It completely cured him.

The interesting thing is his kids, they don’t have to stay on it permanently. Whatever that pathology is, they can reverse it within a year or two. So they started giving advice. They have a dietitian there who gives advice to epileptic children on how to institute the ketogenic diet. But as the years have gone on, they’ve noticed that people are coming to them will all sorts of problems like migraines and hot flashes from menopause. All these things, sort of neurological problems. So they changed their name from The
Charlie Foundation to Help Cure Pediatric Epilepsy to that of The Charlie Foundation for Ketogenic Therapies.

They’re finding that the ketogenic diet helps people with this diverse array of problems: from headaches to depression to anxiety, and all these things. It seems an almost better state of being, if you can get past the induction phase. I think that’s the hard part for most people, is that two weeks or even a week where you’re transitioning over. That’s tough. But if you get to the other side and your body acclimates, then all of a sudden you start to reap all these benefits you’re talking about.

JM: Yeah. One of the other benefits – a sort of anecdotal story, but I think a really powerful one... Many people watching have heard of Tim Ferriss before, he is the author of *The 4-Hour Workweek*. He was recently struggling with intractable Lyme disease. It really debilitated him that he was essentially out for nine months. He couldn’t do hardly anything. He implemented the ketogenic diet, and Lyme disease gone. It was like a miracle. He’s been a big fan of it. He’s had Dominic on his show. He’s a real big advocate.

It can improve these chronic infections. Not only that but radically improve your resistance to chronic coughs, colds, and flus that people are exposed to on a regular basis. You just don’t get sick. You’re just phenomenally healthy.

TC: Yeah. It’s kind of surprising. I almost feel… I try to tone it down when I tell people what it can help, because it sounds like you’re crazy.

JM: Oh yeah.

TC: Yeah. But just so you know, it’s incredible how many diseases emanate from that point and what it helps.

JM: Now let me tell you what it won’t help, and I’m sure you’ll agree. If you want to be a bodybuilder or a highly competitive athlete... although you can compete, especially in endurance events. There’s no question that if you have absolutely improved your fat-burning metabolism and do exceedingly well in these endurance events. But for strength and some of these other things, you’re conflicting goals – versus health and longevity, versus big muscles. So it’s not going to work for that. You’re going to need more protein, protein’s going to stop and inhibit gluconeogenesis. It’s going to cause gluconeogenesis, your blood sugar will not get the benefits, the ketosis.

TC: Dominic D’Agostino might argue with you on that.

JM: That’s right, yes.

TC: You’ve seen Dominic, right?

JM: Well, I know he got the world record for deadlifting the most weight in 24 hours, I believe, or an hour.

TC: Yeah. He’s a beast of a guy, muscular and strong. He lives in a ketogenic diet. He’s done a study… I don’t think it’s published yet. It’s kind of getting kicked around by a
couple of journals. But they took resistant-trained athletes and put them on a normal
diet, the Western diet, and a ketogenic diet, and then monitored them over a period of
time. They found no decrease in muscle mass and no decrease in performance.

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The interesting thing... when you look at all those pathways, you're right. It looks like
that's one thing it would inhibit. But it's got this protein-sparing effect, because you burn
fat. So it spares all the protein tissue. I think the jury's still out. It may be that you can
even get by... if you want to get to an anabolic state and build muscle mass, you might
still get by with a ketogenic diet.

JM: Not only is it protein-sparing, but it is specifically branched-chain amino acid
protein-sparing, because ketones, they have a very similar structure to branched-chain
amino acids valine, leucine, and iso-leucine, which are the three of the most important
amino acids to build muscle. Fifty percent of the amino acid is muscle. It specifically
targets those amino acids, which is quite interesting. In addition to the ketones, they're a
powerful signaling mechanism within the cell.

TC: Yeah. Yeah. I think that's the next phase of the research, this signaling properties
of the molecules. Because we know as a fuel, they're thermodynamically incredible.
They're so superior to glucose. They just burn cleaner, they're more efficient, and
there's more energy there. When you look at the ATP content of a cell burning a ketone
body, the ratio of ATP to ADP is shifted in favor of ATP tremendously. The ratio of
reduced glutathione to oxidized glutathione is shifted in favor of the reduced form, which
is the antioxidant form.

So as a fuel, it's incredible what it does within the cell. Now as a signaling molecule,
what it does to the architecture of the DNA and the expression of genes, that's equally
compelling. We're going to sort that out slowly. But that's where the new phase of the
research is, because it tampers down all these anti-inflammatory effects.

It looks like it has a very similar effect to just a sustained caloric restriction. The jury is
out on humans, but all evidence points to maybe not a huge increase in life span, but
definitely a huge increase in health span. Like if you were predisposed to getting type 2
diabetes in mid-life, if you were in that state, you may never get it. If you want to live
well for a long time, that's what you're after. That's what it looks like the benefit will be.

JM: I just want to make one comment on the clean fuel before we wrap it up. This
mitochondrial theory of aging, which is so present but refined, because we know that
these reactive oxygen species are, in fact, not all dangerous. We need them. They
actually are important signaling molecules, and if you suppress them indiscriminately,
you are going run into complications.

The problem is, you don't want an excess. That is exactly what you're going to create
when you burn glucose as fuel, which 99+ percent of the people watching this are
doing. That's what they're doing. They're not burning ketones. It's kind of like having a
car, a regular gas car. Of course, we know that when you burn gasoline, no matter how
efficient or new the engine is, you're going to have some pollution. That's just the name
of the game. That’s why your catalytic converter is in there, to minimize that pollution. Well, guess what? I’ve been driving an electric car since 2012. There’s no catalytic converter. There’s no muffler. It’s clean.

Ketones are the same way. It burns really clean. Do they generate some reactive oxygen species? Yes. But you need some. The goal here isn’t just indiscriminately take large amounts of supplemental antioxidants to suppress these things. The goal is to minimize their production. There is no doubt in my mind that’s the most powerful effective strategy. Let your body’s intuitive wisdom figure it out, and not you trying to second-guess it with some supplements.

So that’s the key. Let the body do it. That’s why I’m so excited about this. You want clean fuel. Once people get that, then they can get motivated and understand that they can do it. That’s why I’m so excited, because it’s the core of all these other diseases. It’s just the diet. Yes, there’s some mitochondrial upregulating approaches you can do and that’s what we talk about, but that’s the final 10 percent. Maybe 15 percent. But 80 to 85 percent of it is the diet. No question. That’s all it is.

**TC:** I couldn’t agree more. I completely agree.

**JM:** Yeah. So that’s my take on it. Now I’ll let you reemphasize any points you’d like to, or add some new ones, and then you can summarize it.

**TC:** You know that last point you made, just about the fuel, from glucose to ketones. I would always tell people glucose isn’t inherently evil, from a cancer perspective. It’s probably not going to cause cancer, but it’s certainly fuel for cancer.

I had a long discussion with Young Ko about that. She’s got so much unpublished data in her lab, just a treasure trove of stuff. She said actually, if you just give too much sugar to cells they start exhibiting all the phenotypes of cancer. So I was very surprised to learn that glucose by itself, at least within that model, can start to shift the cell towards… What it does is it upregulates the expression of this super important enzyme called hexokinase II, which by itself, is responsible for the Warburg Effect and responsible for a large degree of the immortalization of the cancer cell, not allowing it to die. It inhibits what’s called apoptosis, or programmed cell death.

Young noticed just feeding or giving cells too much sugar, they start to upregulate this enzyme that is so devastating for causing cancer for the shift to the cancer phenotype. I kind of have to revert, take a step back to what I was telling people. Sugar may actually be contributing to the cause as well.

**JM:** Yeah. Hexokinase II is actually as pathologic [inaudible 1:06:02] hexokinase I, and is the very enzyme that 3-bromopyruvate inhibits.

**TC:** Exactly.

**JM:** It’s the metabolic target that it focuses on and smashes that cancer death. It basically allows the lactic acid to build up and poisons the cell to death.
TC: That's it.

JM: Are you working on a new book? What have you done since you’ve written the book, personally, and are there any books in the works?

TC: Since I’d written the book, I’ve started a little foundation that has finally got enough money that we’re supporting some new research. We’re doing some very interesting studies, 3-bromopyruvate. We’ve done some really interesting stuff with Tom Seyfried that I can’t talk about until he publishes it, but it's very exciting. So I’ve done that. It’s called Single Cause, Single Cure. I’m working with Young. We’re working together to find the best route for 3-bromopyruvate, however I can be of help in that regard.

With Dominic D'Agostino, we’re starting a blog. We’re sort of collaborating on a book. Not sure what’s it going to look like.

JM: You’re doing it together?

TC: Yeah. We’re coauthoring one. The blog is going to be called Metabolic Optimization. I hope to have some interesting podcasts with some of the scientists involved in all this. I’ve been extremely busy. Writing a book, I know you have to get the right state of mind because you have to retreat from society for a while. I'm not ready for that right at this very moment, but I'll probably be coming soon.

JM: Well, to be in the right state of mind you have to be in ketosis, nutritional ketosis.

TC: Yeah, that helps.

JM: I’m so grateful for you being inspired with all this, and doing all the hard work, because there was a lot of work in five, six years, to compile this book and really present just a very powerful argument. If one is open-minded and carefully reviews the material and the references that you put in the book. I don’t know how you could come to any conclusion other than the one that we have both reached.

I’m sure we’ll be talking again together. I’d like to work with you and Young, because I’d like to see her therapy available to everyone watching this. Within the next few years, people will know this program, there’s no question. But they are going to still need resources for advanced cancer. This will prevent cancer and this will help treat most early cancers, but if you have advanced cancer, you need some very sophisticated interventions. It’s not simple. You need professionals. There’s a lot of people in that case right now.

Hopefully, we can have some resources that they can avail themselves to, that the foundational principles of this program are integrated into a more comprehensive strategy that will help eradicate and put them in permanent remission.

TC: Well said. Thank you so much, Joe.

JM: Alright. Well, thank you for your time and all the great work. And again, the name of your book is Tripping Over the Truth: The Metabolic Theory of Cancer?
TC: That’s correct. It’s on Amazon.

[END]