Discover Cancer's Origins From A Different Perspective Transcript

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Introduction to the cancer theory discussion

Hello, everybody. I am incredibly excited to have this very meaningful conversation today with Mark Lintern He is the author of the cancer resolution Cancer Interpreted Through Another Lens.

This is all about the cell suppression theory that he has, burst forth into the world and a whole new perspective on the origins of cancer. Just to give a little context, before I dive in and let this brilliant man, share with all of you for the past 110 years or so, we have been focused primarily on this idea of cancer as a genetic disease or more formally known, the somatic mutation theory of cancer.

This genetic mutation theory has pushed forth $1 trillion industry in the field of cancer care. And yet, unfortunately, we have not made big inroads to both prevention or treatment of this disease.

And so the interesting thing to me is, we might have made a little bit of inroads on maybe overall survival rates, but we are doing nothing with regards to the explosion of cancer diagnoses, which is expected, per the World Health Organization, to double globally by 2030.

So in the last 15 years or so, another theory has emerged and been talked about a lot. In fact, you listeners might have heard about it yourself. It's one of my passions and purposes, which is this concept of the metabolic or the mitochondrial theory of cancer, which also started up about the same time as the somatic mutation theory back in the 1920s, and is sort of gone in and out of favor and in and about exploration and deeper dive in the last few years or so.

But this theory, you know, opened our eyes to the fact that we might be missing the boat in our focus on the genes for the last, you know, 100 years or so and so.

But it's still even with this, it's still not making a dent. It's not really taking full credit for any really impactful prevention and treatment of cancer.

It's better, but we're not there yet. So enter stage right. We have Mark. Mark Lintern started out as a lay person, graphic designer turned citizen scientist.

So wait a minute before you close the umbrella of your mind, please hear me out. In the last decade or so in medicine, I'm here to tell you that the biggest changes in the field of cancer understanding of cancer treatment of cancer approaches to cancer has not come from the big academic institutions.

It has, in fact come from the systems thinkers that are out there, such as the engineers, the physicists and the biohackers.

Mark Linternu2019s diagnosis and research journey

Sometimes I think we as clinicians or researchers get so consumed by focusing on the answer or focusing on one target or one cause and one treatment that we lose sight of the bigger picture, that we lose sight of the forest for the trees.

So when you are so focused on one thing, you naturally lose sight of that bigger picture. And so I'm really grateful that it's folks like Mark who are going to continue to keep us curious, keep us surprised, and keep us humbled.

So with that Mark, I'm really grateful to have you here today. Welcome. Thank you. Nasha. You have a wonderful introduction. it's a pleasure to be here. So thank you.

So good. And let's let's just get right in. I mean, first of all, can you please give us a brief synopsis of how you moved from being a graphic designer into this?

Someone coming up with a new theory on cancer. Yeah, it seems quite unbelievable, doesn't it? It's even unbelievable for me. when I think back on it, sometimes.

But unfortunately, as with a lot of people, who get into this field from outside the field, it's because of a diagnosis. And I had a particular diagnosis myself.

and at the time, I was 28 years old. So that shocks me. thought I was healthy. and, when I asked my doctor, my oncologists, what particular mutations had caused my disease because obviously, up until that point, I was under the consensus view that, mutations or cancer was a genetic disease that, they couldn't describe the reasons for the mutations or even, in fact, the mutations themselves.

It was very much open ended. And, that confused me somewhat, given the fact that we're constantly given the impression that, for definite that the, disease is genetic in origin.

So I thought I'd do a little bit of research myself. I still believed it was, genetic cause of the disease, because I just simply wanted to know, which DNA mutations cause mine.

And in delving in that initial research, it became apparent to me that actually, there's a lot more to the disease than than we realize. And that I felt like scientists, didn't from the genetic perspective, didn't.

But we're really struggling to understand of the disease. There was so much randomness involved, and that gave me a sense that we didn't quite fully understand the underlying mechanism.

and in that process, in the initial research, I stumbled across the fact that there were other theories. I didn't realize this, many theories, in fact, as at least seven theories as as several more than that, but at least seven credible theories, all vying to identify the underlying mechanism driving the disease.

And one of the main theories that, really impressed me in that period was the metabolic theory. now, I stopped my research after a short period of time because I was given the all clear, given that it was a, a rare skin cancer.

And I had it, removed. But then a friend of mine got cancer at the age of 30, and, unfortunately, she passed away after a year. But during that time. I in, had a deep dive into the research again in order to try and help her.

And through that process, I then developed a greater understanding about the different theories that were present the metabolic theory, bacterial theory, tissue organization, field theory, atomistic theory.

All these are the theories, the involvement of stem cells to the extent that after my friend passed away, I felt I had enough information to provide to family and friends, that I could just put into a leaflet to advise them.

There's a different way of looking at the disease potentially targeting that cancer metabolically as as you know, you do, and as I progressed with that, I did more research and I went down a rabbit hole, and I found there was even contention with the metabolic theory in some aspects.

and I absolutely agree with the fact that, abnormal metabolism in the Warburg fact are key, a pivotal mechanism in cancer. And, it appears as though the hallmarks of the rest of the hallmarks of cancer follow on from the instigation of the Warburg effect.

So I'm totally agreement with the metabolic theory that that abnormal metabolism is absolutely key in driving the disease. but because there are 1 or 2, contentions with that, then we can go into this a little bit more detail later.

I decided to carry on my research, and as I carried on my research years past, I thought that I would stop after a couple of years. But four years came to fruition, and I stumbled across some other evidence that could offer an

alternative interpretation of the Warburg effect.

and this also allowed me to explain, another fundamental aspect of the disease that often doesn't seem to be, addressed, which is the consistency of the disease.

So, for instance, that the somatic mutation theory or proponents of that theory, highlights that with cancer as a genetic disease occurs from random mutations.

Now, there's a consistency to the disease highlighted by the, the hallmarks, the ten hallmarks of cancer that show that, well, there's this consistency that can't really be explained by randomness.

Randomness kind of explain consistency. And this was a sticking point for me. So with offering this alternative view on the Warburg effects, I also was able to explain and understand, the consistencies that are involved in this multifactorial view, that there are these consistencies that can then lead to the disease through this mechanism I had identified.

So it all seems to encompass within this particular view. so from there I thought, well, I've got something quite substantial here. I'll continue with the research and I'll try and write a book.

And then eight years later, I write the book, get it published.

From metabolic theory to a broader framework

and, because I'm a layperson, I realized that, no one's going to take me seriously, even though I've got I over 800 references in the book supporting the theory.

And every, every aspect of the theory of produced is supported by evidence. And the only reason I got to reach that point or that conclusion, I realized that I still wouldn't be taking seriously. So, I then got in touch with a fantastic chap called, Robin Daley from the cancer care charity Yes to Life. Yes.

we worked on a collaboration for a year and a half, to have my theory presented to, some international cancer experts. Ten facts of which you were, a panelist of of one of the experts in front of an audience of over 200 medical professionals during a six hour, webinar.

And I presented my research, put forward my ideas and my theory. And, thankfully for me, after that lengthy eight years, the candidates that were present voted favorably to acknowledge the significance of the theory and that it required more investigate, not necessarily that it was correct, but that there was sufficient evidence to to support the theory and that it, you know, required further, investigate from scientists.

So and and I love that that so many of the studies we have in standard of care, in integrative care, the final conclusion often says just that, you know, we can't definitively say, but we definitely can encourage more investigation and even to reach that point to get published.

And even if your publication ends with that statement, that's a huge movement forward. It's like kicking the can down the road a little bit further as we learn and just want to highlight this, that when you first when when Robin brought you and I together, for the first time, when I got to be exposed to as you guys gave me, like a chapter, I think, a little intro of your book, I was, really honored to be one of the early readers and adopters of this.

You know, just to consider this concept. And one thing that happens for me is that my brain always defaults back to it's all about the terrain, right?

And the evolution of my own thinking over my, my own health journey through this, my own career, even you spoke to this, that, you know, we all started out in the genetic mutation theory.

If you're alive in the last hundred years, that's what you know, right? That's what's that's what's in front. And, front

and center and all of this. but then as my understanding evolved from my own journey, back into the 80s and moving into the 90s and beyond, I started to understand more of this metabolic, mitochondrial, you know, issue of the pathways that were really upstream and that they were, in fact, the protector of the genome.

And then at that point, when the genes express when those places, you, on the other hand, took my thinking, opened up my umbrella even further to consider, hey, there actually might be something upstream of the metabolic mitochondrial effect that makes the Warburg process wake up.

And for me, I always thought about it just generally in terrain. And you definitely allude to that in your writings. And you're you're kind of, like the what you've gotten to in your own thought process.

But there's one particular thread that is so interesting and so compelling that absolutely warrants more discussion, that I'd like you to let at the listeners know about what made you realize, hey, genetics.

Yeah, there's an expression of the genes, but upstream of that in this metabolic mitochondria. But guess what, guys? There's a few things we can't, you know, answer with that alone.

So let's go even further. And that's what you're here to talk about today. Absolutely. And I just want to say, well, what I love about your, the way that you approach the disease is not just that you're looking at the metabolic side of things because you're looking at, maybe starving the the glucose and the glutamine, as, professor C3 suggests.

But you've also taken on board, the tissue organization field theory that, you know, the terrain you look at the microbiome as well is is a holistic view that needs to be the approach.

And this is why it's so important, for patients to actually take the time. I know time is of the essence, but take the time to understand cancer theory, because you'll get to realize, the differences between these theories and that treatments are developed from the cancer things that have been, produced in the moment.

And at the moment. The cause of cancer is unknown, even though there are several theories that are more accurate than others, they they can't fully at the moment explain all of the hallmarks.

This is the thing that I was trying to do was trying to explain these hallmarks because, the metabolic theory comes really close. but as I was going through the research, that certain scientists and certain studies show that, I mean, just to go back to what Professor Zephyr you're saying is, he suggests that defective Oxford pathway with the mitochondria is the reason for the switch to the Warburg effect.

So defective mitochondria, essentially. however, in a number of studies, Michael Asante has shown, that if you target the ox loss pathway, you can actually, kill cancer cells, specifically cancer stem cells.

so there's this contention, small contention, where there's a suggestion that and under certain nutrient deficiency conditions, Ox can be reignited, which suggests to which suggested to me at the time, just maybe this suppression mechanism going on here, other than it being defective, because how if it's defective, how how can it it can't be repaired, how can it revert back?

So that was one of the main reasons why I continued my, investigations. and of course I went down many more rabbit holes, but I came across, an immunological paper which was talking about infection because I was looking at everything from an objective standpoint.

And in this paper, it actually mentioned a Warburg like effect resulting from infection. that made me stop for a second to realize, well, okay, let's let's follow this further because and they actually talking about a Warburg effect that's similar to, the to cancer that happens in cancer.

And indeed they are. and then I suddenly realized from that that there might be a potential explanation here for, the Warburg effect in cancer itself.

Could it be due to infection? So, I did a bit more research and I came across, a guy called, Professor Robert Navia, and he talks about his cell danger response model.

Now, I have actually got a, graphic for this. Just very quickly. I can bring this up, share my screen. Visuals are so helpful, especially if this is pretty heady for a lot of folks.

So great. I can see it perfectly. Okay, that's that's the one that I want to put up, but. Right. Forgive me. I'm a little bit hectic on the screen, so I'll, change it to something more simply, but very briefly explain the, cell danger response model is all about the repair of cells.

So when cells become damaged, whether it free infection, toxins, injury, stress, no matter what it will go through a cycle of three phases CDO one, CDO two, CDO three.

And the whole point of these phases is to, initiate an inflammatory response in order to generate a repair process, the involvement of immune, immune cells to take away that destroy the pathogen or the heavy metal or whatever the toxin is involved.

And as it goes through these processes of repair, once it completes, the cell goes back to homeostasis, the immune cells move on, and all the normal proliferative signals, the switch to aerobic glycolysis that occurs in, a repair process is, reduced and normal homeostasis occurs.

So we'll have normal respiration occurring.

Cell danger response and the suppression model

However, Robert now points out that actually, cancer results from the cell getting stuck in the CDR two phase of the repair process. So which means it's it's proliferating within anaerobic like glycolytic, metabolic state.

Yeah. He's suggesting that the cell becomes damaged and the process is unable to move forward. And because of that damage, that's the cause of cancer.

Now, just before I carry on with this, I want to move to another graphic. Now, this is the graphic where I points out the difference between my paradigm and the paradigm of most theories.

And that is, that when you look at most of the mainstream theories, including the metabolic theory. Right. they all view the disease as a result of cell malfunction. So because carcinogens infection, they cause inflammation, cell damage.

And that results in something going wrong with the cell, similar to as I stated with Robert Navios. that CDR cell danger response model, they get stuck in this this, phase, this phase two, the proliferation phase.

So that's the idea, we have this, this process of some of some function and all these mainstream theories come in the paradigm of that. So you could arguably say there's only really one theory of cancer that's currently out there.

And that is the notion that the cell has gone rogue. It's become damaged and it's gained autonomy. Yeah. The problem I found with that was that, That is random damage occurring and that there's no real consistency that no one's been able to pinpoint, a consistent damage that can highlight a particular feature of the cell that then causes this switch to the more big effect to occur.

Now, Professor Thomas group will argue that, cardio lipid damage is consistent throughout, in mitochondria, throughout all cancers, which is true. However, cardio in on its own cannot explain.

the switch to, the Warburg effect of can go into detail with an emulator as well. But for the purpose of this and for time. so the point was that there's no precise, specific explanation of damage, that the moment can explain the consistency of the disease.

So going back to Robert Navios model, I was looking into his research and he specifically speaks about infection. And when he speaks of infection, he explains it as like this.

When a cell encounters, an invading pathogen, it intentionally switches off Oxford's the access pathway. So the mitochondria intentionally block its own pathway utilizing oxygen.

This six forces the cells switch over to glycolysis because it turns out that, glycolysis or the Warburg effect is an anti infection response uniform invading pathogens.

Now mitochondria do this because they want to utilize the oxygen that's being absorbed by the cell in order to flood the cytosol and, inhibit the invasive, attributes of the pathogen.

So the whole point of blocking Oxford's is to actually use the oxygen to inhibit the Pathogen's invasion, invasion process. So I want to pause you there, just to clarify, for both my brain and anybody else listening here, instead of which is what many of us in the field have kind of learned to believe or come around to believe that, you know, this is a cancer cell gone.

You know, a cell gone rogue. Yeah. You suggest that there's wisdom here of the cell that this process you're describing is more of a means of supporting the host versus going off, you know, going rogue.

Yeah. So it also has a very different energy behind it, like a different thought process behind it. So I think that that's really interesting and that what you're about to show us is that okay.

Well what is you know, you're like, okay, well here's the potential supportive mechanism versus rogue or attacking mechanism, you know, really blowing people's minds.

Like right here is where that philosophy really goes, you know, goes off course. So what I think I'm hearing and where I think you're taking us is that it's not that it's just the cells gone rogue.

It's rather trying to find a way to support the host being us. And unfortunately, that's where you're taking us in the next phase here, it meets a hijacker along the way.

Absolutely. So millions of years of evolution, cells have learned to, combat toxins and infection. And so what I'm saying is this process of the Warburg effect clearing is a response in order to help us, cells.

Cancer cells are not cells, in my opinion, that have gone rogue. And I've decided to develop a mind of their own, an attorney, evil and working against this.

They are part of, they've been working conventionally with us for, eons, you know, since our evolution started, way, way back. So in the process of them trying to aid us every glycolysis is the defense system they are using because for one, it allows mitochondria to free themselves up to target the pathogen once it's trying to invade the cell.

But to a robot like this, allow cells to absorb the glucose in the surrounding environment, which is the strategy that immune cells use, because specifically with fungal pathogens, glucose is the primary food source.

So it's a strategy for inhibiting the pathogen. Now, when I saw this, I was looking at this and it already broached the concepts in my own head of, a suppression mechanism being the cause of cancer rather than a damage mechanism, so to speak.

And I'm not saying that cancer cells on damage, of course they are. I was looking at it from that perspective, and I was listening to Doctor Robert Navios interpreting, which is that his interpretation is that the infection is cleared.

the toxins are cleared, bought, the cell is damaged, and therefore it gets stuck in this proliferative state. I'm I just asked the question at the time, even in my own head, I was saying to myself, this is how crazy I was in that was working for eight years on my own and in my own little study.

I was talking to myself, and the question I asked myself was, well, what if the infection persists? so if the infection persists and this is also confirmed in the literature that, the glycolysis or the work effect will persist until the infection is eliminated because it's an anti infection response.

So here we have now a process where infection actually can not only trigger the Warburg effect if the conditions are right, if the train is damaged because these are opportunistic pathogens, they are present within the tissue.

I'm not saying that you will get an infection. Suddenly you'll develop cancer. That's not the type of infection I'm talking about. This is, a nuanced infection.

but if the infection persist, then the Warburg effect persists. And then if the Warburg effect is an upstream event of all the other hallmarks, because it's producing these, metabolites, these corrosive, lactic acid, ion overload and all these other metabolites that are potentially causing dysfunction in the immune system and inflammation, chronic inflammation within the tissue.

Then you've got this chronic inflammatory process, which is ongoing because the the pathogen is not dealt with. and not only does lactic acid and iron feed the pathogen, but it also produces a corrosive environment, suppresses immune cells.

Infection, pathogens, and the Warburg effect

Now this is where it all beautifully kind of comes together. you begin to realize that if you've got a bout of, say, flu or whatever other illness, you've got chronic inflammation, you've got weakened immune immune cells because of this, you've got an opportune moment and you've got cell damage occurring, inflammation, chronic inflammation that you're not even really aware of, low grade chronic inflammation because of poor diet and what have you, you've potentially got an opportune moment, within particularly damaged cells if particular pathogens.

And I'm saying fungal pathogens, if those pathogens are present within that tissue, they have this opportunity to invade the cell. And if your immune system is not strong enough to defeat that, pathogen a moment in time, you've got a possible moment where an intracellular pathogen can sustain itself within the cell.

And over a short period of time, you've got proliferation occurring of all these metabolites occurring, when you have over past a certain threshold, if this these metabolize lactic acid ion overload isn't being, removed and pathogen removed.

It creates almost this barrier around this, this, tumor inflammatory niche that protects the pathogens from within. And and therefore, you've got this ongoing state of aerobic glycolysis, the Warburg effect and this proliferation and and a slow growing tumor because inevitable, inevitable.

The immune system is going to be drawn to the tumor to the inflammatory mass. And there's going to be this battle that ensues, depending on what, diet you have, your lifestyle, whether or not you're, you're eliminating toxins, you're eating organic, good, nutrient dense food that contain, the antioxidants and the anti-microbial compounds that can actually be utilized by your cells to kill the pathogen.

Unless you're you're doing this, then you won't kill the pathogen, and the tumor will progress all the while, if you are doing that and you are consuming a very healthy diet, then you're going to kill the pathogen.

You're going to kill the tumor, and you will never, ever know that you had the the beginnings of a tumor growth or cancer in the first place. and so just again, a recap here, because this is so elegant.

You know, again, we talked about it forever. We've been looking at somatic mutation. And that wasn't enough. We had to go upstream getting back to the metabolic saying, okay, that mitochondrial metabolic dysfunction, wound healing like this is also kind of a revisiting of Doctor Chow's work from the 1880s of a chronic wound healing process going amuck.

This is taking us up even further, saying you cannot turn off this Warburg metabolic effect if you do not deal with this pathogen. And so that's what we're stepping into here of what you started piecing like the, the, the the cycle became almost complete for you in your evaluation of this because a lot of, of folks stop at this is the one cause and this is the one cure.

And so because I've always been terrain centric and the the doctors I train in this arena, we're always looking at all the things, all the things that are going in the diet, the lifestyle, the toxin exposures, the emotional, you know, milling, you, all of those pieces.

So perhaps why maybe, one of our colleagues and myself might have better success with the patient isn't so much about the treatments. It's that we are inadvertently, accidentally making that tumor microenvironment less hospitable and less opportunistic for these infectious agents to take root and kick this whole cascade of events off in my on the right path of your thought process?

Absolutely, absolutely. And I would argue as well that, without damaging the terrain, you're not going to get cancer because you're not going to give these pathogens the opportunity which they need.

And the opportunity is a weakened immune system, weakened cells themselves because inflammation, facilitates the infection invasion process of these pathogens.

So you'll you'll with the toxins you produce and damaging your terrain, you will producing the perfect environment for these opportunistic pathogens to take hold. If they are present in that tissue.

It's not to say they will be present in that tissue. And that's one of the other reasons why and partly explain why. If you do get the mutations thought to cause disease, it doesn't occur because pathogen might not be present in the damaged tissue at the time.

So you're saying that as a way of explaining that too. But if I if I may, I go back to the previous, slide. so looking at this, this where we're looking at the summer function side of things, all the, the mainstream theories seem to fall under this paradigm.

and we can't identify the specific DNA damage that causes the disease at the moment. So the idea is that randomness can explain the consistency, which you can't say, I'm proposing the cell a cell suppression mechanism, and that is that carcinogens, cause inflammation and cell damage.

And this facilitates infection, leading to the suppression of key mechanisms within the cell, including the apoptosis and cell death mechanism, so that the intracellular pathogen, can sequester the nutrients it it requires from the cell in order to survive.

And thrive without damaged patient eating. So this then led me on to once I thought of this, it led me on to, explaining pretty much all of the hallmarks through this mechanism.

and like I said, I've got a lot of research in my book, to the detail of all this, but the beauty of this is that, I'm able to explain carcinogenesis, the entire process.

So the initiate initiation stays all the, all the processes through. And it also, I can explain the consistency of the disease, how it starts. through facilitating this particular of infection.

So a space just to stop the slide there for a second. Beautiful elegance. Thank you. the question then is did this real with real world data support? I'm saying, you know, for, for me, for my theory to have any valid validity that needs to be shown to be, pathogens within tumors, within cancers.

So this is where, Robert Strassmann and colleagues come into play. So around 2017, he discovered that actually all cancers, harbor intracellular fungal and bacterial pathogens.

Amazing. Chambers harbor their own microbiome, a chamber microbiome it's known as now, but prior to 2017, prior to his research, it was assumed that tumors were sterile.

So what we have here, I'm not saying necessarily just because microorganisms now shown to be present in all cancers, that they are the underlying cause.

They could cancer could have form first and they can you can. Enter the egg concept here. Right, right. Exactly. Correlation doesn't equal causation. But what I'm highlighting is that, actually this now, supports my theory and therefore it, it warrants further investigation, surely, to, to see if there is and indeed evidence to support the notion that if you do target certain pathogens, you can reduce the tumor and that so especially in pancreatic cancer Malaysia, fungi have been shown to, drive the disease.

Yes. Right. Yeah.

Therapies, off-label drugs, and holistic care

That's there's a correlation there with, looking into and this is why it's so important to, like I say, to reiterate that that point that it's important for cancer patients to actually study cancer theory, because once you do and you learn about my theory as well as a metabolic theory, you realize that it it's not necessarily just pathogens.

It's looking at the metabolism. It's looking at the strain, it's looking at the microbiome. But also that if you, are aware of the different theories that are out there, you can you can change your therapy or add additional, protocols to your therapy that are going to aid, your outcome essentially.

And ideally, the idea I would push forward is that and I'm not a clinician. So no, this is, medical advice, obviously, but it would be for my research to, try and understand and identify which pathogens present in your body at the time, particularly fungal pathogens.

And that allows you then maybe to target those pathogens to see if there you have another beneficial result on top of the other additional treatments that you are.

undertaking already. But, and I know you are active in and I'm part of a group of, clinicians and researchers around the globe that are looking at putting together clinical trials on this from cell line to human to animal studies, everything in between, that is actively underway.

So I know it's like you have to get curious. You got to start somewhere. You have to ask the questions. And so but part of this is we have had some sort of anecdotal evidence and you, and many others in the space have sort of accidentally stumbled upon the utility of some off label drugs or repurposed drugs or even just direct standard of care pharmaceuticals that we weren't quite aware of their mechanism of action.

But your theory starts to get our wheels turning to consider them differently through a different lens. Can you speak briefly about what kinds of tools might already be on the market that are worth investigating further?

Yeah, so there's a number of off label, drugs that are actually available. so the care Ecology clinic in the UK, oncologist in the private clinic, working alongside the NHS in the UK.

And I think there's a cow ecology clinic in America as well. they've identified, a number. And for off label drugs, metformin is one that of us that ins and November benders.

I know that doctor cycling as well. I did initially with these drugs was to target metabolism and this is exactly what they do and that they're showing efficacy.

But as I've gone through my research, I've noticed that pretty much most of the drugs that are currently in use, including these particular for off label jokes, are antifungal in their own right, right.

So that they can they're also targeting the fungal pathogens that I'm saying, the underlying drivers of the disease. so could it be a combination of both your targeted metabolism, because this is one of the beauties of metabolic theory and target targeting, metabolism, metabolism as well, is that if you're restricting glucose and glutamine, you're also restricting the primary food source of the pathogen within the cells.

It's taking advantage of absorbing that those, fuels. So it's very much worth looking at not only antifungal drugs, but other off label drugs as well.

But one thing I do want to emphasize is that, there needs to be caution made. I don't want anyone going away from this, and thinking the antifungal drugs, the answer to cancer.

Because whilst they may be beneficial in some circumstances, they are extremely toxic. For one. So you must always consult, obviously, a medical professional on this, depending on your medical history and what stage you are at.

toxic because, fungi also share, homology with our cells. So, cells and eukaryotic. Yeah. So, so the toxic to us, plus not all antifungals combat every single type of fungal pathogen available.

there's resistance as well. One, one of the big reasons for resistance is the biofilms that are produced that these microorganisms live within. That's that's one of the most important things.

So taking an antifungal drug can be toxic and it may not even work. So it's not a not not it's not a solution in and of itself. But I think one of the things that people need to realize, and I know you're pushing this, is that the reason why you got cancer in the first place is likely because, there's a fundamental issue with, the health of your body.

You're in a disease that even if you don't know it, you've allowed. Maybe it's not your fault necessarily. because we're just following, you know, reading us.

We don't know what you don't know until you know. Yeah, exactly. But, your body is is not in a healthy state. So your body is telling you that you've got low grade information, a lot of other things going on.

You've allowed the, the, pathogens to, populate certain areas of your body, and you've caused damage on a level that you're probably not even aware of.

And so it means that just using one drug is not going to solve all those problems that you've potentially caused by the diet that you're pursuing in the lifestyle it pursuing.

So really what needs to happen is a combination, a holistic approach where maybe you use, antifungals or natural antifungal compounds alongside off label jokes and alongside metabolic therapies and alongside nutritional therapies.

And look, really looking at your diet and really looking at your health and your mental health as well. That plays huge part in In Distress, levels, you know, glucose levels.

And these will play into feeding pathogens as well as causing systemic inflammation. So I love this. And you know, just to kind of just recap because this is just so it's just so elegant and so appreciative of of knowing you and knowing what it is that you're, you're throwing into the world for us all to chew on and consider here because it there's a, a level of just resonance with what you say, you know, and I'm hoping this gets people curious to dig deeper, to read your book, to look at your sort of, I guess, presentations or even courses, I guess in this arena.

But, you know, I just want to reiterate this fact that as much as we'd like it to be that simple, one target, one treatment, one pathway, one intervention approach, that's what we all, no matter the theory of cancer that you are beholden to, no matter what, we still we keep trying to make it that simple.

The environment in which these theories are made manifest is actually where our attention needs to be focused.

Long before a diagnosis of cancer, and in conjunction with a cancer treatment.

And so I really appreciate that you set the set the stage here that folks can't just go, oh, I'm just going to go out and get myself on a forced antiviral, you know, or through the antifungal medication.

And it'll all be good. I don't have to change a thing. If somehow it takes us out of the personal responsibility we have to taking care of this container in which that infection was able to take root, which then set off that, glycolysis pathway, which then set off the genetic vulnerability.

And the, you know, mesenchymal you know, organization of our cells and our microbiome, the whole bit. You just showed us sort of the ignition where the catalyst of where all this started.

But really where it starts is how we tend to the container that we've all been blessed in this lifetime. So I really appreciate that you also brought up the importance of reestablish connection to ourselves and to our self and to our mind, our bodies and our spirits.

So what final thoughts might you want the listeners to take away from this, and where can folks learn more?

Hope, resources, and where to learn more

Okay, well, just that there's hope that, because a lot of patients will have maybe a terminal diagnosis and then that terminal diagnosis, quite often is based upon, the DNA theory or the somatic mutation theory, interpretation of the disease, because most oncologists subscribe to the somatic mutation theory that cancer's a genetic disease.

So if you if you're looking at a disease from a limited standpoint, then you can only diagnose. And if you're using treatments based on that limited standpoint, the limited treatment options, then of course the disease can't.

It's going to look like it's a terminal disease, because you've only got a limited toolkit in which to, treat it. so the message I really want to send is that there's hope, because if you look at other theories, if you look at the metabolic theory, if you consider the notion of what the tissue organization theory is all about, the involvement of stem cells, the stem cell theory, all these come together to highlight that as a holistic approach, there's different ways of looking at the disease and targeting, the key mechanisms within the cell.

and that, by looking at these, I say that there's hope for you to look at other aspects of treatments that potentially work. Brilliant, brilliant. So tell everybody, I mean your book here.

I've got my little Vanna White moment here. Cancer. This is no joke. You guys. The the last few, you know, I don't know, not quite 100 pages, maybe 100 pages are references.

Those 800 plus references you talked about here. This is a really first. It's a really good primer on oncology in general. No matter your understanding of cancer.

Like this is something you work with. And the other thing is don't let this, you know, overwhelm you. if you're like the layperson, Mark writes this in such a way that's absolutely accessible by everybody.

It really is an easier read. There's a few things that stretch you a little bit, but you've got plenty here that you will grok even as a fifth grader, in my opinion.

And so I'm super excited about that. Where can they find this book? Where can they find access? And I believe you're offering a very generous discount to, some of your presentations and course materials.

Tell us more about that, Mark, and where they can just follow you. And this in innovative thought process and this up and coming theory of cancer. Okay. Thank you. Nasha.

the book is available on Amazon at the moment. and I have a website which you can view and download some free

information, free PDFs, a synopsis of the theory so you can get to know more about theory before you buy the book.

It's it's all written in layman's terms. the website is to be the reader without cellsuppression.com. and on that website, I also have available to purchase the presentation videos I provided at the February event, where I presented my research to all the scientists.

And my theory was essentially validated at that point. So that gives you a quick rundown. And it's about 40 minutes for all three presentations. a quick rundown of the research I presented and how I view the disease through this process.

so it's a quick way of understanding how the theory, relates to all the other theories that are out there and how it relates to the disease. that's also available on the website as well.

But by all means, download the free PDF. Since on that synopsis, I've got some questions to ask your oncologist and various of the little tidbits and helpful resources for for patients to view.

I'm so excited! Mark, thank you so much for picking our curiosity and for pushing us all to think about this. And a very different way through a very different lens.

So, Mark Lintern, blessings to you and thank you for being here. Thank you. Nasha. The pleasure's all mine. Thank you.