Introduction and Dr. Ahmed's Background

Hello, everybody. It's great to be back with you. And even more great to have someone near and dear to my heart. And to my mission and vision. This is Dr. Ahmed Elsakka who is now part of the Metabolic Train Institute of Health mission and vision.

But he is also much more and comes from such a beautiful background that I'm really excited to share with you, and to share with you what vision he's bringing to the current and future landscape of patient care in the ecology space.

So first of all, Dr. Ahmed, welcome. Thank you for being here. Thank you. Thank you for letting me be with you. That love it I love it. And so first and foremost, you are an expert in clinical metabolism.

But that wasn't always your your your jam. You started out in neurosurgery by your medical training. you were very passionate about neuroscience. And you have an uncanny nature.

I mean, I've met a lot of people in my career, but you have this blend of both the practice and art of medicine. So as the clinician as well as this clinical and laboratory research side of you, that's just as potent as your medical hat, but even more so, and you have this sort of alchemist product development side of you.

So you're like a three legged chair. Yeah. All of these, these qualities that all come together, that you are truly a master of both bedside to the bench and bench to the bedside, exploration and, you know, administration of really innovative therapies.

And I just it's, it's incredible to meet someone with your brain. I'm very excited. I'm going to say this here. I say this all the time when I introduce you, but one, you guys take a nice long look at Dr. Ahmed Elsakka because I have every confidence that this man will have a Nobel Prize in our lifetime.

So I say that because I want you to watch him closely, of what he's done and what he will continue to do. So let's talk a little bit about this. You moved from neurosurgery and focusing in this to kind of getting into the drug refractory epilepsy world and then beyond.

Tell us a little bit about how your practice and your philosophies have evolved and what what puts you on this path. I believe that everything is happening for a reason.

God, make me go through this path. what's happening to me in my life is not an easy thing to be. The first of all, I was a neurosurgeon, as you just mentioned, I love surgery, I love neuroscience.

At that time, I graduated from the Faculty of Medicine in Alexandria University. Then I spent like 5 to 7 years, in neurosurgical residency. Then during my, clinical training as a neurosurgeons, I was fascinating about epilepsy and epileptic surgery.

and to be honest, this was my subspecialty in the neurosurgery. and I was just focusing on how many brain operations have a problem with epileptic seizure.

So they may propagate seizures after the surgery because of surgical, manipulations. Brain is so sensitive. So when you touch the brain with a scalpel or with anything, a surgical instrument, it's very valuable for epilepsy and epilepsy.

the the eclipse, the after surgery usually is a drug refractory epilepsy. So I was just thinking about we trying to help the patient with the surgery.

However, actually, we harm him. We actually 95% of the patients is having, drug refractory, seizures. That's happening after surgery. And I was trying to focus on the epilepsy more.

And more. So during my digging and during my research, I was, I was invited by one of my patients who she's a girl, 21 years old, and she had, like, a low grade glioma.

And we perform a beautiful surgery for her. it was awaking to a new to me, which is a very new thing for me to breathe. I wish that one of his co operative and he can talk to you during the surgery.

And, it was at, a neurophysiological monitoring surgery, which is actually having connecting the brain with all of this wire and trying to prove a or stimulate an area of the brain before resetting the tumor to making sure it's not a functional area.

From Neurosurgery to Epilepsy Research

However, one of the most the problem was a new physiology monitoring during the surgery, if that the small electrical currents we used to stimulate the frontal cortex, all of them, the brain lesion, it's actually can propagate seizures.

So she had a seizure during the surgery. And she continued to give this fits of these seizures days and days after the surgery. I remember that I watch these patients for, for, for for continuous hours sitting with nothing affecting long hair.

And every time she fit in like that, they would have to sleep. So when she woke up from sleep, okay, she it again and again and again. So I remember my professor was looking at me like that and he told me, you know what?

If you're smart, you're still young. Try to find something, try to look into the decision to alter that and to find anything that can affect him. So I just made a basic research about the drug refractory epilepsy or post-surgical epilepsy.

And I go through multiple thing. One of them is ketogenic. But or in a broader terms, the metabolic anti-epileptic effect of the blood. So I basically read about everything that very quick in this day.

Next day I went to my doctors and I told them, okay, there is something called Genic diet and we need to put it in the fasting state. So what about if we just put it in the first thing stayed just water, giving her water and putting it in fasting.

So we did that. Okay. We, cut all the, carbohydrate is fluids, like dextrose or, glucose levels. We actually put it in the fasting state, just giving it only waters.

After 18 hours of fasting, she started to reduce the fits. After 24 hours, she stopped reducing significantly. She just had 1 or 2 minutes a day after today fasting, she was completely fit free.

So when we joined to introduce the food again, we introduced it by our own way. Because I'm not expert at this media, at that time I was I am trying to find a way for refractory epilepsy.

when introducing the food by wrong way, when we predict the keto that the ketosis state or the metabolic state that affecting the epilepsy she saw to having the foods again. Wow.

So good thing for me. Which means it's not like a coincidence because somebody may tell, okay, it's a new incident. It will, stop. she. Was just stopping anyway. Wow.

But it was responded very well to the metabolic ketosis. And once we break this metabolic ketosis by wrong way, it having that she had a fits again and again.

I mean what a pause you there because this is what I think is so extraordinary. You and your team performed life

saving surgery on a glial. Like I'm assuming a brain tumor.

Yeah, it's a tumor, right? Which that by itself could have taken her life. And so you saved her life by removing the tumor. But you left behind an effect of a new seizure disorder secondary to what you described as the vulnerability and sensitivity of the brain and a refractory meaning, not responsive or not well controlled by drugs to help with the seizures.

And so this young woman, at 21, she kind of jumped from one frying pan of, okay, I might die from a brain tumor to a loss of quality of life, of having just massive amounts of seizures like and waking up from that.

And like I said, every time she woke up, she'd have fits or these seizures. You'd have to put her back to sleep. That's no quality of life for a 21 year old.

She probably would have chosen to be dead from the tumor versus what was handed to her. So what you did was you got scrappy and started looking through the research and found a tool that you initially thought maybe was a winky dinky, a full on coincidence, and that you found very quickly had a much bigger impact.

Surprising you and your colleagues all alike. So that's where this journey started to go. Tell us what happened next exactly. At that time, it was I think that was like 2008 of 2009.

So at that time, do you think that it was just. Yeah, it was great. No, we didn't. Have secrets book yet. Yeah, yeah, yeah. And just to go through this felt so, I was fascinating about that.

And I was focusing on my patients or the future surgery and every single patients who will do the surgery. I trying to read more and more about the ketogenic diet and to apply a basic or to be up, to be honest, low carbohydrate diet.

It's not a ketogenic diet forever putting him into the fasting state after surgery, not rushing to give him a carbohydrate or, sugar based, IV fluids after surgery.

And I found that when we applied this maneuver, the ketogenic diet or low carbohydrate diet and fasting after surgery, we have a very good prior control on seizure.

So, it was repeated not only in one case, but in a lot of cases. So I want to point out the timing here. 2008 2009. This is before doctor Tom Siegfried's work.

You know, his book came out know he was doing research at the time, but his book didn't come out in 2011. But what I want your, the listeners to know, who might be new to this type of conversation is that we had been using a therapeutic ketogenic diet in the pediatric population since the 1920s as a control for epilepsy, and it was really a lot.

Let me oh, you're going there. You're bringing them there. Perfect history of oh, the key to learning by because it was before 1920, 1920 or 1932, exactly in the first anti-epileptic drug discovered, which is a phenytoin sodium.

By the way, it's still using till now. Okay. So when we first discovered that you can take it and stop the seizures, we underestimated the response of the ketogenic diet.

And this is the former word. When you have a bell in your hand, you totally ignoring the lifestyle modification that may be more important and more effective than the former.

So happening. Gelignite was invented so many years ago. Okay. Even before the invention of antibiotics. Right? What? Bring the ketogenic diet again to the scene is Charlie and Charlie Foundation.

When we having a problem with, the anti-epileptic drugs, he can he was at that time, he was receiving six anti-epileptic drugs with no response. So when the former failed, the first options came again to the market.

We went back to the old way. Exactly. Drugs. And it worked better, which is now that we, all of us, thanks to his story and the movie that was made of his life with Meryl Streep and others, and the ongoing work of Charlie Foundation in this, in this arena, it's what's given us clinicians, something to hang our hat on today.

To be honest, the first time I heard about metabolism and I heard about the ketogenic was from Charlie, so they helped us a lot to spread this, science to the world.

To be honest. I got chills. Yeah, to resurrect it. And then within that, it's amazing how close that community, the Charlie Foundation, the Brain Tumor Foundation, the neurobiology ag neuroscience, that's metabolic psychiatry, the metabolic oncology, we've all found each other.

It's like we all kind of came out of a slumber from the Otto Warburg time to the pre medication ketogenic you know time to all these things have kind of found their day in the sun again after some time and brought up into new information and new research that we've learned since the original research back in the early 1900s.

Amazing. Exactly. So what happened with me that after that I was deciding to know everything about the ketogenic diet? So I started digging and digging and sort to contact peoples outside my, country, outside Egypt, to find out how can I train more and more?

How can I find resource about this?

Ketogenic Therapy and the Charlie Foundation

And by the time I found myself dragged into the metabolic world, at that time, I was, I was thinking about biochemistry as a just a theoretical science.

As a basic science. We will not use the biochemistry in the patient. And we having this funny, thing that we usually say, okay, I didn't know what kind of sciences of the biochemistry, what he did to save a life of the patient.

And I actually was told that everything regarding the metabolism is about chemistry. So I started to study biochemistry again. Okay, I start to take a course in clinical applied chemistry and how the chemistry affecting the disease and affecting the metabolic, the metabolism of the cell.

So, at that time, I love I fell in love with metabolism, biochemistry, and I was in the very awkward situations I love neurosurgery, I found the future and, you know, surgery surgeons elective.

When you are a surgeons, you have been addicted to surgery to do surgery. Dopamine baby. however, I found a new lover. I found that the metabolism, the biochemistry.

I loved them too much. I found there is no future for this. If you know them, it is until now, however. Life, I love it. Yeah. So it was a very tough decision for me.

Wanted to continue as a human surgeons because it's very hard to do both of them together or to continue as a neural metabolism or indomitable is more.

What did the decision for me okay, is that a digit, accident of my mother's my was actually, in 2014 that by the way, since then I was studying the, the metabolism biochemistry from 2009 to 2014.

Wow. Okay. Did you ever think about metabolism, about biochemistry, about the basic science of them? At the same time, I'm trying to do my surgery. I'm trying to, have my decisions or to decide which can.

But I will be on the rest of my life. And I didn't take the decision yet at that time, in 2014, my mother had, some problem with the calf, in the shoulder injury, and she need to do arthroscopy, which is a very basic procedures, usually taking ten minutes.

Okay. No problem with that. It just arthroscopy, you know what I mean? There is no even surgical incision. So, at that time, the hospital that I worked for was a Newton surgery hospital.

And also having a sector in orthopedic. So, I arrange everything for my mom to do the operation in my, hospital or the hospital that was worked for. I pick the most talented, orthopedic surgeon for his.

The most talented, and I think the effort here, and I see the doctor for it. Everything was arranged very well. Yeah. My mother at that time was a 55 years old.

She was still too young. She didn't have any kind of chronic communicable disease. She didn't have a baby disease or hypertension or she didn't take any chronic medication.

She was perfectly healthy. So she went to the surgeon. And not having was here, she spent like one hours, two hours, three hours in a very small procedure and simple procedures that supposed to take only 15 minutes.

And I started to ask, okay, what's happening? My mother still in the operation room for more than two hours, but now, at that time, the, anesthesia doctors talk to me, and he told me, okay, we have a problem with your mother.

Actually, the surgery is completed and the surgeon is out since two hours. He finished the surgery in just 15 minutes. However, your mother didn't woke up from the infusion, and we don't know why.

And you will transferred to the post-surgical ICU room and trying to investigate more to find out what's happening. Who's here. So at that time, my mother was in deep coma and we don't know why she didn't woke up from here.

and then day after day, we sought to find, as a strange sign. We start, she start to have seizure, which is, ironically, have seizures at that time. And when we perform an MRI for her brain after surgery, we found first we found that she had, a problem with herpes and ganglia.

She has, like, obesity or hyper dense early, that is ganglia. We don't know why at that time. And then today or three days later, we found that she developed subcortical hemorrhage.

She spent 14 days in deep coma. Every single day she have a new neurological honest. And I'm a new decisions and I can't help him. Horrifying. All the science.

I worked with all my professors and all the years I spent in training. We can't even explain what's happening to her. Long story. In short, she died after 14 days.

Unfortunately, and she died with her secret. Nobody know anything about that. But it keep me alert and something enlightened in my head. Okay. What? The purpose of science and medicine.

If I cannot help. My dearest one. To my loved. Ones. I meant it to be a neurosurgeons. And my mom. I can't help it. I can't even explain what's happening to her at that time.

I start to dig more and more, and to find the reason why my mom developed this neurological insult. And I found that because we had the family history of Parkinson.

So the Parkinson's. So actually at the family history, my mother didn't suffering from Parkinson's, but she was affected by some way and how. Because actually the Parkinson the basal ganglia disorder or disorder that ganglia.

And then there is a most part in the brain responsible for oxygen consumption. So when she went into the beach, she had positions during the surgery and the hypertensive anesthesia.

She had a jeopardizing brain tissue. And brain tissue is okay, but the mitochondria is not too well. Right. So when locally fusions happen with no oxygen saturations having to her brain, if she had a healthy mitochondria, she will accumulate and she will survive.

If she had a bad mitochondria, she will. Having this kind of, basal ganglia specifications or the problem most hypoxic central. Yes. So actually it was opposed to hypoxic encephalopathy.

And we managed her wrong. We put it into the hyperbaric directly. She had a problem with the mitochondria. So hyperbaric oxygen, high pressure at that time will not treat the high that the mitochondria will put more oxidative stress.

Exactly. that's why she developed the action, which is somebody can hemorrhage. And then she developed seizures and then she died. So okay, this is daunting or something coming from God to change my life to trying to change the patient life.

There is a many neurosurgeons like me or more talented than me, but there is no metabolic doctors, at least in my country. So I will get metabolic. I will be an unbeatable.

Mother's Case and the Shift to Metabolic Medicine

I will do everything okay to be this kind of man, to save as much people as I can at that time. And by the way, I found that the case of my mother's in arthroscopy is recorded three times, and my mother with the force all over the world.

Okay. And all of them having a mitochondrial disorder or jeopardizing brain tissue, mitochondrial disorders. So I developed my own protocol. And by the way, I treated two patients having the same problem with my mom.

Okay. After becoming a metabolic doctors and the other lifestyle that so your. Mother is still she's still with you through others? I know, I know, you know for sure, for sure.

And then I become a new metabolism and, opened my new metabolism center in Alexandria. And I saw to thinking about treating the brain of GBM, glioblastoma form with metabolism.

So, I start with, one of my patient and give an extraordinary response at that time, the first one of the one who performed the ketogenic diet before surgery and during surgery, in the, the surgical sections, while the vision on the fully ketogenic state.

And we found that only two weeks of, ketogenic diet or metabolic therapy before surgery and reforming the surgeon on ketogenic state, actually, we found that the tumor biopsy showing a very good response and difference.

Just in this moment when I published this, study as a poster sessions, then I told the United State in the metabolic health Summit, I meet with my professor of philosophy.

At that time, I introduced to him, and he loved my work. And we published together and we worked together, and I trained after him for so many years. so, I need to take this a chance to, to thank him so much for all his dedicated work.

And then I shifted from just a neural metabolism to with a word can call as you want. Yeah, yeah. And, my gosh, you are the game changer in this space.

And though we didn't set off to have this conversation about the therapeutic diet of ketosis with cancer and other conditions, this is the path path, the data that has taken and showing us the utility of that which we have other people in this summit discussing more in depth.

But what we've brought you here for today is for folks to understand one of your other areas, because you became a biochemistry geek in this and understanding of these redox pathways, this oxidative stress process, the having people kind of think back to where you know that if someone's already in high oxidative stress, that pushing more oxidative therapies from standard of care, conventional interventions like chemotherapy and radiation, or even what would be considered integrative or alternative therapies like high doses of vitamin C or even hyperbaric oxygen.

Those can have a backlash. And so you, in your journey, in wisdom of learning things from a very devastating, you know, pain to purpose story to applying what you learned from your mother's situation to other people who are still thriving today.

Because of that knowledge that you learned, you've also evolved into understanding things about, like I said, metabolic oncology in general, this oxidative process and when to press pulse, which is such a discussion that Doctor Siegfried has brought to our attention as well, and your, awareness of a therapy that is emerging not just in the alternative cancer space or the integrative cancer space, but is very highly sought after, to be studied in the conventional cancer space, which is really what we're focusing on today.

So I want you to talk to us about ion. My friend in general, before we give away what this process is called, let's talk about Ion and its role in the healthy tissue and how it acts in the cancerous tissue.

Okay, I don't have a very funny story with me. Also, when I start working with a cancer patient, I find that the most of them is suffering from symptoms and sign just like an iron deficiency anemia.

So any clinicians will see the patients who are difficult to express and he having a tachycardia. And he's very tired and very weak. And he he look pale.

Who will think about the iron deficiency anemia. But I found that every time I'm asking for a total iron profile for this patient, I found that the serum protein is elevated to skyrocket to.

And the Maritain the iron storage metric. Fact the the, storage of the protein, which is actually the opposite that I wasn't thinking or thinking that the protein is low.

So the suffering from an iron deficiency anemia. But I found that the having a very high storage of this iron in their body. I know that sometime it may be, acute is acting due to the inflammation or the chronic disease, but not to that level, not over a thousand, you know what I mean?

So this make me think more and more about the iron and the role of the iron cancer and I found that this area is underestimated too much can iron is very essential in cell growth in general in any normal cell.

Okay. Forget about the iron as an oxygen carriers because this is a very important. But this is the first mission of the iron and 13 to be at oxygen carriers.

However, iron is still having too many options other than carrying an oxygen. One of them is iron is a Co enzyme and a rate limiting enzyme of the. In the applications being made.

Since this is, which is actually every time your soul is divided, to have a new cell in one. The healing and inflammation in growth. In normal growth you need iron for that.

Got it. And of course because cancer cell division rate is so high. So you need to secure a lot of iron for this process. Yeah. Well the security this iron the sick was treat this iron inside the cell.

And because iron is so oxidative it can be turned to oxidative. For in this action called in two reactions and this drug the. So because of the over oxidation they will protect themselves from the harm of the iron by beating the iron into kind of protein protect it's transforming to oxidative form.

So when the back this item is protein it becomes storage iron in the form of facility. So actually ferritin is a kind of iron but it's not so subjected for oxidation okay.

And when they need it they take just one ferritin and go through the endoplasmic reticulum and perform a kind of autophagy called for do not. Then you move the back from the iron and use it.

So by this way there is no free iron. Was free iron in the iron that prone for oxidation? There is no free iron. All the

iron is bagged with absolutely protein.

And when we need it, we just back one by one and use it in DNA replication. There is no free iron, there is no oxidation. So I got the positive effect about which is a DNA replication and the negative effect which is a Fintona action.

I get away from it and also because sometime the filter not or the autophagy of the 15 okay. By a rate higher than the ability of the cell to resistant, he will it suppress the glutathione system, the antioxidant in the body.

So the cancer so love iron because it need iron for replication. However, the iron can kill the cancer cell because of the over oxidation, the cancer.

So try to protect itself by two mechanisms. First, it back the iron with the protein and tightly regulate its baking or fruit of AG. And at the same time it's overexpressed with the same system or antioxidant system.

So if any oxidation happen, I can reverse it and can buffer it at the same time. So the cancer is getting smart and utilizing the iron differently than the healthy cells.

Exactly the healthy. So it made the iron slowly because it had a slow rate of the diffusion. So there is no problem of that. The health also having a healthy mitochondria.

So we have a lot of anti mitochondria antioxidant enzyme like so. So this beauty is like catalase like seven groups. It is all of them can protect the oxidation stress coming from iron.

So is important in both non medicine and cancer. So cancer so need the iron more than that than normal cell because it's it's the rate of division is higher.

it protects itself from this test by production of glutathione system and try to get it the baking of diet the problem really quick. I want listeners to hear there are a few things that the cancer is preferential for in an energy like where it draws its energy from.

You have, you know, because of your passion, keep ketosis. We know that, you know, glucose is one of the favorite, favorite fuel sources for cancer cells.

Also in your work, especially in your work in studies in the metabolic oncology space. And with doctor Tom Siegfried, you also learned about the glutamine potential of being another preferred fuel source.

And what you are speaking to right now is a third preferred fuel source for the cancer cells, which is iron. Is that kind of source? No it's not it's not a food. It's it's not.

So that's where I want to clarify. So we did not iron to produce energy. You know what I mean. That that the difference between the glucose and I mean most of them is used to produce ETB to produce good clarification.

So however, to, to, restrict the glucose and glutamine or to interfere with the glutamine and glucose utilization, it's called metabolic stress. Yeah.

However, to increase the ion oxidation in the cancer or the oxy therapy in the cancer called oxidative stress. And this is a business. Got it. So it's choosing the ion not as a fuel source but as a, an ability to self-protection, self self-regulation.

It's use the ion as a signaling. It's used the in the signaling molecules to produce more cell to producing more DNA divisions.

Iron, Oxidative Stress, and Ferroptosis

so it's harnessing the ion to make more cancer DNA, cancerous cell DNA. Exert a biochemical. That's a good clarification because it's not about a fuel.

It's about a signaling process that it's dropping in. Okay. Thank you. Please carry on. So, then we from in 2009, which is very recent. Okay. They discovered a new bit or cell death.

We can is different than obtrusive a necrosis. We know that the cell this it may be abrupt loses a programable cell. This only process and programable cell that.

You know what I mean. However having something new cell death can is different than that. The traditional one it called and the name it fructose is error process.

The name referred to this is just came to the world in 2009. Before 2009, there is no proposals. They look at that. This definition is so dead due to accumulation of free label.

I a lot of iron in journals because accumulation of iron in iron storage, which is bagged in 13. It's protected from the internal reactions. But the free label iron is very ready to go through the internal reaction and reduce oxidative stress.

So it's a cell. This due to accumulation of free fire and failure of antioxidant system. So how do need to be have it inside the cell to be died because of that condition of iron.

The iron should be free three labeled iron okay. The cell should have a lock in this system. Or we need to say this system, right. Why do I open up this system?

Because this is the only site, the cytoplasmic antioxidant system. After the net, it's a net bitch. So the main antioxidant system in cancer, the group that I use cancer choose to look the mitochondria.

She can't reduce mitochondrial antioxidants. so cancer cell is more prone for fructose this than normal cell. And by the way, when I say produces a cell that's due to accumulation of iron and failure of antioxidant status, it's not only for cancer cell, but this also can happen to normal cell.

Normal person can die because of accumulation and failure of antioxidant. And this is a lot okay. In, brain trauma, traumatic brain injury and some alternate hemorrhage, the high, blood outside the brain due to trauma or subacute hemorrhage, the blood containing iron.

And this iron is oxidative, and it can kill the normal cell doctors. It's also happened with, in the in the process of secondary hemochromatosis, of being over iron transfused or blood transfused.

That's right. We see that a lot. And then we discover that this actually is involved in too many pathology. One of them is Alzheimer's. And you mentioned that Alzheimer disease as a form or part of the human disease or dementia is, therapeutic.

So this of the neurons. So who first we need to protect the normal cell from the therapeutic cell. This however for ptosis the good to kill the cancer.

So so and this is what bring the doses to the ecology markets. We need to induce cell this in cancer cell while protecting the human cell and multiple others disorders.

Fascinating fascinating. And so the actual definition of up ptosis is that it's a it's a different type of cell programed cell death. And it's relatively I mean it's been around forever, but we didn't classify it or characterize it or understand it until a recent time.

And yet we actually have been accidentally using therapy to assess protocols in the integrative oncology space for quite some time since at least the 1970s, when we started using high doses of IV vitamin C, maybe speak a little bit

to that and that, because that really does harness that Fenton reaction that you were alluding to.

As you see right now, we have initial factors that may, induce cell this due to of doses. The first one is the sequestration or increasing the deposition of reliable iron in the cancer cell, the free form of that iron.

And so the cancer cell. And the second thing is the antioxidant capacity of the cancer cell. So anything that increasing the available Earth, like Artemisia, by the way, Artemisia is being used in, in war recruit is being used in the metabolic oncology since became.

Yeah. But at that time they say we know this. So this but with unknown mechanisms. Now we know that we can have the artemisia actually, back the protein into its free label so it increases the chance of oxidation of iron.

However, there is multiple therapy like high dose of vitamin C will increase of internal reaction, will increase the oxidative stress, will consume much of the antioxidant of cancer.

So we'll call that glutathione on depletion. Deplete the cancer cell from the glute the same. It decrease the net age. And then we age, which is, reactive oxygen species.

The scavenger. So high dose of vitamin C, hyperbaric oxygen ozone any kind of oxy therapy will increasing the rate of oxidation, decreasing the reductions with increasing the chance of oxidizing cancer cell and die, and will decrease the chance of the cancer cell to fight against the oxidation reactions.

Exactly. So to for people to understand this, what needs to happen in the cancer cells need and what needs to happen in the healthy cells need to happen in tandem.

And they're polar opposites. So what happens is a lot of people have the tendency to say, let's just give all of our cancer patients tons of glutathione.

And yet that could actually protect the cancer cells from oxidative therapies. But you want to have this press pulse process of making sure you're keeping the glue to fine levels proper in the healthy cells while depleting it in the cancer cells to induce this cytotoxic effect of other oxidative therapies.

So it's confusing because people want it to be black and white, always this, never this. But you have two processes happening at the exact same time. And so a really good integrative oncology just recognizes how to protect the terrain, the environment surrounding the tumor, the tumor cells in the tumor metabolism.

While targeting the tumor or the tumor cells in the tumor metabolism. And it is an elegant dance. And why this should not be taken on as an individual.

It should not just be read about and applied. It should not just be. I'm going to go read a few things online and apply it to myself, because this requires very careful monitoring by the clinician to know when you're overshooting it, undershooting it, or even who was a good candidate for this therapy.

And so Doctor Ahmed could use speak to us about who would be your ideal patient for this therapy. Cancer patient for this. Okay. The ideal cancer patient for this.

Usually the patient who have, a high rate of treating. And so this, So endogenously elevated iron storage that is not being applied from the outside in.

Thank you. I'm just come to modify this process right now. So when you having a high protein level okay. Very high for the teen level usually you would be a good candidate to induce a cell.

This in your case of this to the cancer. However we should know that the inflammation actually can increase the folate delivered. but falsely elevated.

Okay. Just to add a marker, it's called the acute phase reactant inflammation and autoimmune disorders. Most of them doing that. You made a lot of cancer by the way.

Receiving a blood transfusions and receiving an iron transfusions not perfect doses, but because they have been a symptom and sign of iron deficiency anemia or the loss of blood or the having, lose more globin.

So they having, supplementation or they having a blood transfusion. So when I say that when you cancer patients with a high purity level, but this little level suppose not to be increased because of this supplement iron or blood transfusion.

So ask the cancer patients who had a high falutin level, you don't have an iron supplement. And the last three months, if he's saying yes, he he's not a candidate right now, you will be the first after he both in three months of blood transfusions or I. V.

iron transfusion or even oral supplementation of iron. So the iron need to be sick was treated as a board of cancer biology, not at the board of intended, including the iron inside the body.

And also we need to differentiate between the high iron of the chronic disease and inflammation of that disease and autoimmune disorder and the high on the cancer.

So yeah, that's why. You're speaking to. So just to clarify, before you go into this next piece, this is not a therapy for everybody, number one. And it's actually very rare.

Like it's it's not highly common to need this therapy is I think what I'm saying is that there's a lot of reasons why you could have high ferritin that are not related to your cancer, and you have to distinguish if it's cancer centric or other.

And so the other metric that you use is you look at some of those inflammatory markers and you look at various ratios, to determine if this is truly a therapy ptosis opportunity carry on value.

And also why it's not too common that those is in cancer because in good luck or we are likely that. So most worst case of the cancer is prone for heart disease.

Because once you have a high rate of cell divisions, most probably the cancer cell will need high iron to provide this fire rate of the cancer cell. And that's why the worst cancer ever, which is a been created carcinoma ketones and beat off mutations to mutation of the highly aggressive cancer cell is the most responded.

Cancer for the those. Because once you are having a high rate of divisions, you're having a high chance for for those. I love this. And so here's that opportunity that if you are a patient with a tumor or a blood biopsy that suggests that you have a cross or a Braf mutation, you look like you could be a very good candidate for flare up ptosis, at least on paper.

We can then look at your labs and go a little deeper to see if that's the case, because these are the cancer types that tend to be very aggressive very rapidly of, you know, DNA mutating and DNA proliferating, cancer cell proliferating, that is often very poorly responsive to standard of care or standard of care alone.

And so where the studies have shown and what network has shown is that this is often an ideal environment. And I've gotten to witness this in real time with many patients that I've shared with doctor I've met over the years to see this to be the case, because many of these patients are sort of sent out to pasture.

No one really knows what to do with them and standard of care, or even in alternative care. And yet here is a very powerful tool. So maybe just super briefly and then tell us about what is and what is Braf mutation. Just super briefly.

So people if they've heard about it they might just know what that means or what it stands. You know. So and beta is an inverse also or that's let me get us in.

RA is a signaling switch in the cell. Yes. When it's wild tired you also can turn on the signals so it can divide the cell when it's needed, like in wound healing.

And if you turn it off it will stop the cell signaling of cell cross cancer. So with Kira's or Beta or Eleanor's mutation, we have a damage signaling switch.

It's always on. Yeah. It didn't go off. So you have a continuous signaling. Of course. And that's why chemotherapeutic agent is not effective. Because most probably, having a bad response in killers or beat up even the radio therapy, even the immunotherapy, because they have a very, very high rate of, divisions.

so the thing is, we could be having a very high rate of division. They provide a very high amount of the ion inside the cell to provide the high or to, to be the bell of high DNA divisions.

You know what I mean? Absolutely. The very good opportunity for us to unpack this high ion storage inside the cell and to induce oxidative or internally action or oxidative actions inside the cancer cell, while at the same time, we shut down the antioxidant defense mechanism of the cancer cell.

And by the way, I you the goodness induce that. Yeah. When we try to induce it in the cancer cell we're giving some drug to to stop or switch off the antioxidant capacity of the cancer cell.

And we giving a drug to unblock the ion and to the free label ion to increase the compound reactions. Beautiful. And so we're I want people to hear this is not that you just go, oh, I'm going to read this protocol and I'm just going to put myself on it for God knows how.

I hate the word protocol. Thank you. Because, yes, medicine in general, specifically metabolism, specifically metabolism in cancer is highly individualized, which means it should be customized for every mission protocol.

It just a standard guideline that I follow is everybody, and it's completely opposite the situation right now. Okay. The medicine, the metabolism, the metabolic oncology is a science, but at the same time, it's an art more than science.

It needs to be customized and need to be, visualized according to the situation. According to, a lot of factors. And you just mentioned the genetic factor, which is a killer.

You know, it's not only the killer, somebody we have a bunch we we discovered, by the way, it's a new science. Okay? That's we're still learning.

Personalized Metabolic Oncology and Patient Selection

We just we're still learning. So every day we discovered a bunch of the genetic materials that can increase the chance of the. This later on. And the old time, we just we only use the kiddos.

And because then we discovered that there is a bunch of the genetic mutation that may increase the sensitivity and the response of the cancer. So to the therapeutic drugs or the to the sodas.

Okay, so any bunch of genes basically that controlling the antioxidant right, or the reactive oxygen species is giving genetic and the edge. Yeah. Or the fatty acids to is because at the end the reaction will oxidized the fat into lipid.

Brookside and Brookside will be the end destructive force of the cancer. Everything. And yeah. Yeah. So when you having, any gene mutation that increase the fatty acid senses or the decrease the antioxidant capacity of the cell, okay, most probably you will have a good chance for the tooth Excel this we found more than 12, kept of gene mutation.

12 gene mutation. Not only the killers of beta that can increase the chance of the mutation of that. This was done, and it. When you think about this in in the lab, it's different than when you think about the fructose in clinical trials.

So I had a chance to have a pre clinical trials and clinical trials in Egypt okay. I have a chance to run a cell culture and animal model trials in vitro and in vivo.

Physically we are thinking that okay so fructose is is sufficient. So what about if I give the cancer patients bio which by the way unfortunately a lot of doctors doing that right.

So what about if I give the cancer cell and then give this are this cancer. So a drug to unpack this oil and oxidize it can increase the doses. To be honest, theoretically it's a very good idea.

On paper it's good. exactly. beauty dish, animal model on on humans. We found a completely different scenario when we adding high fat to the cell cultures.

Okay, to induce this, it didn't do anything with the free ptosis. However, it increase as the cancer cell division rate. Yeah, which means we have in two culture or two animals, one of them having therapeutic drugs, only one of them having the drug with ion.

He will we will, expecting that when I give you ion within the block, you will have more action and you will live longer, and you tumors will die. We found the opposite.

Yeah, that. Decision us ion. She lived long after she died. The cancer cells that did excision is over. She live longer. And this is a cancer. So and so just to reiterate this and I've seen this clinically because like you said on paper it seems intuitive that oh, we should be giving our patients an infusion of iron before we give them some fair up ptosis medications or off label drugs or neutral nutraceuticals or herbal, you know, medications to induce fear up ptosis.

And I've seen exactly what you described clinically. And of course, the literature shows this. And your research showed this as well, that great on paper or in the petri dish, that might seem like a good thing, but in the human it backfires terribly and it increases their mortality rate, and it increases this oxidative overwhelm to the system that takes out the good in the bad simultaneously.

And so it's so critical for people to understand that. First of all, I am really, really, really against any form of iron supplementation in any patient dealing with cancer.

Anyway, period. much less somebody dealing with, you know, flare up ptosis potential process. But that's just something we can talk about in another time.

But I think. You. Listen. Once and unless it's lifesaving. Well, yes, there's that. But most people sit there. They do like you. Like you said, they look at, oh, low blood, blood count, low hemoglobin, hematocrit.

And they assume and they give. And yet to your point. To give you a guarantee it's always much chemotherapy okay. Go with the iron. Because I would say 95% of my patients who go to a standard of care in college, or even an integrative oncologist, have been wrongly placed on iron supplementation, if not 98%.

Like, it's really unfortunate to see that. And then when you look at variations in these patients, almost all of them are in the hundreds, if not thousands or even further.

and so I want to point out something interesting, because I know we're coming to the time here, but I want to give a really good example and throw your I'm going to throw a curveball into this.

So data on that. And I have a really beautiful patient that we're working on together who is the classic crass Braf mutated a thyroid cancer that's gone everywhere in her body.

And she this is a papillary thyroid carcinoma that historically for somebody like me, we've never even needed standard of care. We changed diet, we change lifestyle.

We do a few very basic things, and the patient goes into remission and has a long, healthy life. But she has these crazy mutations that makes it show back up again and again, louder and more aggressive than the previous iteration.

And we didn't know because we finally just got the testing to know why. Which are those mutations that doctor I met talked about? Her switch is always on for proliferation and growth, so we all been very excited because her OB ptosis is the approach we definitely know we need and should take with her.

But we did just get back some news that she also has AG6 PD deficiency, which is a mutation where she has a higher tendency to be extremely oxygenated very easily with a variety of therapies, even things as simple as methylene blue, which everyone's taking over-the-counter today in the biohacker world.

And so this is that place where it was talking about the importance of knowing your patient well inside and out from their blood and their tissue biopsy information to their SNPs or single nucleotide polymorphisms, their labs, their symptomology, the history of their previous treatments, the imaging, you know, baseline they have.

What I'm explaining to you is like all these pieces matter because if we had just jumped in and started grab ptosis hog wild, we probably could have harmed her healthy tissue simultaneously.

Now, I know there's a way. The doctor I met is working to how to press pulse her with this because it's still going to be a powerful tool, but we have to be very mindful of her healthy tissue while we're doing the therapy ptosis, because we don't want the fear of justice to overwhelm the oxidative process of her healthy cells as well, because she has a genetic issue that makes her vulnerable there.

This is a very unique situation, and one that, had she just shown up in another integrative oncology practice, they would have probably killed her by using practices.

Or had she started with another integrative oncology practice where they might have started putting her on a high dose IB vitamin C, they could have killed her.

ozone or any kind of aqua ozone hyperbaric. And she did do hyperbaric a lot of it, which we think is what exacerbated this to begin with. Now that we understand this crass Braf, mutation.

And she's got very, very high elevated ferritin. So here's I'm bringing this up for the listeners because I want to caution you again and again that this is a very sophisticated and very individualized, personalized evaluation necessary to see if this is the right tool at the right time, at the right dose, duration in combination, and that what Doctor Med is presenting here is a very powerful tool that is in current understanding and research.

But we are certain that it's going to evolve and we're going to learn more. And so because it's being talked about in a lot of circles today, we're seeing more backfire from it than good.

And yet we want to really have the listener here hear that this is a very powerful tool when it's applied correctly. And Doctor Rothman is one of a handful of doctors, I believe in this in this world, who knows how to think about it and approach it correctly.

And so I just want to make sure anyone listening doesn't just run off and read everything they can and apply therapists to themselves willy nilly. this is so critical.

And so doc and closing words like, how would you describe like I gave you the most challenging possible patient imaginable in this scenario, but what would you want the listener to take home from this conversation?

And where would you encourage them to to pay attention for what's coming in this field? A very important research in the conventional and non-conventional cancer space.

Okay. It's not, just changing the diet. It's not, just taking that short supplement with no harm. First of all, of the

medicine is doing what harm? So when you realize that even changing the diet may harm you if it's done by unprofessional doctors or health care providers.

So even the diet or the supplement can harm you one day. You just mentioned the z6, PD. I want to give you a very beautiful example. In the neurology ward, we found, that two children, having refractory epilepsy went in two clinical trials of ketogenic diet for, treating their epilepsy.

two of them children took this children's, suffering from hemolytic anemia, which is unexplained. There is nobody book about the ketogenic diet at all.

So after performing the multiple, blood tests they found that these two children is coming from, a village in Saudi Arabia. This village is had had a high, a very high incidence all over the world at 46 PD deficiency.

Wow. Okay. Because genetic thing in this village. So after deep investigation, they found that when you apply ketogenic diet you reduce carbohydrate and diet.

So one of the most important metabolic pathway using the carbohydrate in the body called Pinto's phosphate pathway is this phosphate pathway. When you taking glucose producing what's called NAD BJ, which is a cool enzyme that hugs red blood cells and protects the blood cells from the oxidation, especially the blood cells, because they have no mitochondria.

This is the only normal cell in our body have no mitochondria, and it's created with no mitochondria for no reason because it carries oxygen. If it having mitochondria do it having a severe oxidative stress.

Yeah. So the only antioxidant that protected red blood cells is and then in your in ADB it's coming from carbohydrate metabolism inside their blood cells.

When you before making to genic diet in normal patients okay you're giving from 20 to 25g of carbohydrates. Even if you're giving zero carbohydrate you're still able to secrete some of carbohydrate from gluconeogenesis.

That can be enough for red blood cells to support the reduced phosphate pathway. However, if you're having a genetic defect in the phosphate pathway enzyme to Z6, PD.

So when you restrict the carbohydrate in diet, you severely impact this pathway, you severely impact the antioxidant net. BJ productions. So when you reform the ketogenic diet with a severe deficiency patient of net, but you may harm him or you may put him under severe oxidative stress and hemolytic anemia.

Don't freak out. Right. She's a very rare. Yeah, it's a real disorder. And even just the patient can be shot but with a specific proportion. Okay, I was giving a specific not on direct antioxidant, not the direct antioxidant.

So everything is it we did and everything is, should be monitoring. It's not just the diet. It's not just a supplement. General treat. the first dose is in a chapter book or in the post on the Facebook, and then you apply it to yourself or to patient.

If you are doctors, you need to know everything about that. You can have. Unfortunately, we can harm if we don't know. So you need to know everything about that.

So my take home message for that we have a very good opportunity in the cancer oncology world, okay. In the metabolic oncology ward, we can be one day having, very, simple drug or off label drug that can kill cancer in some patient and more effective than $1,000 cost, chemotherapeutic lesions.

However, still, we need to know more and more. Still, it's new. subject. Yeah. My take home message for the patient. Do not apply everything was. I would be carefully monitoring by a trained physicians.

My take home message for the health advocate or the health care systems trying to investigate more, try to learn

more, try to train more and more before putting your patient in a kind of therapy that you do not know about it.

Yeah. I love it. I love it. Ahmed. You are. You take dosage, doctor, teacher to heart. You are such a good teacher for me, for our community. You've now trained over.

You know, you have now impacted over 300 clinicians in 39 countries. Directly just from our training program. You've been training clinicians in your own hospital in Egypt.

And other places around the world. You present prolifically at conferences. You show your research in posters and research articles in peer reviewed journals all over the world.

You are a force to be reckoned with, and I look forward every day to learning more and more from you. And I'm so grateful for your time today, and for your wisdom and for your your passion to make a difference in the patients that you serve.

Thank you so much, doc. Pleasure to meet you It's my honor.