Introduction and Book Overview

Well, doctor Dach, it's, such a pleasure to to chat with you again. I mean, you've you wrote this amazing book crank here can. Yeah. Cracking cancer toolkit.

I mean, it it's a it's a solid, solid book with I mean, this is a very extensive and I, I recommend anyone out there battling cancer to, to, to get this one.

So thank you so much for spend some time with me today. Oh, it's my pleasure. Michael, you know, we were just chatting earlier, and and you asked me, you know, how I got into, writing a book on cancer and, you know, I'm not, I'm not an integrative ecologist.

I don't see cancer patients, in my little office here in Davie, Florida. I but, you know, I specialize in, biomedical hormone replacement, as we discussed last time.

And natural thyroid, I consider myself a reluctant eye radiologist because I started doing hormone replacement 20 years ago for menopausal women and realized, you know what?

I have to if I, if I leave the thyroid up to the primary care doctor, they'll never do it, right? If I want to get it done right, I have to do it myself.

You know, my background, as you know, I, you know, I did a regular, internship. I rotated through medicine, a surgery to Rush Medical Center in Chicago, and then, I did a radiology residency.

And then, I did interventional radiology fellowship at the University of Miami. Jackson Hospital. And then, I became board certified in, diagnostic and interventional radiology.

And I worked in the hospital for 25 years that year in Hollywood, Florida, in the memorial system, doing both diagnostic and interventional radiology.

And, and because I had some eye trouble, I retired about, 25 years ago. And, I got a promotion. I started my own little office. We're where we do outpatient, medicine, you know, and I sort of return back to my old days doing, doing internal medicine, and, so it's, so we mostly specialize in, in, as I, as I mentioned, menopausal, hormone replacement, and, natural thyroid, but, you know, we're full service, you know, our patients have other medical problems.

We address those as well. So getting back to the book, I did, I read a book on cancer. Well, it turned out that, during, you know, during the early days of, my little outpatient clinic, three of my close family members, contracted, aggressive forms of cancer.

And, so I realized I had to get up to speed on, you know, the, the new, research because, current cancer treatment is, you know, that we're back in the 1950s and 1960s, as you know, you know, chemotherapy, was, you know, is, is really hasn't changed that all that much in, in 70, 80 years.

And, you know, there's been tremendous advances in, in cancer research, mostly pre-clinical, you know, in vitro and also, animal xenograft type studies, which, you know, and that so there's there's a tremendous amount of money that's gone into research, from the through the eye.

And, it's, since the Nixon started the war against cancer. So they've really, you know, dumped, billions into and, and a lot of it is, pre-clinical animal studies.

So we have this gigantic volume of research which is pretty much untapped, and so what I, I, you know, learn some of this material and used it and, for the my three family members with aggressive cancer.

And all three had good outcomes. So I thought, you know what? I should write a book about it so everybody could, you know, everyone else, you know, can, receive the benefits of this type of, this type of a book.

And, so just to kind of these three family members, you know, you s you learned that they had developed cancer. You you dove into research and then utilized some of the things that you learned, and that benefited them on their cancer journey.

Is that my understanding that. Right. Correct.

Cancer Research Journey and Chemotherapy Critique

And, so, you know, one of the things that I learned early on was the, the chemotherapy as and this is something that, you know quite well, the chemotherapy treatments, will shrink the tumor temporarily.

But the net result, is that the tumor then recurs with a much more aggressive, cell type. And, that'll and the reason for that is that chemotherapy, causes a tremendous increase in inflammation, which can be measured, with, you know, the cytokine IL six is on the most commonly measured, which and, you know, the inflammatory pathways are the master controller is the nuclear factor, Kappa B, which is massively upregulated by chemotherapy.

So the net result of chemotherapy is that actually in most cases it makes the, the cancer worse, that it becomes, you know, the cancer becomes drug resistant and much more aggressive and, and spreads, becomes metastatic.

So, you know, the chemotherapy is sort of an I consider it obsolete technology. You know, the exception, being testicular cancer, where, they came with the the cancer stem cells are sensitive to chemotherapy.

And and that brings us to the cancer stem cell. Question. And the you know, what is the real reason why, cancers recur after chemotherapy and, the the bottom line is the chemotherapy eradicates the bulk cells, but leaves the cancer stem cells unharmed.

And the reason for that, is the cancer stem cells are hibernating. The they're actually dormant. Sort of like a hibernating bear in the winter. And they're and, they're not actively, dividing, and they're not actively, so chemotherapy, will, target the actively replicating, actively dividing cells in the, in the cancer and also in the human body.

And so, and that's the genesis of the most of the adverse side effects of chemotherapy is that we have we have rapidly dividing cells in the gastrointestinal tract.

The epithelium will will denuded our bone marrow. Cells are rapidly dividing so that that's targeted by chemotherapy. Patients will receive a chemotherapy, will develop anemia, and low white count.

So the combination of, Denuding edition of the epithelial layer of the gastrointestinal tract from chemotherapy, which causes leaky gut and, and, gram negative, endotoxin.

You know, that combination along with, leukemia, which means a very low white count. That's, that's a very, you know, highly fatal combination, you know, septic shock with no white coat.

Yeah. Yeah. That's not a good combination. The question is, I mean, do you mind talking a little bit about what cancer stem cells are? And also, I'm really curious, you know, here they're in the hibernating state while you're doing chemo.

What makes them all of a sudden get active? Yeah. Once that chemo is done, the, you know, the the reverse triggers for, for cancer cells, for example, lymphoma cells, lymphomas are known to be, dormant.

Many of them tend to be, slow growing, which, you know, in this measured by the K 67, proliferation marker, you know, many lymphomas and even breast cancers can be, you know, have a low K 67 and, you know, what triggers them to become more aggressive?

And, that's a good question. You know, the, there are some triggers, you know, in the environment, that will do that. One of the triggers is inflammation increased and inflammation in the body, the cancers, the vast majority of cancers will, will become much more aggressive in an inflammatory microenvironment.

And, and so that's one of the drawbacks of chemotherapy is that it creates a massively inflammatory microenvironment. And so, the, for so the, you know, the lymphomas, you know, some of them will respond, well to chemotherapy and you can, quote, have a cure, but the aggressive ones, just become much more they, they will, you know, regress.

And then the, you know, patients have a negative Pet scan, but then, a few months later, you know, the lymphoma comes back with a vengeance, and, and I believe that's the inflammatory, conditions in the microenvironment created by the chemotherapy, and it causes it to come back. Yeah.

And it makes sense. I mean, because we know when we have an injury in our, you know, body, let's say we hurt the knee or something, you know, that triggers an an inflammatory response to then signal to stem cells and signal to other, you know, growth factors to get to that location to repair the area.

So I mean, it makes sense and that the inflammatory environment and the tumor microenvironment, you know, really activates then the stem cells, cancer stem cells to try to heal that, that that damage.

Exactly. You know, I think that's that's a good point. Yeah. So, you know, the other thing that's perpetuated the chemotherapy paradigm, which, it's amazing how very few people know this and that is that, it's called the chemotherapy concession.

And the rest, most of medicine, it's illegal for a doctor to, self-refer. For example, when I worked in radiology, it was a large group. And, in our meetings, we'd always talk about how there are laws that prevent us from referring patients to, a, an imaging center that we own.

If we own an outpatient imaging center, we can't refer patients to our own center. It's illegal. It's called historic law. There are a number of laws.

Some state laws and, federal laws that they make it a criminal offense to, for a doctor to self-refer. And, it's, you know, it's these are called anti kickback laws.

It's, you know, it's illegal for a doctor to receive a kickback, for example, if I, if I refer a patient to the clinic down the street, and ask them for a kickback, you know, the patient generates, a certain amount of money for that clinic that I refer to.

And, and I asked for, a 10% commission, you know, which is what commonly done to real estate and other business transactions, but in medicine, that's illegal.

And the doctor can go to jail for doing that, for receiving a kickback. So for some reasons, inexplicable reason, there, the oncologist receive a kickback from the sale of oncology drugs from the sale of chemotherapy.

It's called the the chemotherapy concession that's been going on now for, going back to the 1960s, 40, you know, 60, 60, 70 years. And, it's not well known because, it's, you know, the mainstream media doesn't, doesn't publicize that the colleges are receiving a kickback.

And that's one of the main, one of the main, mechanisms that is perpetuating, an obsolete, technology called chemotherapy. It's basically, you know, an obsolete technology from the 1950s.

So I think, you know, it should be illegal for oncologists to receive the kickback. And, you know, if you think back, it was common practice for oncologists back, you know, the 20, 30 years ago to do, high intensity chemotherapy that would wipe out the bone marrow in women with breast cancer.

They would they would get, you know, they would because they received it receiving a kickback. So they wanted to use more and more and more chemotherapy.

You know, the more chemotherapy they use, the more money they would make. So they came up with the idea, well, let's we we can't give too much chemotherapy because it wipes out the patient's bone marrow and then they die.

So let's do this thing called bone marrow transplant. We'll harvest the bone marrow first before the patient receives treatment. And then after the bone marrow is totally wiped out and the patient is is helpless, they're going to die with, they then receive a transfusion of the bone marrow, the patient's own bone marrow, back into their body, which saves them.

And that's, you know, the bone marrow transplant for, breast cancer, which, and that was that was done. For many years until finally a, you know, it was studied was that was the subject of a, thorough medical study.

And they discovered, guess what? Surprise! It has no clinical benefit. It has no additional benefit. And, so the whole practice was discredited, and they stopped doing it.

Cancer Stem Cells and Metabolic Pathways

You know, you have other, other problems in, conventional oncology. The drug companies, decided to invent a, recombinant erythropoietin called procreate.

And it was approved, for, kidney failure patients, because they would frequently have anemia and, later was approved for the anemia of, cancer patients receiving chemotherapy.

And that was, that was given coming commonly to, to increase the blood count in, chemotherapy patients who had anemia. And they did that for many years until finally they discovered that the, the the, erythropoietin stimulates red cell production.

And guess what? Surprise. It also stimulates cancer growth in cancer production so that it was discredited. And, they just sort of, you know, that sort of became, something that I colleges don't want to talk about.

But, you know, they, they don't they don't do it anymore, but they, it's, you know, it's one of those things that sort of, the, you know, it's politically incorrect to talk about them, but, and then, you know, the drug that replaced, you know, there's a new drug which has been around for a while called, just, granulocyte colony stimulator CSF, which is like, the, which stimulates, white cell production and, that's very is still very commonly used, because in patients, in chemotherapy patients, because the chemotherapy will wipe out the white cells and they have, leukemia, which is, you know, it's dangerous because they can die of sepsis when the white kind of slow like that.

So they they will give them this. Glucagon is the brand name one of these drugs, which is a g CSF, you know, stimulates, white cell production. So the big question is, well, what's happening to the cancer?

You know, if the patient has the leukemia, for example, guess what? Leukemias are white cells. And, so there are a number of studies done showing, you know, what, the the, The cancers are probably being stimulated by the, the new production.

And, they're just, willing to accept that, in order to avoid, you know, the septicemia and septic shock. And so, you know, that I think we're going to find, over the next few years that this, you know, going to be a real problem because it makes, you know, asking, well, what will stimulate cancer stem cells, trigger them to grow again?

You know, a drug like that, you know, a drug like, New pigeon can do it, so that, you know, that's one of the things, you know, that's one of the many problems with conventional, oncology.

You know, it's it's really money oriented, based on a business model. And, you and it's to the the whole system oncology system has got to be 40 to 60 years behind current current, cancer research.

So, you would think that writing a book like this would be difficult. And it was difficult in the sense of, you know, learning a new jargon. You know, the micro about the molecular biology of cancer is sort of learning a new language.

So that was the difficult part. But the, you know, the, the fun and enjoyable part is, you know, we have all of these new, new cancer treatments that actually do work.

And, so in the dedication of the book, I made this statement, this book is dedicated to the half a million patients who died last year of cancer and to the half a million patients next year who won't.

Yeah. And that's where we're at. And, using, you know, use using repurposed drugs and, understanding, you know, the various, path cancer, metabolic pathways.

It is now possible to cure cancer. So, so with, with your all the research, you're dead. I mean, because there's always this question, you know what? What is what causes cancer?

You have all these different there theories, you know, full blast it you have metabolic, you have, you know, parasitical, you have genetic. I mean, what in your I've after diving into this, I mean, what is your opinion?

Obviously we may not know fully. Yeah. Well it is the, you know, when it comes to, choosing combinations of drugs, you know, right at the top of the list, we have, the metabolic, disturbance in cancer, which was described by Warburg that, you know, early 1920s, you received the Nobel, Otto Warburg received the Nobel Prize for discovering that the middle of the cancer cells have a different metabolic, set up, the that's called aerobic glycolysis.

And, so, you know, we have much better molecular biology tools. Now, we, we know that the metabolic derangement involves a, a translocation of a HEXO kinase to the from the cytoplasm of the cell to the mitochondria, these little pores on the surface of the mitochondria, which are called, voltage dependent anion channels, you know, Vtec for short pdac.

So easier to pronounce if you do it. And they're, they're, the, the, the HEXO kinase enzymes, the first enzyme in the, metabolism of glucose. And, not the actual kinase two is not the normal habit of kinase.

It's an embryonic form. And so the normal exocrine is is replaced with an embryonic form of exocrine is called HQ2. And that is translocated to the vitek not supposed to be there.

And it's it then it then influences the mitochondria. And one of the things that it does, is it immortal? Immortal is the cell. The, normally cells that are damaged, or metabolic deranged in any way will will undergo programed cell death is called ptosis.

The three doctors who discovered the received the Nobel Prize in medicine and, it's that important. So when the when the, is it the hetero exocrine is to is relocated to the that turns off the ability of the cell to undergo apoptosis, which is controlled by the mitochondria.

So now we have immortal ization of the, of the cancer cell. And simply by a maneuver, you know, a drug or, some agent that, will detach the two kinase to from the VTA, that will restore, the ability of the cancer cell to undergo programed cell death.

So that's a valid, anti-cancer strategy. And then there are a number of repurposed drugs that can do that, which we

highlight in the book, because, you know, they're widely available at the corner drugstore, you know, drugs like, of Ebright.

It's a kind of cell phone of your brain is an old, lipid drug. And, it's recognizable as an old antifungal drug. Been around for decades, both of those two drugs.

And there are others. But we highlight those two because they're widely available. And they will separate the HEXO kinase two from the VDC. And then the, when we look at the metabolic pathways of, you know, the we're interested in eradicating the cancer stem cells.

So the the thing about cancer stem cells is that they have metabolic plasticity, which means that they can shift their their major, energy pathway from, the, the three, the three major pathways.

They will shift back and forth depending on what's available, depending on what nutrients are available, depending on what kinds of drugs are being treated with, so the, some stem cells will use the glycolytic pathway preferentially, which is the you know, the, the Warburg effect that was originally described by the Warburg and that's this massive utilization of glucose, and, by the relatively bypasses the mitochondria.

The second major pathway is the, oxidative phosphorylation through the mitochondria, which is much more efficient. So if in effect, the cancer stem cell is using mostly oxidative phosphorylation, and they give them a drug that blocks oxidative phosphorylation, the the that's stem cell will then switch to the glycolytic pathway, in the cytoplasm.

And then the third major pathway, which is is highly active in cancer stem cells is called autophagy. And autophagy is it's pretty much ignored by conventional oncology.

But the autophagy is the, was I discovered, the importance of autophagy when I was researching, an anti an anti-malaria drug called artemisinin, which, was actually discovered by the Chinese.

There was a Chinese doctor, by the name of, two. You you they have these weird names, and, she received a Nobel Prize in medicine for discovering this anti-malarial drug, which is which is now first line treatment.

It's given a trial initially in third world countries. It's first line treatment for for malaria. It turns out that artemisinin is also an excellent anti-cancer drug, as are many, many of the anti-parasitic drugs, will have a dual purpose.

And, will can be used as anti-cancer drugs, very effectively. So, when I was reading about the artemisinin, the a lot of the studies did a lot of these studies are done, in cancer cells in vitro.

And they also, inject this, you know, have animal models where they have cancer cells injected into mice. But in the in vitro, cancer cell cultures, when they add the artemisinin and they look at the cells with under the microscope, they see the lysosomes migrate from the periphery of the cancer cell towards the center near the nucleus.

And that's called protective autophagy. The autophagy is really a, a process involving the lysosomes. The entire lysosomal system, the lysosomes are these little bags of, of digestive enzymes and acid.

These are acidified the little bags that digest, old unwanted proteins and unwanted organelles. And they're also involved in, you know, other processes, for example, there's a process called macro pino psychosis, which is shared with single celled organisms like the amoeba.

You know, everybody remembers biology class. They showed you a, a movie, of an amoeba under the microscope. And it's kind of it sort of looks like the blob and it's, you know, slides around and engulfs its food.

You know, it sends out these pods, which engulfs its food, and then it brings it into the cell and digests it. But that's, that's autophagy using the lysosome system.

So our cells do the same thing. The cancer cells, when when the cancer stem cell is dormant, the lysosomes and this entire autophagy system, you know, the lessons lysosomes migrate essentially where they're hibernating, when they when the cancer cell becomes aggressive and wants to, meaning the, you know, metastatic potential, the lysosomes will then migrate, towards the cell membrane where they can release acid into the micro environment and digestive enzymes into the micro environment, actually digest the, the proteins and the, the, the, the, the, the, the, tissues surrounding the cancer and, to obtain nutrition that way, just like, you know, other organisms, other parasites, just like a parasite would do.

So, autophagy, is the autophagy pathways are used by the cancer cells. And to eradicate cancer stem cells, we have to use an autophagy inhibitor. And it turns out that we have a lot of other fish inhibitors, that just, in the corner drugstore.

A lot of these drugs are auto fish inhibitors. For example, some of the some of the antihistamines, loratadine, as it is, is a very good accumulates in the lysosomes and inhibits lysosomal function.

Repurposed Drugs, Autophagy, and Immune Support

We also have proton pump inhibitors, antacid and cycle members, all, those, turn off the acid production in the lysosomes, which is the, there's a, the ATP, ace pump, which is the little pump in the lysosomes molecular pump that makes the acid.

So the the proton pump inhibitors inhibit those little pumps. The lysosomes can't make acid there. They become dysfunctional. Some, some cancers, such as leukemias, have unusually large lysosomes.

And they're much more sensitive to, sensitive to autophagy inhibitors that, that, you know, caused the lysosomes to become dysfunctional. And that's the one of the main mechanisms for artemisinin is, the artemisinin.

Accumulates in the lysosomes of the cancer cell. The cancer cells have a very high iron content. That's one of the major differences between normal cells and cancer cells.

The cancer cells tend to accumulate iron in the lysosomes. The artemisinin has the this, peroxide bridge, which is, you know, you can imagine a, it's oxygen molecule.

It's sort of sticking off the molecule, ready to, ready to react, with, pretty much anything. And so the oxygen will react with the iron, in the lysosomes, and create reactive oxygen species, which then, causes the lysosomes to burst, releasing contents, you know, all that acid and goop into the cytoplasm and, then will then kill the cancer cells and and the artemisinin or or Tizen.

I mean, it has a, preference to then cancer cells, from my understanding. Yes. Yes, it's, you know, it's, extremely safe. You know, it's, the it will, you know, everything is a matter of dosage and, you know, and iron content, normal cells, don't have, you know, the same amount of iron content as, cancer cells.

So the, and that's the main, differential feature that protects normal cells. So, so that's you can reach a toxic dose, you know, if you give enough of our, to our, our destiny, and obviously so, but, you know, you keep the dose low enough so the cancer cells are affected and, you know, they, they treat patients, malaria patients with IBD or destiny.

And, it's, you know, consider a very safe drug, you know, some of the other autophagy inhibitors, do have, more, more pronounced, more worrisome, adverse side effects.

For example, there's a whole class of, autophagy inhibitor anti-malarial drugs, mefloquine, hydroxychloroquine and methyl and chloroquine, hydroxychloroquine.

They're all related originally, you know, from the quinine, molecule and from the central cinchona tree and, mefloquine. You know, the safety is the safest, is of the three is hydroxychloroquine.

The probably the the, you know, and the most effective and yet the one with the most adverse side effects is mefloquine. Mefloquine is, you know, accumulates in the lysosomes and, disrupts lysosomal function.

It's highly effective. And, it's a it's an excellent cancer stem cell. Agent has an excellent anti-cancer. The problem is, and it's also a very good anti-malaria drug.

The problem is, it causes, neuropsychiatric disturbances. And, you know, in animal studies, you can actually see, you know, degeneration of certain parts of the brain.

And, so the, you know, with lysosome, some inhibitors and many of these anti-cancer drugs, you have to be careful about adverse side effects, especially with the, you know, the, the Ox fast inhibitors, oxidative phosphorylation inhibitors tend to be mitochondria, mitochondrial agents that are toxic to the mitochondria. And, you know, maybe one, agent is fairly safe and like, metformin targets, complex, one of the, the, the electron transport chain, you know, there are millions of people taking metformin. They do okay with it.

But if you then you add another mitochondrial agent, you know, another, or a third or fourth, then you start to get more, more adverse side effects from mitochondrial toxicity.

For example, if you add a statin drug, which is also a mitochondrial toxin, to metformin or, another, you know, another oxidized inhibitor, niclosamide is a, is a mitochondrial uncoupling agent.

That's another anti-parasitic drug which is commonly used for, tapeworm niclosamide. That's a very good anti-cancer agent because, it's both an Oxford inhibitor and a, fish inhibitor.

And so, you know, you can add a, a glycol glycolysis inhibitor to niclosamide and you, you know, blocking all three major pathway sites for us, glycolysis and autophagy.

So the idea is to get synthetic synthetic lethality, which means, you're you're preventing the, metabolic plasticity or preventing the cell from, from the cancer cell from shifting to a different pathway.

And, and, thereby, defeating the, the, the drug cocktail. You have to, block all three metabolic pathways. Oxford's like, glycolysis. So here we talked about glycolysis.

We're talking Warburg effect and or autophagy. Those three so, you know, the the last the last third of the book is devoted to well, you know, once you do those three, there's other areas.

There are two other important, aspects of cancer that we really have to address. One is, cancer. The cancer cells will hijack the immune system. They make themselves invisible to the immune system.

And so there a lot of, you know, a lot of treatments, treatments are involved in, restoring the, the immune system, the the restore the immune surveillance.

And then the very last, as we talked about earlier, is the, the inflammatory pathways tend to be hijacked by cancer cells, and they use that to state, you know, make themselves more aggressive.

So, you know, the, the anti-inflammatory drugs such and we talked about celecoxib, Celebrex as well as one and other and say it's and the, you know, and those studies have been done and published showing that, you know, if you give Celebrex along with chemotherapy that downregulated the inflammatory, the inflammatory cytokines produced, but by the chemotherapy and the patient does better, so, you know, they're considerations like, you know, getting back to, you know, the immune system.

I was intrigued by a, a research project at, I think it was Wake Forest. Medical center. They, they had medical students injecting mice with cancer cells, and, they this went on for months and months, and all the mice died.

All the mice died. And then, out of the blue one day, medical student held up the mouse. The this this one mouse

didn't die after a cancer ejection, so they named it the the SRT or mouse.

Spontaneous regression, complete, response mouse. And, they, they were very excited about finding this mouse that could defeat cancer. And so they, they did further studies.

And so the big question was, well, what if we transferred the mouse immune system, the T cells, to another wild type mouse with that give would that confer protection?

And sure enough, there that was the beginning of immunotherapy. They were able to transfer the immune system of a, of a resistant mouse and to give, confer resistance to the second mouse.

So, so the doctor who, really developed out the most was at first was a pioneer in immunotherapy was William Coley, who was a surgeon in New York. Who was he was an orthopedic surgeon in those days in the early 1900s.

They didn't really have much that much to offer. You know, the big cancer of the bone was osteosarcoma. Still is. And, he must have done 100 amputations for us to a sarcoma.

And, and then, he had a young, young female patient. This is the story that's in the medical literature here, a young female patient with an osteosarcoma of the hand.

So he did an amputation, and a few months later, she died of metastatic disease. And he was so distraught that he reviewed the last 200 cases, that he operated on.

And, there was only one survivor. And that's he noticed in the chart that that survivor had a, after surgery, the patient had a strep infection. And so he he speculated that this the bacterial infection must have stimulated the immune system.

And that's what, that's what made the difference in that one surviving patient. So he came up with a concoction of, bacterial toxins. It's called. And it was dubbed Coley's toxins.

Named after him. And he would inject these toxins into the patient's. And, he had some remarkable remissions and published this. Is that not everybody? Of course.

But, he did have some remarkable remissions that he published in the medical literature of his of his day. And, of course, when, you know, chemotherapy, was invented, then, the FDA, basically stopped, Coley's toxins from, seeing the light of day after that.

And, but that was, you know, he was one of the pioneers. And now current day, we have, immunotherapy with, checkpoint inhibitors. So, you know, we talk about that quite a bit in the book.

The problem with the checkpoint inhibitors is, that they don't work on everybody. And, the most of a lot of patients will develop, and, it doesn't number one doesn't work.

And number two, if it does work, they develop resistance fairly quickly. So there is this mad rush to find, well, what can we do to make the checkpoint inhibitors work?

And so, the discovery was made that guess what? It's the microbiome. You know, the friendly bacteria in the gut that, that makes the immunotherapy work.

And how did they discover that? They noticed that, you know, this, the very, very common for chemotherapy patients to develop sepsis because of the low white count and the, leaky gut.

And so it's very common for them to receive antibiotics. So they look back and they saw that in the patients who received the antibiotics that wiped out the microbiome.

They did not do well with the checkpoint inhibitors. So they did further studies. And sure enough, there are certain, bacterial strains in the microbiome that make the, immunotherapy, the the checkpoint inhibitor immunotherapy work.

So the I think that was discovered around 2017, that was like the year of the microbiome. And, so, you know, another surprising discovery that was made in, I think, the 1980s, late 80s, early 90s was, and regarding immunotherapy, the, The, the, the, the there was some case reports of, gastric lymphoma cases, and, the, you know, they had endoscopy, they would endoscopy patients.

He had a big mass in the stomach with a huge ulcer in the center of it, and ulcerated mass. They do a biopsy. It was lymphoma. They could not write right away because the patient had a huge ulcer.

So they would treat the patient with an anti ulcer drug, you know, for a few weeks, you know, say six weeks and then operate in those days, you know the the anti ulcer drug was, was tagamet which is brand name for, for cimetidine which is actually not a histamine type drug, which is also an anti acid and you know, cimetidine, it was, has been replaced since then with by the proton pump inhibitors, the newer antacids.

But, it's cimetidine is still available over the counter. You can buy it over the counter in the drugstore. So they, noticed in these two cases where the patients were treated for six days with cimetidine, they go in to do the operation.

Six weeks later, and, the ulcer is gone and the the lymphoma is gone. They, they're biopsy and the lymphoma is totally gone. So they they realized that the cimetidine cured the, the the cancer, lymphoma, cancer in the stomach, gastric lymphoma.

And sure enough, you know, when you do the preclinical studies, cimetidine upregulate, the immune system and, the, so, you know, it can be used to break the, upregulate the immune system, for, cancer treatment and also for, antivirals and antiviral treatment.

For example, there's a whole series of, that was published in the medical literature on, her children who received, cardiac transplants for whatever reason.

And, in those days, of course, they still do give immunosuppressive drugs. So the patient does not reject the cardiac transplant. And, because they're on immunosuppressive drugs, they are set up for viruses, that the can go rampant and the, the, the common one is there's a strain of HPV, human papilloma, papillomavirus that is apparently very common when in these immuno suppressed children they develop these, warts.

They're sort of like long, large, protruding warts on the fingers. And, you know, they had some photographs in the, in the journal, and it's, you know, you can't miss it.

It's very obvious. And, and so, they one of the, one of the doctors had the bright idea of giving cimetidine to the children with the, viral warts and, virtually all of them cleared up within 6 to 12 weeks because of the upregulation of the immune system.

So that gives you an idea of what, you know, a over-the-counter repurposed drug can do to to boost the immune system. You know, use along with the, you know, these other, you know, repurposed drugs that you make, you know, using it as an entire package, an entire, program, you know, it has it it's, everything is additive, and and it does help, the, you know, the other, available, immune stimulator, which is widely available over the counter.

Immunotherapy, Case Studies, and Book Inspiration

And so vitamin find it at the vitamin store, it's called beta glucan. And the, you know, the it can be it's a, it's a long chain, sugar like molecule found in cereals, certain mushrooms, yeast, and it's, it's in food.

So, and, the these, have a molecular pattern. It's called, it, it it mimics, pathogenic patterns, pathogenic pattern

mimicry, something like that. And, and it and so it stimulates the immune system of the patient, and, you know, there's a, there's a, number of case reports of, for example, the one I remember from the book, is an elderly man.

I guess he was in his late 70s with breast metastatic prostate cancer to the bones. You know, they tried everything. Every conventional treatment you can imagine.

His family refractory. His PSA is sky high, and PSA is, like 1100. And, he declines all further treatment and takes a, you know, one of these beta glucan products, you know, the, the probably the, the, the most, the most popular one is called HCC, which is comes from Japan.

It's a Japanese product. And, you know, six, 6 to 8 weeks later, they recheck his PSA and it's come down dramatically. And his feeling much better is, you know, exercising and, you know, that's a dramatic history of, success with, you know, just up regulating the immune system with a beta glucan type product, which, you know, that's very impressive. So, you know, there are a lot of these very impressive case reports in the medical literature.

And, you know, so we put a lot of these into the book. The other one that was very impressive, I thought was, Steven Biggles who? He was he's a doctor at Cornell Medical Center.

Who who comes down with, metastatic pancreatic cancer and, he, you know, was a smart guy. He realized that, you know, prognosis with chemotherapy is not good.

So he researched the medical literature. He came up with his own, you know, device, his own program, which was, a, high dose vitamin D analog, cimetidine and, and, some other repurposed cancer drugs.

And, he's now cancer free, is now cancer free and is still alive. I looked it up. You. So this is, like ten years later, and, so, you know, that's a really remarkable story. And, so, yeah, because, I mean, that kind of prognosis, I mean, if you get three months, you're lucky sometimes.

Yeah, exactly. You know, the it's it's great. And then, you know, there are a couple of, cancer survivors that were the inspiration for my book. One is, Ben Williams, who wrote a book, Surviving Terminal Cancer.

This is his book was back, I think, in the 90s and 1990s. He he was a psychologist. In, at one of the universities in Arizona, maybe the University of Arizona.

They, or. I'm sorry, it was, near San Diego, one of those universities. And I remember it was close enough to the border with Mexico. He would research in the in the in the library of the, of the, University library, because that was before the internet.

You had to go directly into the into the medical, textbook, the medical journals in the library. And then after the research, you would drive to Mexico across the border because it was close and buy repurposed scrap drugs.

I remember one of them was was, a vitamin A catalog and Accutane, which is used, you know, for acne, which is so vitamin A, and, you know, he he came up with an entire cocktail of these repurposed drugs, which we talked about in the book. And, he is still alive today.

He had a a glioblastoma, an aggressive glioblastoma, which, of course, is is basically a virtually 100% fatal, as you know, and, he's still alive today and, you know, he, so his book was one of the inspirations for my book.

And then the other inspiration is Jane McClellan's book. Starving Cancer. And I met, Jane McClellan at the Annie Appleseed meeting one one year. That's not too far away in West Palm Beach.

So I was able to drive to it. And, I also met Ben Tip-Ins, then Tip-Ins, at the same meeting that they were both there. Then Tipton's of course, his story was, I mean, Joe Tippin.

I'm sorry. Joe Tip-Ins. Yeah, yeah. Thank you. He was a, he was like a rancher, and I think one of the one of the,

little he lived out in the country and one of the either Oklahoma or one of those states.

And, he was an interpreter, entrepreneur and very successful. And, he came down with, non-small cell lung cancer, metastatic, and I think he went to MD Anderson.

They put him on a checkpoint inhibitor drug, and told him it probably wouldn't work, and send him home, basically the to, you know, with the poor prognosis and, right about that time, he had a good friend who is a veterinarian who said, look, why don't you try this veterinary drug?

Veterinarian drug? It's a dog de Wormer called Van Benders. All. You know, it's a if it's similar in molecular structure to Mendoza, which is, FDA approved for human use.

And the both drugs are, microtubule inhibitors, which is an entire class of anti-cancer drugs. So he took different vendors all along with a few other things.

And, the next time he went back to and got a Pet scan at MD Anderson, everything was completely clean and the doctors were amazed. He was the only patient under that entire group, that receiving the checkpoint inhibitors that survived.

So, you know, he, tells his story on his website is, you know, large group of followers and, so often vendors, all is widely available at the you can go online or at the local pet store.

Anybody can buy it. You don't need a prescription. And, you know, for human use is probably you're probably better off using the member, Anderson, which is, similar.

And, so, you know, those are the inspirations for my book and, the, You know, if, you know, if if, you know, these types of, approaches are widely, adopted, I think we're going to see a huge turnaround in cancer in the country.

And, you know, it's integrated my colleges that I think are leading the way with that. And, we're, you know, we as I said earlier, you know, I don't treat my myself as in my own practice.

I, I do not treat cancer patients. You know, I don't see cancer patients on a regular basis. It's not my specialty. And so we have a list of, integrative ecologists.

Of course. You're just, you know, you're on the list, and, we get a lot of calls and, you know, emails from patients wanting information. And, so we will then send them the list of integrative ecologists, and hopefully they'll, they'll be getting, good care and good outcome.

Yeah. You know, the, the, the, the research that you've done, to put this together and such a, you know, a clear and concise matter, to me is phenomenal.

And I, I mean, for someone that does not specialize in this to, to put this together in such a detailed fashion in a way that's very digestible, I, I applaud you.

I mean, that that. Thank you mazing. Well, you know, I was thinking about this the other day, and, it's, you know, when you, when you're working, with cancer patients, you have a busy oncology practice.

You know, doctors really don't have time to to, you know, to do this type of, book, and, you know, for it really, maybe it does take somebody who, you know, I'm, I'm only working 20 hours a week.

I have, you know, extra time to to study the medical literature and all this stuff together. So maybe it does take somebody who's not, you know, actively working in the field to do this, and maybe that sort of, you know, big question is, Will, you know, how do all the oncologists you have, you know, mainstream oncologists, why haven't any of them done this in a written a book like this?

And the answer is, you know, there are a lot of reasons. But, you know, part of it is they're just working 24 seven and they don't have time to breathe or eat, let alone write a book.

So, you know that that's another factor. So, yeah, it is. I think it's, you know, it's a trailblazing breakthrough book. And, you know, once, once you started doing it, it sort of, you know, it takes a life.

It has a life of its own, as you might say. And, it's actually fun because, you know, you're, you're putting together all this information that, you know, is going to make a difference. And, you, you know, it's going to propel the entire field of integrative ecology forward at least 30 years into the future.

And, you know, if, if that, is a, a lofty goal, you know, if that's a worthwhile goal, then, you know, that's makes everything worthwhile. I agree, I agree.

Well, doctor Dash, thank you so much for for all the hard work you've done and for what you're contributing to this field. I yeah, that that is wonderful to.

You know, the other thought I had that I wanted to share with you, Michael, is, you know, the book, the book came out in 2021. So it's been four years now.

And there's new material. Left over for years. And so eventually, maybe in a year or two will come out with the second edition. And, one of the things that could really help with the input from, people who are in the field, and I consider you, you know, a colleague, and I would be I would be honored if you would, you know, help make some suggestions and, you know, you know, there there may be some mistakes in the book that, you know, because medical research can say maybe it was correct at the time, but the new research shows, you know, maybe this particular pathway is really targeted that well by this particular substance.

And, So, so yeah, that type of input would be very, very valuable. And, you know, if you you don't have to answer right away, but, you know, that's something that that's one of the thoughts that I want to share with you.

It'd be my pleasure. You be my pleasure. Thank you so much. Well, Good. Thank you so much. I. Yeah, this is wonderful. This is. I mean, just this information alone.

Well, I know that will help, you know, thousands and thousands of people and then, you know, be able to go on your website to, to learn more and then also get the book out to have that as a, as a reference to really help them through.

So thank you so much, doctor Dash. Thank you. Thank you so much, Michael. Yeah.