Jane McLellandu2019s Mission in Orkney

Well, I'm absolutely delighted to have with me Jane McLelland. Thank you so much for for spending some time with me today. Pleasure, Michael. Always for you.

I'll try and make time available. As you know, you caught me on the road, so. Well, I mean, that's. Yeah, the the good heart is is willing to serve wherever, wherever she's at.

And that's what you're doing. You. Yeah. Yeah, it's a bit of a mission, actually. I'm in Orkney and, in the Scottish Isles, so very north part of Britain.

And I brought with me a sort of a suitcase load of my books, to just take around the GP surgeries and, you know, they have very high Bracher levels here.

So I'm kind of on a mission to try and change things. And they have very bad access to, treatment. They have to travel to able to fly or ferry to Aberdeen.

And it's really hard for them to actually get consistent treatment. So I'm kind of partly here on that mission, partly here because there is the inter it's an international competition for islands going on in Orkney.

Right now I'm in Guernsey where I'm from. My little island, is competing doing quite well, here with my sister and her husband as well. So we're just having a bit of a, a fun time.

And I'm also on my mission on the side, so, a lot of it. I'm glad you get to do some fun, because you you are. I mean, when I see everything you're doing, you're on the go.

I mean, your Facebook groups, your trainings, your conferences. I mean, you you seem to be 100 miles an hour or so. I'm glad you get to enjoy it a little bit in between.

Yeah, I do, and I was on a yoga retreat and I've been quite recently, and that was marvelous. Marvelous. And I might try and get a retreat together for people next year.

Make it an educational retreat with intravenous vitamin C, maybe, I don't know, just an integrative, integrative approach which will just be beneficial for for cancer patients.

Yeah, I love that I love that.

Cancer Diagnosis and the First Drug Cocktail

Well, so for for people, the majority of people, they know who you are. But there may be somebody out there that that does not really know your story.

Do you mind just can I sharing quickly. Yeah. Yeah. Give you a quick whistle stop tour of my journey. So in 1994, I was diagnosed with cervical cancer.

Very aggressive. And, it was quite a large tumor. They gave me hysterectomy, they gave me chemo. Radiotherapy. Obviously lost my fertility at that point.

Very depressed. But then I thought I was okay. And then eventually it came back in 1999, in my lungs. So, you know, they said that's it. You know, terminal.

I'm on that slow road, down. And, I was maintaining myself with intravenous vitamin C and diet at. And I was kind of coping okay with that. And then I was hit with my low dysplasia, which was a result of the chemo and the right therapy I had because I had two lots of chemo.

I had more chemo when I had the lung tumor. So this then stimulated bone marrow issues. So I ended up with, a very aggressive pre AML condition. So that's the leukemia condition, as a result of the chemo and radiotherapy.

And so this road down that I was on suddenly became very steep. And I was I was on a fast descent. So, that was what prompted me to look for other things, to look for treatments that maybe nobody done before.

I just knew that if I had more chemo, that wasn't going to help me at that point. So I started looking into old drugs or anything. I didn't care what it was, you know?

But I came across, some old drugs that seemed to have some potential and I didn't know quite how synergistic they would be. I mean, I've, I've sort of reverse engineered how it worked a little bit because the, you know, the all the research has come out since.

But, I did things to block like the three macros in the diet, which is carb, protein and fat. And I was actually hitting all three sites. And I think this is a mistake that people make, is they think that it's just sugar or glucose that feeds cancer, and they don't realize there are other pathways that are involved as well.

And glutamine is a protein pathway. And then you've also got fat pathways. And I think fat pathways are another, area where people don't really associate problem.

We can maybe talk about that with things I've learned recently. But the so that's, that's one thing. So I put together a cocktail, I use berberine, which is a natural form of metformin, and I actually use metformin later.

I use departmental, which is an antiplatelet drug. So it stops your platelets sticking together. But it's it blocks the protein uptake, the DNA, recycling.

And then I use a statin, I use lovastatin which you can't get so well anymore. But it's a lipo a fat loving, statin. And I think it's important to mention not every action works for cancer.

And I also use a non-steroidal anti-inflammatory. So, like aspirin. But it was actually a toddler, which is used for arthritis. I did use a bit of aspirin as well.

So I kind of put that combination together and, stuck with it for about seven months and got really good results in terms of my blood markers. Came off the cocktail thinking I was okay.

And then of course, it came roaring back the next year, in 2004. So I then went back home. My cocktail added a few more extras as well, and that put me in full remission. And, touch wood.

Touching wood. I'm still going strong 25, 26 years later. You know, I'm, I'm doing okay for my stage four, so. So that's where I am. So I, you know, part of what I did was educated guesses.

I would say, I look for synergies between the drugs and between the treatments. It was all about knowing that one thing was not going to be enough. That was my thought, was that whatever we have and the treatments we have still have in oncology, a very basic they just give you chemo or radiotherapy and they're not really mixing it.

The starting to use more combinations, but really not enough. The tending to use more genetic treatments. Alongside the chemo whatever.

Metabolic Pathways and the Metro Map

Which are great but then not enough, you know, the metabolism which is it affects every single cancer patient in a

fairly standard way. There is sort of different fingerprints that you have for different cancers.

So pancreatic cancer, for example, will feed in a specific pattern, which is different to, say, leukemia and lymphoma or ovarian cancer. They all have specific fingerprints that they like to, to use to feed themselves.

And I was lucky I put together a cocktail that shut down my cancers. So yeah, here I am. And I, you know, I've spent ages, I thought, well, I've done it, but, you know, yeah, I had this concept in my head of how I'd done it and, and, and that was a basic, you know, starving it because all cancers are hungry.

And the more aggressive they are, the hungrier they are. So the more they need to actually pull in the fuels from different sources. And I kind of knew if you blocked one source of fuel, it'll use another.

And that's the whole concept of my what people may know me for, which is my metro map. This triangular map that's kind of my the biggest thing that people likes me for, is my metro maps.

So that's that was really that's the crux of my, of my book. And the crux of of how my book has done really quite well. Can I blame can I play my trumpet quickly?

It's just. Yes, it's medical book of all time, which is amazing. I'm sorry. It's just been voted by readers on Amazon as the best medical book of all time.

Are you serious? Let's see. I mean, it's simple, but that's that's not that's not a literary prize, let's put it that way. It's just it was based on, reviews and people who.

Yeah, basically the, the likes and the, you know, the basically the five star reviews. It's incredible. We got them. And did they, actually hold your book for some reason as well?

I mean, there were some, some challenges. Yeah, yeah. I don't know why they pulled it in the UK, but they, they pulled both my books down on Amazon in the UK.

I just know that there's a lot of hostility out there. They kind of see social media and you could see me as possibly a social media influencer, but they tend to lump us all together as these wacky theorists conspire theorists, whatever they do, I don't know.

But they they lump me with and there are some weirdos out there who are kind of pushing things that they shouldn't be pushing. But, you know, this is they they, didn't read my book, took my book at face value, thinking it's just diet on keto.

Keto is like one of the things they really hate. They really hate keto. And they really hate diet because it's cheap meat. Any of the metabolic therapies which are cheap, the off drugs, you know, they're cheap.

Big pharma certainly don't want to have anything to do with cheap off drugs. You know, this is this is a problem. And I've received a lot of hostility, from certainly people in the medical profession, they have since taken down that nasty Instagram Reels and things, but this has left a trail of destruction behind.

It's not like this has died. There are a lot of haters out there still. It's left this, Yeah, this nasty aftertaste of people who still think I'm, some wacky conspiracy theorist and they try and, you know, anyway, it doesn't doesn't matter.

I just keep doing my thing going on, and, I'll get there in the end. Well, I mean, and just proof, I mean, here you are, kind of the the top medical book and, that, I mean, that's phenomenal.

So even with all those haters, you see the need and the public. Yeah. For their hunger for this type of information. It's the information you're bringing forth is it's not like you are, you know, channeling it.

You're actually basement. You're going into medical research and looking at pathways and then utilizing tools to

address those pathways. Exactly. And in fact, I sent my book to some researchers at Birmingham University, and I've since had a chat with them, and they mentioned, did I want to come and do a Ph. D.?

That's like, well, that's okay. So, you know, I'm, I'm going to think about that. That would be the most amazing end to my story, if you like, with all the haters out there and the hostility to actually finish off my the end of the story with, I don't know whether I'll do it.

I don't know whether I can do it part time, how long it will take. These are all discussions I'm going to have in September after the holidays. So, yeah, let's see where that goes.

But that's really exciting. And I'm really, seriously, there was so like, oh my goodness. Which just looking at these pathways, you know, the SCD one is what they're looking at.

They've been blocking that with leukemia because they looking into blood cancers. And this is a particular type of pathway that uses mono unsaturated fatty acids.

It's all linked out, processed, to be honest. Which we're starting to get. Besides, see, now that, mono unsaturated fatty acids are found in things like avocado oil and olive oil, and they should probably actually be pulsed with your other, polyunsaturated fatty acids, particularly if you use a kind of, combination that they're looking at, which is, a combination of three drugs, valproic acid, which is normally used for seizures, mid oxy progesterone, which is a contraception.

And what was the other one? They were looking at a fibrates, which is again a cholesterol lowering drug. And those three things kind of lowered like they had an enormous effect on leukemia.

But you'd probably have to do the diet as well, lower those, mono unsaturated fatty acids. And you can look up which, which foods have the most of the monounsaturated fatty acids, but cut them out completely all the time.

Might be a bit much. Zoosk. Some might be pulse it then. That's, you know, but maybe I can go and have a look at that. And I think they probably quite interest in the fact that I'm kind of the bridge between the researchers who I'm sure are desperate to see some of their research make it into clinical practice and the blocks that happened on the way, but never to get there, to never to actually get to the to the patient.

So I'm kind of like the leap from research straight to the, to the patient. And I think, I think it's important and I think going forward, they probably want to embrace me for those reasons as well.

Because trying to reach the patients and trying to make improvements and to use these, old drugs, these repurposed drugs is, it's hard. It's hard to get the backing for it.

There's no money involved in the research normally. So, Yeah, that's that's where I'm going with it. And I don't know, I'll tell you in September what I think about where I'm going or what I'm doing.

Maybe they'll meet me and change their mind. I don't know, but, yeah, yeah, it's exciting and exciting to get that kind of. I mean, I have got I've got researchers constantly, coming to me, and saying that I've kind of simplified a very complex and it's if you look at cancer metabolism on the paper, it's like a spider's web of different pathways intertwining and mixing and just just splitting it down, trying to make it three sides of a triangle.

Repurposed Drugs, AI, and Personalized Treatment

It is very simplistic, overly simplistic in some ways, but I think it's it's helpful. It just helps to clarify the mind. I think a little bit I mean, it it's powerful.

I think for, you know, for a layperson, it is good to be able to have a very kind of easy visual. So they know, you know, the pathways that, that are there, you know, what kind of tools that will hit these different pathways.

I know when you do kind of the the bigger metro map, then it it can get quite, you know, complex. But, if you know, then your, your cancer the type of cancer and you also know some of the genetic, mutations that, that.

Yeah, that the cancer is exhibiting then and then match the different repurposed drugs and natural agents, you know, to try to then block these pathways.

Yeah. Yeah. So that there are different tools coming out which will help that as well. So you can look at the gene expressions and not just the gene mutations.

So the expressions will be more about how it's actually behaving rather than just the switch in the genes. I mean you can use genetic drugs and they will work.

But unfortunately you get more mutations happening. So of course the cancer is going to mutate and do something different. Whereas the metabolism, you can kind of predict where it's going to go next.

So you block say for example ox, once you know that it's going to use more glycolysis or it's going to use more M2. So there are specific pathways that we know work together.

So it's vice versa with the ox box and the glycolysis. So this is cancer fermenting or using mitochondria with oxygen. So it's both both get used in cancer.

People just think it's just the fermentation. This glycolysis which spews out last night. Right. But it's not just, it's that's not just the only pathway.

You know, ox falls can be the key pathway in many cancers, for example, prostate cancer. But of course, if you have genetic mutations in there, say, for example, you've got a P ten loss, then you've got more glycolysis happening.

But of course it swaps depending on the stage as well. So when you get to stage four with prostate cancer, when it becomes castration resistant, that's when it uses more of the glycolysis as well.

So it's a switch around. But you can do that. And you can use I, I mean this is the beauty of the the recent innovations in the last couple of years since I talked to you last is just the explosion of AI and how people can now use that, and I use that all the time now just to have a look.

It doesn't always get it right. I've had to correct it on multiple occasions to start with, but it's getting better and it's learning. And I think you can, you know, you can look up your metabolic phenotype, you know, with your particular cancer behaving whatever.

It's specific dietary requirements, if you like. Does it like these pathways or that pathway. And that's you can kind of work out and then look on the map to see which things might be more appropriate for you.

But I probably need to do more books. I need to write more books, to be honest, Michael, I'm just, you know, I've been I'm sort of updating the second edition for third edition.

So I'm but I'm, I'm thinking, I don't know, I might take fructose out into a separate book on that. Now, I don't know, because people like, you know, fat ptosis requires, a doctor really, to help with that.

You know, it's not something that can be done as a DIY treatment. And I think that's another worry that I have at the moment, is that there are Facebook groups popping up with DIY friends, and it's all in ivermectin, you know, and this is this is worrying to me because patients can't manage themselves in this way.

It's too important you this dicing with your life at this point. Why are you doing that? You know, I just think, you always need to have a doctor involved with this, and, you know, people like yourself are just brilliant for helping to decide exactly what you need when you need it.

And to to try and do it on your own, you know, if funding is an issue. And I appreciate that. But it's, I think for the sake of, you know, do a go fund me, do anything.

Just try and raise enough money to actually get your treatments overseen by doctor always. You can, you know, that may not be practical in some instances, but, it worries me that this is a growing trend, that people feel that Ivermectin and Fenton does all the going to be the answer.

And I know this is down to Mel Gibson on the Joe Rogan podcast talking about how we have these trends who are cured using that combination. Bit of an exaggeration when you dig into it a little bit.

But, you know, people have got this concept that we need these two things, and I have seen some results with it, but very, you know, no, not on their own.

These things don't work on their own. You need a much bigger combination. And ivermectin, for example, doesn't have the same amount of science as metformin and statins, and even the dissolves.

So, you know, these are these are things that, people should add in. And ivermectin can be incredibly useful for, improving the immune response. It actually works on the Oxford pathway a little bit as well.

So that that's why it works. And then benzoyl actually works a little bit on the glycolysis pathway. So you've got blocking of those two key pathways which is important.

But that's not really ivermectin is weak compared to you know some of these other treatments. It's not it's not the it's not the answer for for most people it just isn't you know.

So I think people need to approach it with a little bit more caution. And unfortunately there are doctors out there talking about using high doses of just those two things and getting great results.

And again, that worries me. And they're not looking at enough pathways. I'm much more of a nuanced approach and let's not do anything extreme. I hate the extremes.

I think when people start their journey, they go extreme with the diet, and then they go extreme with, everything really. And I think keto and we'll come to the keto bit now, I think in that, keto is something that can be very helpful initially.

I think, for particularly if your metabolic mess, you know, if you're glucose is, you know, if you're, insulin resistance and, you know, just a bit of the metabolic mess, but, really, it can start to cause too many issues down the line.

And I've seen it in the people. It's a bit like using chemo or some of these other big treatments that you get good result for a certain period of time, and then people fall off the cliff.

There was resistance that comes in. This is one of the problems that I've seen, and I didn't know why. I didn't know which pathways it was switching on.

Okay, I knew that keto would switch on a pathway. And of course, you know, one of the pathways it switches on is of course, fat. So the free fatty acids that are released into the bloodstream will kick off a pathway.

And I discovered this recently, called e I f 40. Right. I'd like it to be a little bit easier to, but it's not. You could probably say it looks like elf or a.

But anyway, so this is a pathway. And unfortunately, this triggers resistance. All right. But it triggers resistance down two other pathways. One is the pathway and the other one is the, to the pathway I'm to.

And both of these can of course be affected by metformin instruction. So again you know and there's a lot of hate about oh you shouldn't take metformin because it's going to affect the mitochondria.

And then you know, it's a very weak effect on the mitochondria. And it's beneficial. It's a longevity drug. So that's, you know, I get a lot of hate for the metformin.

I get hate for the statins. Oh, I'm a sort of big pharma shill. No, that's and that's a cycling. I'm sure it's not a big favorite. Yes. No, I know exactly because that's going to mess up your microbiome.

But the whole point is to use these things effectively in the right way and personalize them as much as you can. So there are some, you know, I wouldn't necessarily say toxic cycling is great for colorectal cancer.

It can work, but you're going to have to be very careful about it. Pulse it carefully. And then make sure you flood you yourself with, things that are going to help rebuild the, the microbiome.

And metformin is great for that. It rebuilds this, a commencer, which is a really important, probiotic in your gut. So it's kind of like a, it's a protection inside your, your gut and helps, helps you with all sorts of, immunotherapies and general treatments.

Keeps your immune system going. So there are lots of reasons why the off label drugs are great, lots of reasons why we should be looking at better synergies.

And people just get fixated. That's my big concern. They get totally fixated, go down a road of thinking this is going to be the answer, and they're not looking at a big enough picture.

Yeah. Combinations. And I think that's a challenge is that you you have a lot of promoters of very simplistic therapies. And, and people always want to have, you know, I'm going to take this and that will fix everything.

Not recognizing that that cancer is a it's a complex disease. And it is a, it's an organism essentially within yourself that have figured out that have many, many pathways.

And yes, if you blocked one, then it tries to figure out other pathways. Exactly five. Yeah, it's a shapeshifter and that's what people don't get. So you can treat it on day one.

By day 30 it might be completely different and it might be behaving in a completely different way. You may need to switch your approach. And that's that's what we need to be testing.

It'd be great if we could have these sort of gene expression tests a bit more regularly for everybody, so we could actually see what the cancer is actually doing, which direction it's moving, what it's likely to be doing.

And you can tell a little bit with pet scans with the sugar uptake and things like that, but it doesn't really show up the glutamine.

Glutamine, Testing, and Metabolic Fingerprints

You can get glutamine traces for Pet scans. But I have known people on the scanner sound really worrying for people that you can have a Pet scan done.

Which that the cancer is switched entirely to glutamine. So it looks like the cancer is metabolically quiet. Right, that it's not feeding, but it's actually become very quiet and not doing very much, when in fact it is, but it's just using glutamine and probably fat as well.

So you then have this issue. And in fact for prostate cancer, they look at the uptake of protein is what they use sometimes for the Pet scans in order to look at the the metabolism.

So it's yes, you can kind of rely on the Pet scan up to a point, but if you've really been working hard at blocking the glycolysis, the glucose uptake, the, you know, all of that side, you do have to note that you have to block some of those, glutamine pathways as well.

Green tea's kind of still on my list of things that people should take for blocking, glutamine. But then things like sodium phenol butyrate, which the things like triple negative breast cancer, I've seen amazing effects because that helps to reduce the glutamine in the system.

But yeah. What else? Yeah. So so the question then is, I mean, so there is certainly just to kind of reiterate, there is, say, glutamine Pet scan that people can do outside of the normal Pet scan.

Right. You'd have to find people to do it. They do it sometimes for brain cancers because brains take up a lot of glucose anyway. So your healthy cells, as well as your cancer cells will be very avid glucose consumers.

But if you want to look at, a more contrasting Pet scan, glutamine can often show it much better, because you can see where the activity is. So yeah. Yeah.

And the patient would then just ask their oncologist, can I also do a, a glutamine Pet scan because I'm concerned. Yeah. Yeah, there's there's obviously the issue with increased radioactive exposure, to do more than, but maybe you could space it out, maybe do one month the glucose and then maybe in three months time you look at another one with glutamine.

But I wouldn't do them back to back necessarily. I don't know whether you could do. Maybe you could do the Pet scan with both at the same. I don't know, I don't know.

These are these, investigational things that I, you know, I'm not they're doing that, so I, I'm not in the diagnostics, so I don't know I don't know the, you know, the nuts and bolts of how this could practically be used.

But certainly you can look up that there is some new diagnostics coming out to look at, some of the other metabolites that are being used as well. So you can do tests to see whether I've forgotten which ones, sarin or whichever.

But they're looking at particular amino acids and things. But yeah, that there were new tests coming out and found that. And you. So with glutamine, I mean, one of the challenges is that glutamine is obviously the most abundant amino acid in our body, and we don't survive without glutamine.

So how how do, how is a good strategy, you know, for interfering or blocking glutamine going into the cancer? Well, blocking glutamine into the cancer itself.

So they have so cancer cells have specific glutamine receptors that, going to take it, take it up more readily than other cancer than healthy cells. And this is where green tea or EGCg, the metabolite of green tea, will actually be really effective because that actually blocks the uptake into the cancer cell.

So that's where it's really useful, really useful. And I know a lot of people get very ate up with snips. You know, these, they have their snips, a single nucleus, fully polymorphisms that these these are like, little genetic malformations in your body.

That kind of a personal to you. And if you have this particular snip, you know, the Comt snips, people get worried about taking green tea, that it might affect your estrogen and things like that.

But I think this is too much of a worry or, you know, and I've seen people come up green tea come off the, you know, and then they do very poorly. So.

It's, it's a matter of looking at the overall picture. I'm going to, podcast with, with somebody who does this nutritional genome testing and she says that people get to overall with all of that and they take it too far.

So yeah. But green tea. Right. So doing fino butyrate great. Tamoxifen also blocks, glutamine. People don't know that. And that's one of the reasons it works.

People are obsessed again like the estrogen. But actually tamoxifen does work on glutamine as well. And again, there are some snips that mean that you maybe don't metabolize the tamoxifen particularly well.

The CYP2DI think it is. And that's a liver enzyme, which means that you may not be producing enough of this metabolite with your tamoxifen. So I think, you know, snips should be tested.

But you need to take the overall picture of what's going on, really. And what you need to do. And, tamoxifen can be a problem and it can lead to and Demetrius problems later on.

So, yeah, it has to be used with caution. And you have to be blocking other pathways. And it would be helpful to know if you've got these snips, these mutations.

And, you know, again, it's thanks to expense and, what you do and I, you know, I think I think a lot of people just go into the sort of, I go to spend all this money with all these nutritional genomes of snips, the gene expressions, all of these things.

And I think it can lead them in, I mean, funding is a huge issue for people. But I think just get the basics in first stop the damn thing by, you know, blocking its feeding routes and then layer these things in as we go, and, and work on it as you go.

And you can see if it's still, you know, you can see if you're having an effect fairly quickly. So, yeah, I think people have got to, take a pragmatic approach to how they approach their treatment, how they work with their doctor, how they personalize their approach.

It's a complex beast. That's the problem with cancer. It's not just straightforward, you know? And I think we're doing well and I think we're making big progress towards better, cures.

And I think we'll get, you know, for the next few years, we will see a big change, I think, with people using. So the starting to use bigger cocktail of off label drugs now.

And I've seen amazing results with, with people who have got the guts to go for more than just for the traditional care and quality cocktail was always for.

But, and I've always been pushing for a few more than that, and I now know that people are doctors are using a few more than that, and I can see that it is extremely beneficial.

So, I think we're moving into a new realm, which is terrific. You've got new testing, coming out. We've got astronaut health of, emerged as well. So they look at repurposed drugs and what gene expressions etc.

could be, you know, related to which, which drugs. So we've got, we've got lots of different tests coming out and lots of different ways to make treatments more effective.

So I'm hopeful that a stage four diagnosis today is not the same as the stage four diagnosis. Even two years ago. I think we've made quite significant steps forward since just two years ago.

I really feel that it's making big strides now, so fingers crossed. But it needs this integrative approach. We need to have a bigger range of, people working together as a team with each cancer patient.

And of course, and next thing, of course, will have to be preventing cancer because it's just getting to epidemic. You're going to be I mean, you probably aren't completely overrun with patients.

How are we going to cope unless we get formal, integrative doctors, they lower the, access point for for people to go and get treatment. I mean, I don't know how.

I don't know how. It's. I think we're it it's not the go to, implode the whole system. I, I really don't know what's going

to happen. But, you know, and and what I can see is that the wealthy can do well, and people who don't have the money don't do so well.

And that's not equitable. So we need to do something in order to marry that gap between the two. You know, your, health should not be based on your wealth.

And unfortunately, that's the way it's going at the moment. So I don't know what the challenge, I mean, like you're mentioning is that, there's this huge gap between researchers and then the, the practical implication and, and I and I think and when we dealing with the repurpose drugs, like you're saying, there's no money in researching these, repurposed drugs, you know, because they're out of patent.

So the big hospit ALS, you know, they are funded by big pharmaceutical companies, you know, so their studies are essentially going to be what makes the pharmaceutical companies money.

Ferroptosis, Natural Agents, and Targeted Therapies

So they're not going to be focusing so much on repurposed drugs. So now we have, you know, we need to look down upon the educated, you know, physicians out there and also educate the populace so that they can ask the appropriate questions, you know, to their physician as to, you know, what, you know, what can we do that is in correlation with the research that exist, that is inexpensive and beneficial.

And that's why I feel that your work is so important. Because you are being that in between person. Between the research and then the laypeople and also doctors like myself, that can then benefit from the work that you're doing.

Yeah. Thank you. Yeah. So it's. Yeah, it's quite a bit of pressure on me to try and keep on top of everything. Do you know what I mean? I think everybody expects me to know the latest.

And I do try and keep up to date with everything. So I normally start my day with several research papers. It's my, like, reading in the morning. But it is, it is hard to, to keep on top of everything and to see but to see the trends of when things are going as well.

It's trying to have a big overview. You know, I have a lot of Google searches going on different things. So, I yeah, I like to have a handle and I, you know, I can't see everything.

I miss things and I, you know, I try my best. But it's, I think we're getting to a point where I hopefully is taking over from me. Well, so, people can do their own research and not rely so much on me, and that's terrific.

I can guide people in the right way and say, these are the things you need to ask. I ask in this way and then, you know, prompt it with things, ask it with that, and then you can come out with the right kind of answers.

So and I think people are getting better at that. So, you know, that's, that's a, that's a relief for me. Well, and yeah. And I and yes, I don't, I don't want the pressure to be all on you, but I, I'm, I'm what, what I'm saying what I'm saying is that this trend.

Yeah, it's it's a really important trend. And, and I think that more and more people with the work that you're doing are recognizing the importance of looking at it, you know, and, and this way and yes, AI is is, I mean, phenomenal next step tool because it becomes quite complex sometimes to consider all the different pathways. The.

Yeah, genetic mutations, the gene expressions, so all of that can become complex. But then to be able to rely on a, a way, system that can analyze a lot of data and compile it in a, you know, summarize, it becomes really, really, you know, that is phenomenal tool now.

Yes, exactly. And people are even typing in what would Jane McLellan suggest or say ovarian cancer with a particular mutation or whatever. So but it may come out with an answer that is not exactly what I would say comes out sometimes with drugs like, Celine or things like that, which may be quite toxic to the liver.

And I wouldn't, you know, there are other things that I would use for blocking that pathway, which are natural. And you know that, it's not just all about the drugs.

So I'm very much into natural alternatives as well, where you possibly can. And I just worry that it may lead people down the wrong path, but at least it's a starting point.

That's the point, is that people and this is maybe we need to go and write another book and make it more specific towards potential treatments for specific cancers, with maybe the key key mutations that tend to happen and what you would do if you have those mutations, how might that affect your treatment and what would you do if you have that?

So I don't know. I need to see that, but actually I probably need to. Well, I've got to do how to prevent cancer as well. But that's another thing because people just are still obsessed with the fact that they think cancer is all about genes and it isn't, you know, and cancer doesn't start with a gene mutation.

Cancer starts in the cell membrane. The membrane. All right. It's the tumor microenvironment that affects the cell membrane. And then that becomes a little bit inflamed.

You get other factors going on there multitude of different things that happen together. Maybe pathogens. And in fact you always have some sort of pathogenic influence.

And then you get these pathways vital. And that's what then changes the metabolism. And then you get then you get the change from the cancer stem cell to suddenly becoming a proper cancer cell that's fast dividing.

So this is what people need to understand. And that's where I need to go for my prevention plan is just talk about the tumor microenvironment and how that affects the the cell and talk about that.

It's not just about cutting down on alcohol, cutting out your carbs, exercising more, getting more sleep, getting less stress in your life. You know, those are all important and they affect the metabolism of you.

So yeah, it's it's a big picture. It's a big picture. And, but I want to get into the nitty gritty of, of really what we can do to try and prevent cancer a bit better.

So that's that that would be my mission in the future, is to work on that, a little bit more. Yeah. What else can I tell you? Yeah. So and that's the thing, tumor microenvironment, and it's, signaling to the behavior of, of the tumor in itself.

I mean, it's so important, but I would love to in your second book, you know, you brought in and we talked a little bit about it, you know, in regards to therapy, ptosis, do you mind just kind of giving a kind of a because it seems as we're reading, you know, reading your book, you know, about purpose is seems like a very complex, process.

But I, I don't feel it is. Okay. Well, the there are really key issues to look at here. The thing with therapy, ptosis is that there are two real pathways that it can use.

You can either use. So it has a way of sort of stopping therapy choices happening with an enzyme called GG four. So you need to be blocking that with some cancers.

Block in the DPG school is kind of the big thing. And with other cancers it's actually blocking, this act which pulls insisting cysteine is an amino acid, which creates glutathione.

Glutathione is among strong antioxidant okay. And that protects the cell from file choices. So blocking that is another way of pausing for ptosis in some cancers.

But the different cancers actually have an it is quite complex situation. And different cancers respond to fructose in a different way. They all have.

They all seem to have a different fingerprint as what's going to cause up ptosis, more or less. And there are some cancers like breast cancer, prostate cancer.

They don't respond so well is quite you can cause ptosis in them, but they're harder to trigger the process. So there are other cancers like k res colorectal cancer or even pancreatic cancer.

Ovarian cancers are easier sarcomas. They are easier to trigger the up ptosis. So yeah, again, the metabolic fingerprint of what that cancer is doing and how they want to behave.

Yeah. Well, you know, and again, I need to probably write a, as a book on its own, throughout this with the different, different ways that these, these different cancers can actually be triggered.

So, yeah, it's not just a straightforward this is it. But then again, it's a bit like my metro map. If you kind of pull in a few things, then you can block more of those pathways.

We can cover your bases. So if you know that if you if you're not sure whether the cancer is going to be more full or the exact anti porter, those two to ways to trigger the the ptosis, then you can you can cut them both and then you know got these other things that go on to try and prevent that happening as well.

So you need to a a detox pathway called Nrf2. So yeah. And then that was I was worried about sulforaphane because that is an Nrf2 activator. I thought that would actually stop for ptosis.

In fact, it creates sulforaphane is brilliant because it creates ptosis in cancer cells, but it prevents and it protects all healthy cells from fructose because you don't want fructose just happening in your healthy cells, it'll create inflammation.

So cell phones are fine even. So this is why, you know, I need to update the book the whole time because the new research comes in. Okay. And then you have to switch and go, okay, well, this was this is my worry, the reasons why I was worried about it.

But I've now proven that actually it's okay. And then we can move forward and we can use some of these things to, to help improve the effects of, the treatment.

So, yeah. Constant. Yeah. Even mention and the the beauty, I mean, what, what you're highlighting, which is really important I think, to understand as well, is that, you know, natural agents are different.

Interventions have different effect on cancer cells versus healthy cells. So, yeah, it's not a clear cut that because it does this in the body that it does the same on the cancer cells.

Exactly, exactly. So people get worried about that. And I was you know, initially I wasn't sure either. And curcumin high dose again would trigger for ptosis and will protect things like your, your liver and, your spleen from, because you don't want fat ptosis happening in some of your cells, which are healthy.

So and particular detox pathways, etc. in the brain as well. So you don't want, process happening in the brain, in your healthy cells. And this is where ketones, exogenous ketones are taking ketones, taking beta hydroxy butyrate, can be really beneficial to help protect your brain if you're doing a pulse, a therapy, ptosis.

And I don't think people should be doing a continual ptosis treatment because it's a bit like doing chemo or radiotherapy. And that's that. That's how I define a sort of a kill phase being pretty radical.

Push. It's like creating these oxygen free radicals, which can be very damaging for your body in excess. So you don't want to be doing too much. So you pulse it.

And I think we should be doing the same for our process, doing a pulse, but with, with, high dose intravenous vitamin C, you get this pulse of free radicals, these oxygen free radicals which are targeted because the vitamin C

reacts with the iron in the cancer cell.

And there's far more iron in a cancer cell than there is in a healthy cell. So you get this targeted approach where you get the release of hydrogen peroxide and these free radicals, oxygen free radicals in the area of the cancer.

So it's a very targeted approach.

IV Vitamin C, Synergy, and Closing Thoughts

Intravenous vitamin C is perfect in combination with other things to create a kind of a therapy ptosis protocol. And that's why I like it. It's not a sort of a blast like chemo, which really does affect to healthy cells as well as your cancer cells.

That's what we want. Personalized and targeted treatments. Yeah. It's kind of like a the Trojan horse type of scenario where, you know, you you, you have all that iron that's in the cancer and then you can utilize that, you know, like the vitamin C or, or artisan eight or, different things that just triggers that oxidation of of iron. Yes.

Yes, exactly. And so you can make it far more targeted that way. And that's the beauty of intravenous vitamin C with artemisinin. And these treatments can actually work synergistically together.

Yeah. So, you know, I'm hoping with more cells coming out with intravenous vitamin C, particularly with pancreatic cancer, that had just been done at University of Iowa.

And they've seen that double survival with pancreatic cancer. And this is, you know, people still like you. The intravenous vitamin C C's antioxidant.

Not when you're giving it a good enough dose. And when it's, you know, able to get to the the cancer. But yeah I, I, I think we're moving forward. It's a slow process. Pushing it hard.

You are you you are. Well, well, Jane, thank you so much for for taking this time. Yeah. On on your little time off with a side project. Chatting with me about, about these things.

I mean, I, I do appreciate you being such a strong force and and kind of driving this process forward, you know, so that other people can kind of follow along.

And then hopefully you don't have to be the one that's the spearhead the whole time. So, so thank you so much. And I know people can go to your website.

You have a course, you know, crash course and and repurpose drugs. You know, that's I it's like less than 100 bucks. I've taken it's phenomenal course.

So there are there is information out there that people can learn quite a bit in regards to these type of therapies. Exactly. And then use AI to sort of back it up as well, and then check the research, check the references that you get with the AI.

And yeah, I think it's it's a very useful tool and it will be a huge pressure relief for me. So I'll say, well, I, you know, I want you to get to have vacation sometimes.

Right. Well, Jane, thank you so much. It was so wonderful. God bless for everything that you're doing. Pleasure to talk to you as always, Michael. Thank you.