“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

Brad Power
September 11, 2024

“The biggest problem in oncology is not that we haven't got the treatments, but we just don't put them together and use them in the right combinations.” – Jane McLelland

“If you are stage IV, and you know that they haven't got the answer, what are you going to do? Are you just going to sit there and die, or are you going to have a go and try and look at different ways to actually save your life?” – Jane McLelland

Meeting Summary

When you are diagnosed with cancer, you will probably be confused and frightened. Most newly diagnosed cancer patients are unlikely to do much research. You probably lack the ability to figure out what is going on, much less non-standard approaches to your care. But suppose you are an exceptionally active and engaged patient. What should you do? Where could you look to get advice on lifestyle factors? You may look around for every edge you can find to enhance your immune system and general health to fight your disease. In a diagnosis that can arrive “out of the blue”, it is important to have agency and make a difference in things you can do, such as follow a healthy diet, reduce stress, get sleep, and exercise. But what is the science that shows the potential impact of these and other lifestyle factors on your possible outcomes? Your medical team is usually focused on testing and conventional treatment options like surgery, radiation, or chemotherapy. Your doctor may have no opinion or be opposed to potentially complementary treatments like supplements. You may be scared by the toxic effects of conventional treatment and be looking for less toxic options.

Jane McLelland is uniquely qualified to provide guidance on things you can do. After being given a terminal cancer diagnosis with only a few weeks to live, Jane dug up research, some decades old, in her quest to survive. Formerly a chartered physiotherapist, Jane had the medical background to dive into her diagnosis and treatment. Rather than aiming to cure cancer, which in many cases is unachievable, Jane’s approach was to stop it growing. Remarkably, her approach not only stopped it from growing, but it disappeared altogether. There are now clinics following her protocol, achieving successes. Her book "How to Starve Cancer" is a new approach to the treatment of cancer. This inspirational read is updated with a new "Metro Map", Jane’s unique and revolutionary route map to starving cancer. Jane intertwines her remarkable life story of terminal cancer to full recovery, describing how she discovered a unique cocktail of off-label drugs (drugs usually prescribed for other conditions) and supplements that effectively starve the cancer stem cell, the cell left behind by conventional treatment. Treatment for the stem cell is hailed as the Holy Grail, so this book plugs the missing piece into why we do not have a cure for cancer. Lead cancer researchers at top oncology centers are now using this book as a guide, and Jane has a huge following of tens of thousands on Facebook and other social media. Testimonials abound from happy and delighted recovered patients and from oncologists who use her methods.

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Why might you want to better understand how cancer metabolism can be used to treat your cancer?

Cancer treatments attempt to identify something that is different about cancer cells from normal cells, then target those cells or processes to hinder or kill the cancer cells. For example, chemotherapies attack cells that grow faster.

Understanding cancer “metabolism” – how cancer cells use carbohydrates, fats, and proteins from food to get the energy they need to grow and spread – and how it is different from the metabolism of normal cells can lead to additional treatment options. Compared to healthy cells, cancer cells use more glucose, produce less energy when making what they need to multiply and spread, and favor fermentation over breaking down glucose in the presence of oxygen. Unlike surgery, chemotherapy, or radiation, metabolic therapies don’t immediately remove or kill cancer cells. Instead, they keep cancer cells from growing by changing or slowing the cancer metabolism. The tumor eventually may shrink and die.

Researchers are looking for ways to block the unique metabolic processes of cancer cells while leaving healthy cells alone by reducing the food supply to the cancer cells and disrupting the messaging systems (“pathways”) used by cancer cells. For example, inhibiting “glycolysis” – the process of breaking down glucose to release energy – may help stop the development of cancer cells.

What can you do to address your cancer using a metabolic approach?

Metabolic approaches to treating cancer are in the early stages of research. They are not part of the standard of care, but show much promise. Cancer oncologists are not taught about the metabolic pathways beyond the “Warburg effect” (a “hallmark” of cancer cells – cancer cells preferentially break down sugar using glycolysis to produce energy, rather than using the more efficient approach of normal cells). There are multiple pathways that cancer can use to increase its nutrient uptake. Blocking those pathways can weaken the cancer.

- Your treatments must be personalized to you and your medical history, which ideally should involve multiple tests. Do research to discover the unique characteristics of your cancer and identify pathways that you might want to block with supplements and off-label drugs. You can use generative AI tools like Perplexity, Consensus, and ChatGPT. This metabolic approach is a molecular network approach using synergistic combinations, which means that you must be treated differently, such as your dosing. Supplements may work in one cancer, but not in others, or be complicated by other comorbidities you may have. For example, If you look at whether metformin works on its own, it generally doesn’t. However, metformin works well in combinations in blocking particular pathways.
- Get tests to learn what you might need. For example,
    - PET scans can look at your glucose, glutamine, and choline, and merge them to see which one is lit up most.

    - A gut microbiome test can see whether you have sufficient flora to create the right environment.
- Establish your best diet.
- Determine when and how to exercise.

What’s next for Jane?

- She will be working with a lab and clinic to try some cocktails for more aggressive cancers, like pancreatic cancer.

How can you learn more about Jane's unique approach to cancer?

- Read Jane’s book.
- Take Jane's online course (with discount code BRAD).
- Join Jane’s Facebook group: “Jane McLelland off-label drugs for cancer”.

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Meeting Notes

KEYWORDS

cancer, work, metformin, people, pathways, key, drugs, prostate cancer, statins, doctors, tests, bit, glutamine, block, cancer cell, cocktail, jane, point, metabolic, combination

SPEAKERS

Jane McLelland (84%), Brad Power (8%), Rick Davis (4%), Will LaValley (3%), Brian McCloskey (1%)

SUMMARY

Jane McLelland, a pioneer in metabolic approaches to cancer, discussed her metabolic map, highlighting the roles of glucose, glutamine, and fatty acids in cancer metabolism. She emphasized the importance of diet, supplements, and off-label drugs like metformin, statins, and niclosamide. She shared her personal journey, noting that her cancer markers dropped significantly with these treatments. She also discussed the potential of RNA vaccines and the need for personalized treatments based on cancer phenotypes. The discussion highlighted the challenges of accessing doctors who use off-label drugs and the importance of AI tools like Perplexity for tailored treatment plans.

OUTLINE

Introductions

- Jane McLelland is a former physiotherapist, stage IV cancer survivor, patient advocate, educator, and author.
- She is not a doctor but a cancer survivor.
- She has an online course.
- She has a metabolic map, which is a visual representation of the metabolic pathways in cancer cells.

Cancer Progression and Metabolic Pathways

- Jane McLelland has a theory of how the hallmarks of cancer can be lined up to explain cancer progression.
- Cancer starts in the cell membrane, influenced by an abnormal microenvironment.
- Key pathways are activated by the abnormal microenvironment, including nuclear factor beta and Stat3.
- Glycolysis and oxidative phosphorylation affect the microenvironment.
- She highlights the importance of understanding these pathways to develop effective cancer treatments.

Jane's Personal Cancer Journey and Treatment Approach

- Jane McLelland shares her personal experience with cancer, including multiple recurrences and the challenges she faced with traditional treatments.

- She emphasizes the importance of diet, supplements, exercise, and stress management in her survival.
- She discusses the role of off-label drugs like dipyridamole, lovastatin, and metformin in her treatment.
- She explains how she managed her cancer markers through various combinations of treatments and lifestyle changes.

The Role of Off-Label Drugs and Combinatorial Therapies

- Jane McLelland discusses the potential of off-label drugs like statins, metformin, and niclosamide in cancer treatment.
- She explains the concept of metabolic flexibility and how cancer cells adapt to different treatments.
- Jane highlights the importance of combining different drugs to achieve synergistic effects and improve treatment efficacy.
- She shares examples of successful treatments using off-label drugs and the need for more clinical trials to validate these approaches.

The Importance of Metabolic Flexibility and Personalized Treatment

- Jane explains the concept of metabolic flexibility and how cancer cells adapt to different treatments.
- She emphasizes the need for personalized treatment plans based on the specific metabolic phenotype of the cancer.
- Jane discusses the role of AI tools like Perplexity and Consensus in identifying potential treatment pathways.
- She highlights the importance of understanding the genetic and metabolic features of the cancer to develop effective treatment strategies.

Challenges in Cancer Treatment and the Need for Better Clinical Trials

- Jane discusses the challenges in cancer treatment, including the lack of effective combinations and the focus on single-drug trials.
- She emphasizes the need for more clinical trials to validate the use of off-label drugs and combinatorial therapies.
- Jane shares her vision for developing better treatment cocktails and the importance of philanthropic support for this research.
- She highlights the potential of RNA vaccines and other emerging treatments to address cancer at the epigenetic level.

Audience Questions and Discussion on Evidence and Personalized Treatment

- Brad Power moderates a discussion on the evidence supporting the use of off-label drugs and personalized treatment.
- Rick Davis raises concerns about the general applicability of treatments like Metformin and the need for personalized approaches.
- Jane explains the importance of understanding the specific metabolic pathways involved in different cancers.

- She emphasizes the need for more research and clinical trials to validate these approaches and develop better treatment strategies.

The Role of the Microbiome and Exosomes in Cancer Treatment

- Jane discusses the role of the microbiome in cancer progression and the potential of targeting toll-like receptors.
- She explains the concept of exosomes and their role in cancer cell communication and epigenetic changes.
- She highlights the potential of RNA vaccines and other emerging treatments to address these pathways.
- She shares examples of successful treatments using antimicrobials and other targeted therapies.

Final Thoughts and Closing Remarks

- Jane emphasizes the importance of maintaining an open mind and exploring all available treatment options.
- She encourages patients to educate themselves about their cancer and the potential benefits of off-label drugs and personalized treatments.
- She highlights the need for more research and clinical trials to validate these approaches and develop better treatment strategies.

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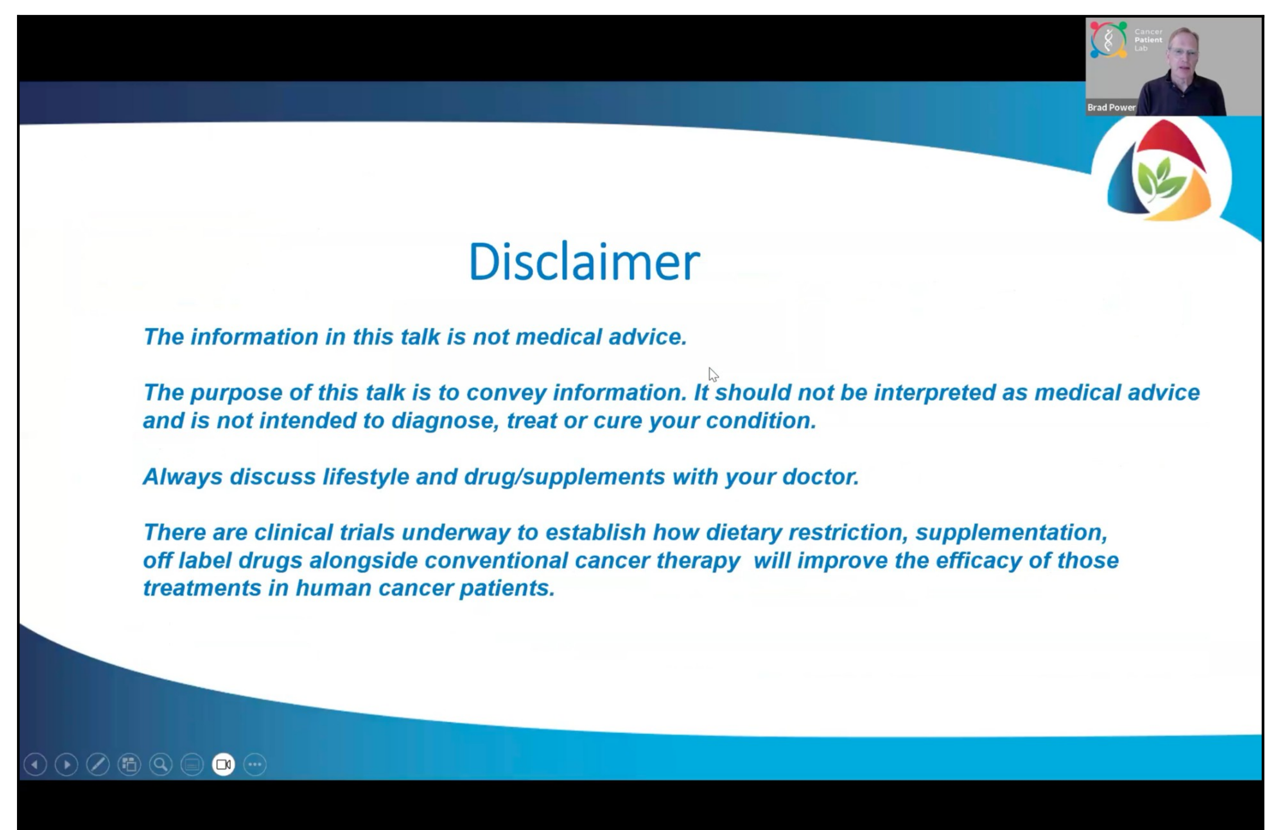
TRANSCRIPT

Brad Power
This is our weekly session of the Cancer Patient Lab. Today, we're honored to have with us Jane McLelland, who's a pioneer and thought leader in the area of metabolic approaches, starving cancer, and nutrition. We've been looking forward to this conversation for some time.

Let me cover a quick few quick housekeeping notes that we have at the beginning of every session. This is for information purposes only. This is not medical advice. What we try to do is arm patients with information that they can take to their medical team. We are a nonprofit patient-led volunteer organization. We always appreciate donations, which you can do through our website.

I don't know if Will Lavalley introduced me to Jane. Many people have introduced me. Bapcha Murthy made one of the connections. She is well known as a leader in the field. We're very interested in hearing what she has to say.

We've had a number of sessions about integrative oncology, nutrition, complementary therapies, from Donald Abrams at the UCSF Osher Center, to Nigel Brockton at AACR, to Bapcha on scams. Many of the people here have a good understanding of your work and are looking forward to hearing what you have to say.



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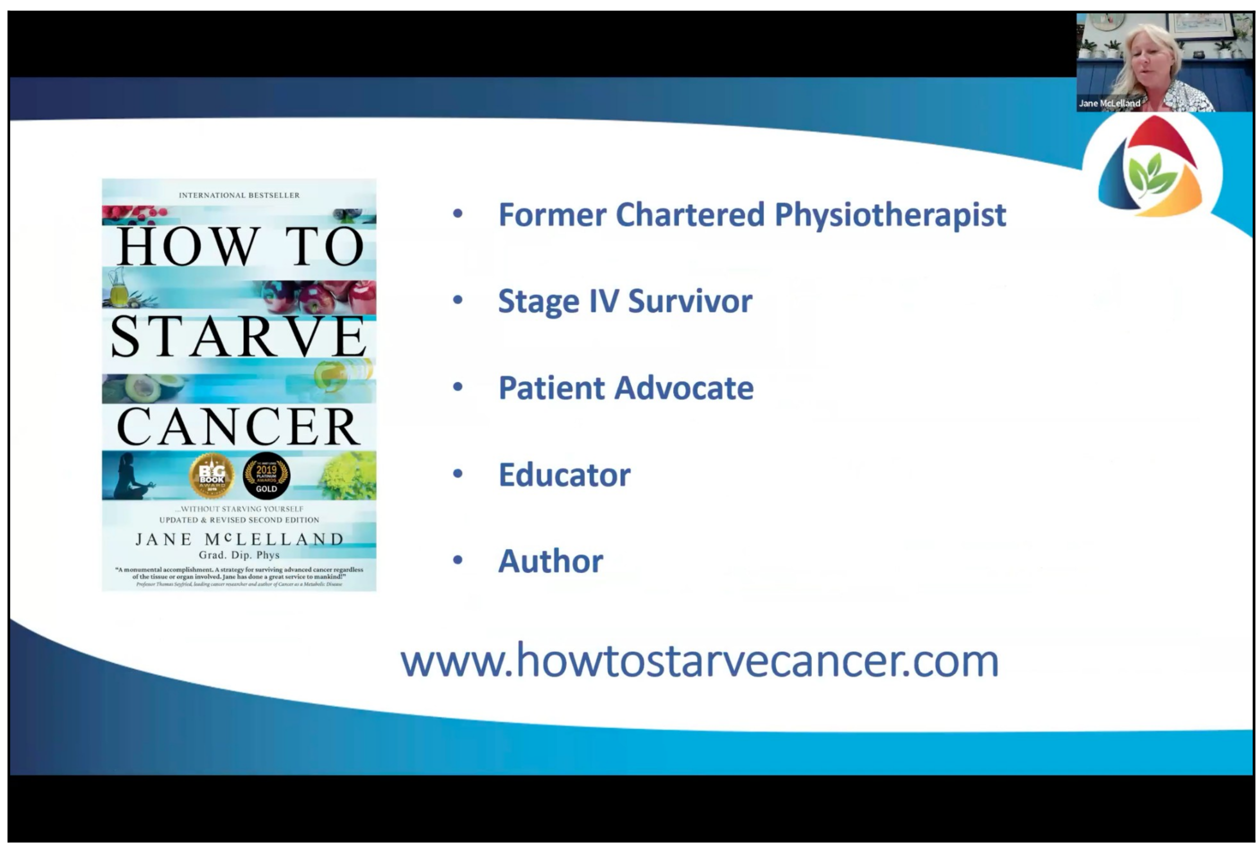
Jane McLelland 2:34
This is my disclaimer. It's important to make sure that people know I am not a doctor; I'm not a pharmacist; and these are suggestions and things that I have done for myself.



This is me, a former chartered physiotherapist. I haven't worked as a physio for a long time. I’m a Stage IV cancer survivor. It came back multiple times. Patient advocate, educator, and author of How to Starve Cancer, which looks like some of you have read.

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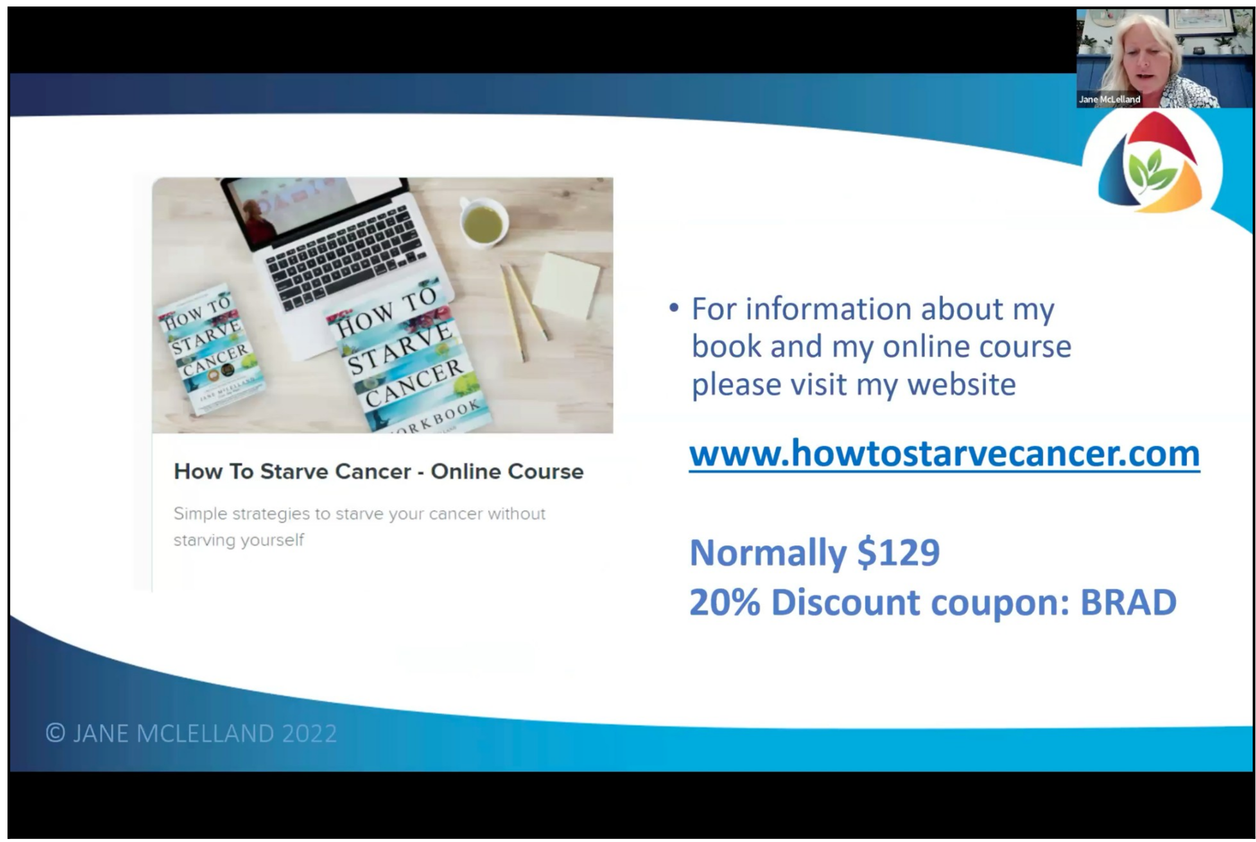

I also have an online course. I'm going to do a special discount for you, BRAD. I'll do that straight after. I'll put that as a discount code if anybody hasn't seen my online course. I don't know whether that's something I'm allowed to do, but I've just done it.

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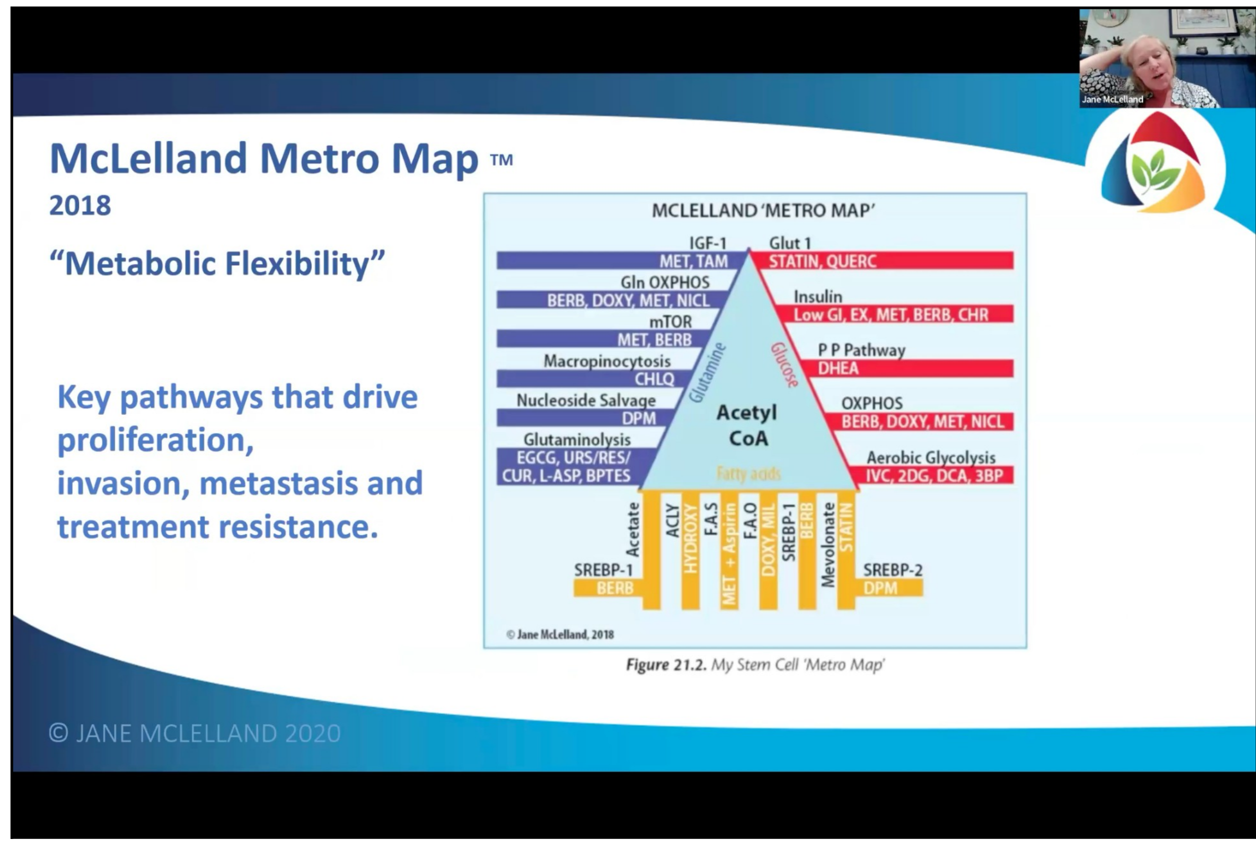

I'm most known for my “Metro Map”, which is a metabolic, quick look at what is happening in the cancer cell. It's essentially looking at the three macros in the diet. You've got glucose on one side, glutamine and other amino acids on the left, and then along the bottom there are fatty acids.

That's a very quick snapshot. I can't say I’m famous, but people recognize me for the Metro Map. That's what people know me for.

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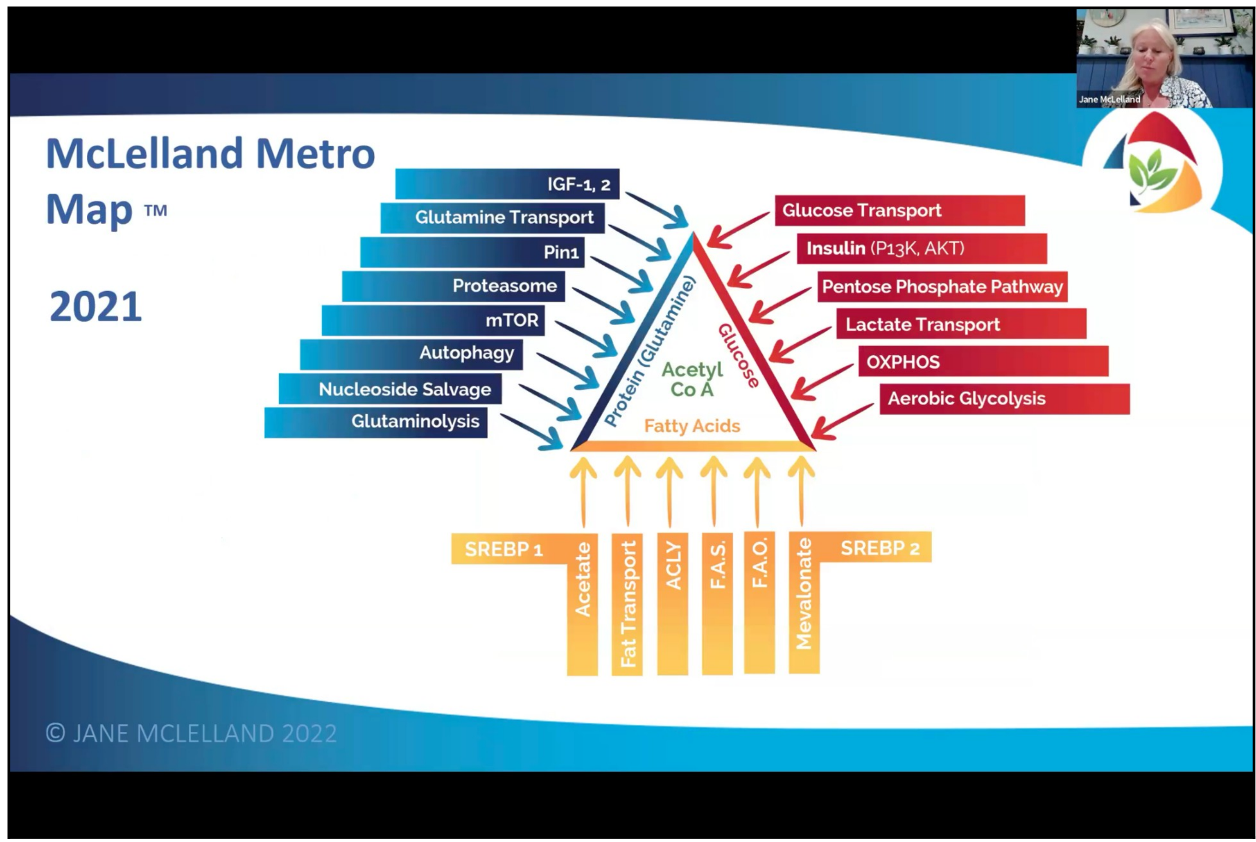
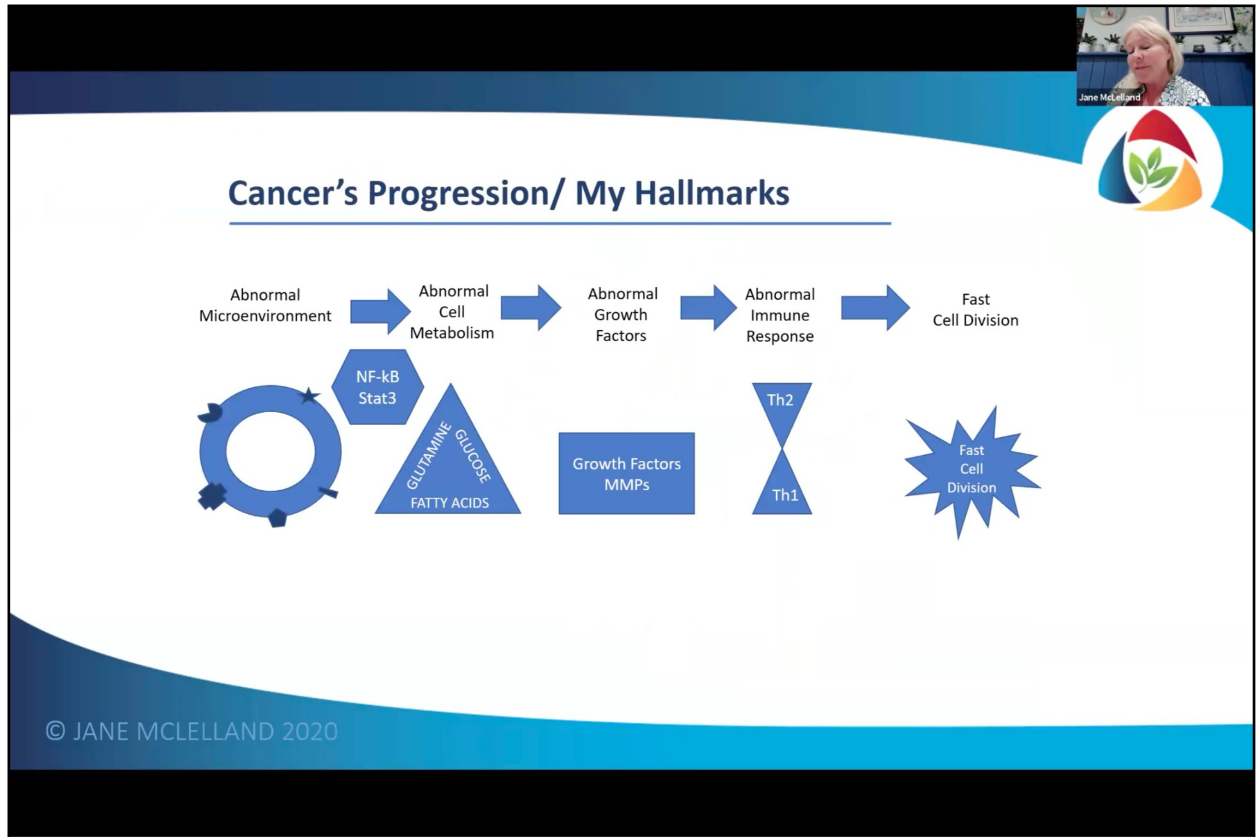


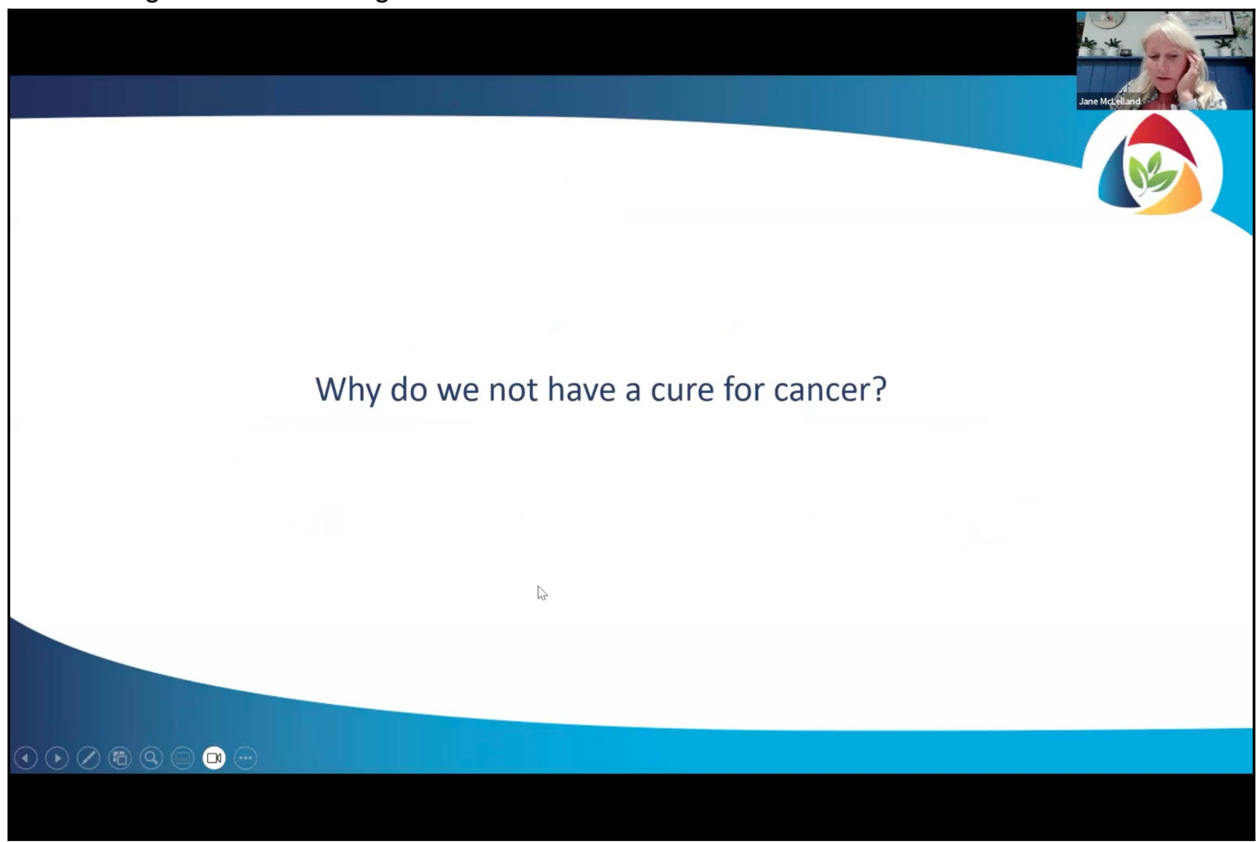
This a more up-to-date one. It's got some more pathways on there. I updated my book in 2021. I put some transport on there, for example, lactate transport. I've also got glutamine and other transporters, amino acid transporters, like the XCT. I do a full chapter on ferroptosis In my second edition as well. And fat transport as well. There are a few other things going on in there, which hopefully paint the picture of what's going on with the metabolism of cancer.



People don't really know this section of what I talk about, which is cancer's progression, or my precis of the hallmarks of cancer. There are about 18 or 20 different hallmarks, with emerging hallmarks, including the microbiome. I've chopped it down to these as my approach to try to look at how they all fit into cancer progression. Because when you look at the hallmarks, they're always noted as a round circle. There's no start and no finish to it. But I've tried to break it down into what actually happens.

We start with the abnormal microenvironment. That's what is going to cause cancer. Cancer starts in the cell membrane. This is something that people don't know. This is my theory. You can challenge me on this afterwards, if you like. But cancer starts in the cell membrane. You can't suddenly get mutations in the nucleus without it passing through the cell membrane. The cell membrane is affected by the abnormal microenvironment and everything that happens with that. I'll go into more detail about that in a minute. That'll fire up these two key pathways, nuclear factor-kappa beta and Stat3. They then switch on and make some of the pathways in the cancer more active, so the glycolysis and the OXPHOS. This is another bit of my metabolic map here. Once you have the glycolysis happening, you have a secretion of lactate. This is very acidic. The environment doesn't like it, and you then grow lots of excess blood vessels in order to take away the lactic acid. You get these growth factors, and also MMPs, which tend to be a little bit forgotten with general oncology. Once you've got all of that going on, this switches off the environment. The acidic environment switches off the immune cells. It switches the macrophages from being anti-cancer to being pro-cancer. You have m2 macrophages, and you also get the shift in the balance of the Th2, which is more of your humoral response to more of

your cellular response, which is anti-cancer. Once that's happened, you then get this fast cell division. That's a very quick precis of what happens. The beginning bit is really key, though. I called the talk “Beyond the Metro Map.” We may go backwards and look at what happens more before we get all these changes.



We don't have a cure for cancer. Stage IV is still deemed to be incurable. I'm going to discuss my thoughts on why I survived.

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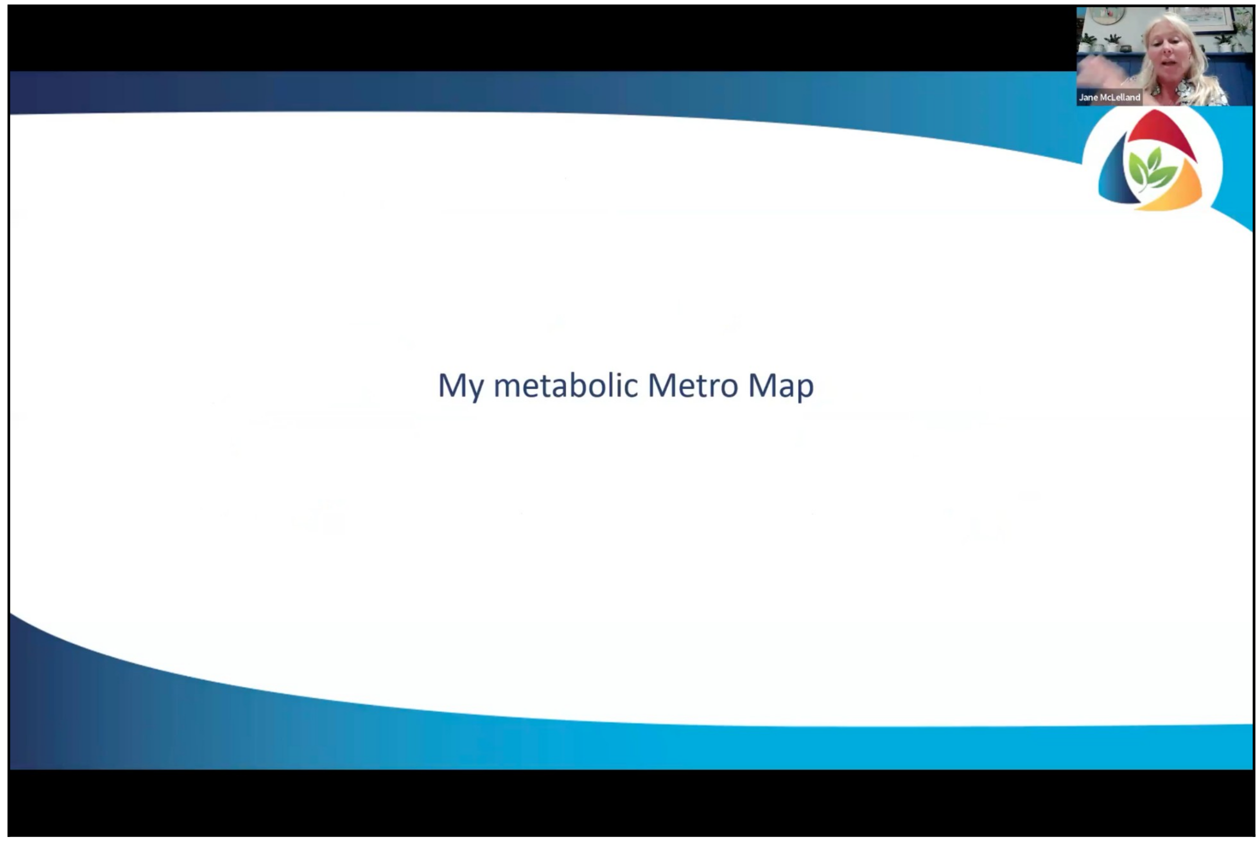

I had a quick chat about the metabolic Metro Map. We'll go into that maybe a little bit more.

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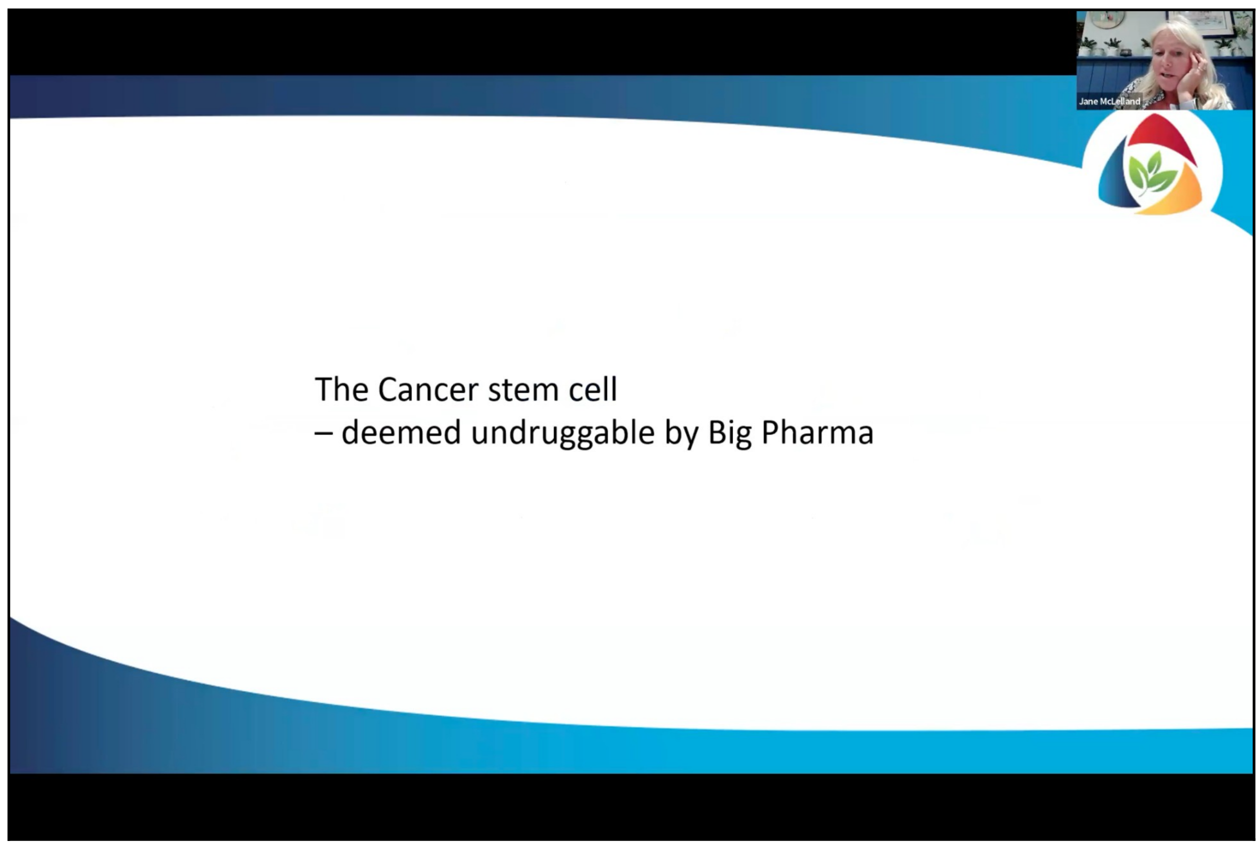

And the undruggable cancer stem cell. It is druggable.

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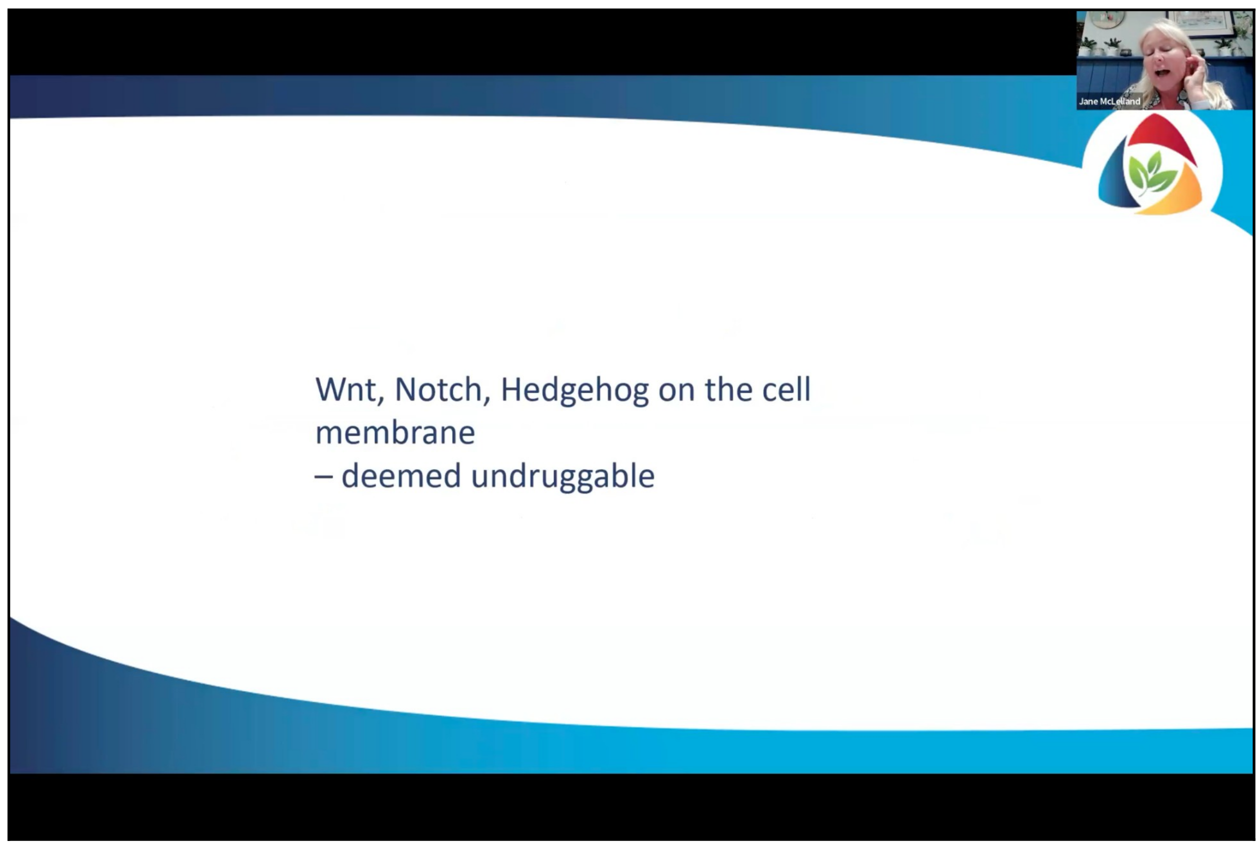

These things that are happening on the cell surface, Wnt, Notch, and Hedgehog, which are also deemed to be undruggable. But they are, and I'll show you what will target those.

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The Problem in Oncology

“…we are not limited by the science; we are limited by our ability to make good use of the information and treatments we already have.

Too often, lives are tragically ended not by cancer but by the bureaucracy that came with the nation’s investment in the war on cancer, by review boards, by the FDA, and by the doctors who won’t stand by their patients or who are afraid to take a chance.”

Dr Vincent De Vita,
former director of the NCI, author The Death of Cancer
Editor of The Cancer Journal

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The biggest problem in oncology is not that we haven't got the treatments, but we just don't put them together and use them in the right combination. “Too often, lives are tragically ended, not by cancer, but by the bureaucracy that came with the nation's investment in the war on cancer, by review boards, the FDA, and by the doctors who won't stand by their patients or who are afraid to take a chance.”

I'm afraid I have been let down multiple times by doctors. I did have some fantastic doctors, but I had some truly awful doctors who would not take a chance on me at all. I had to really push to get the treatments that I wanted.

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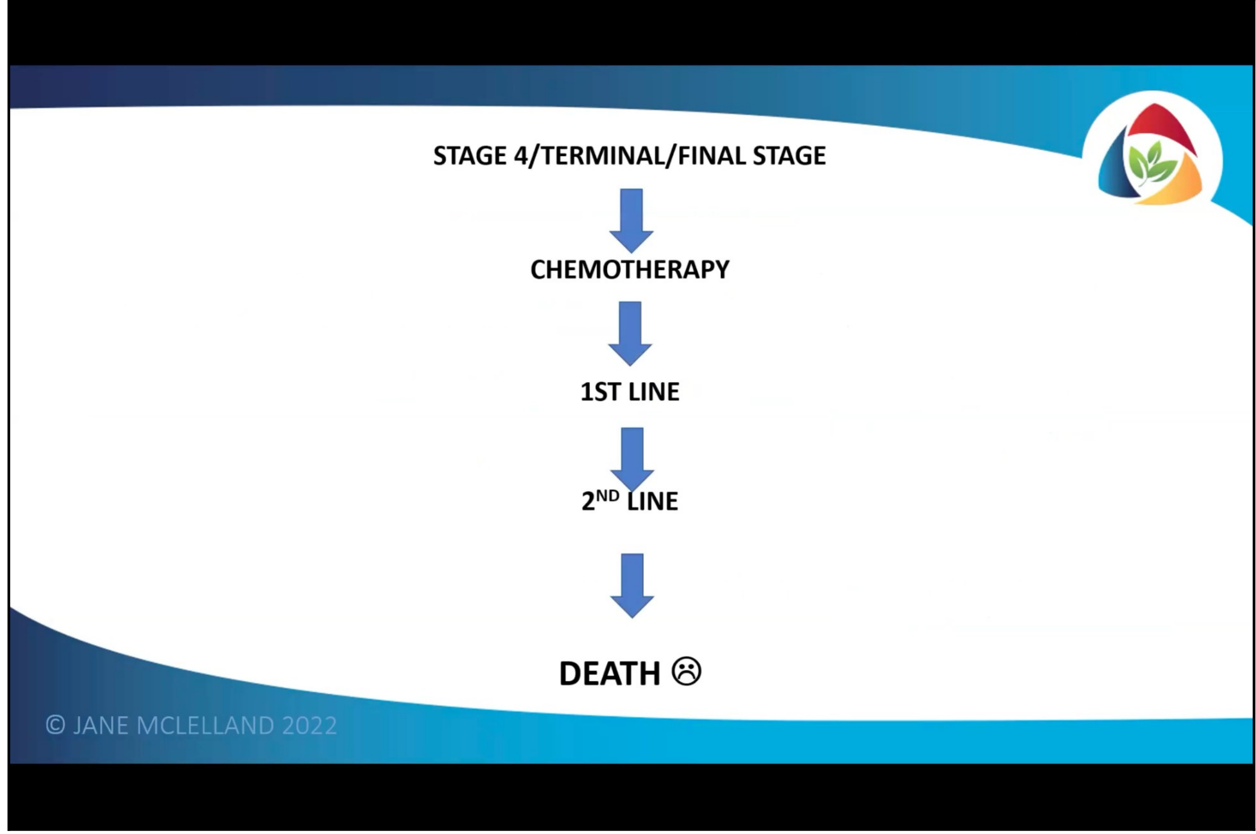

Stage IV cancer patients are normally seen as terminal, final stage. That's it. End of the line. You might be lucky. They might give you some chemo. If they give you high doses of chemo, I think that will probably kill you. In my view, you might be lucky to get a first line and then possibly a second line of treatment. Those things can be very useful if they're targeted. But the ultimate is that doctors expect you to die.

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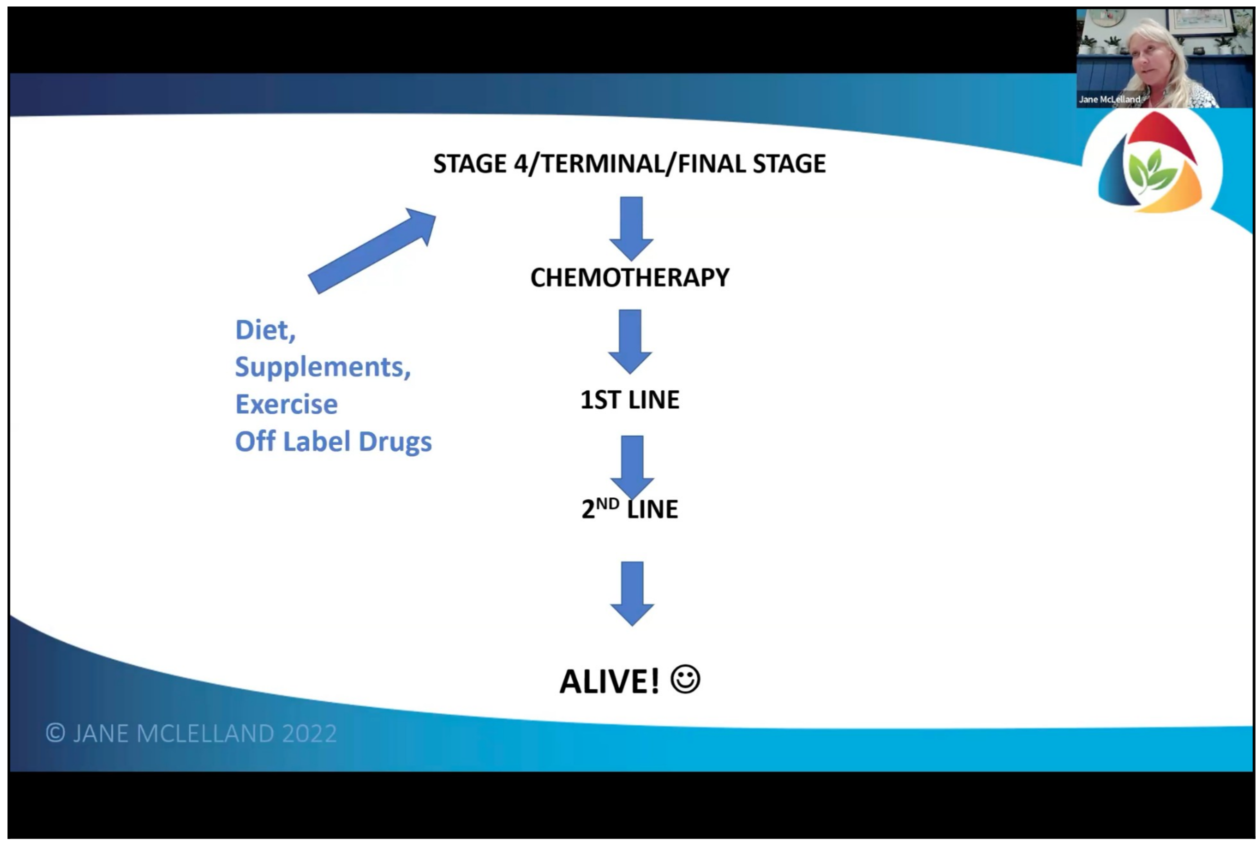

For me, I added several things, diet, supplements, exercise, and off-label drugs. I also did things like manage my stress and try to increase my sleep. All of those things are important too, and I'm still here.

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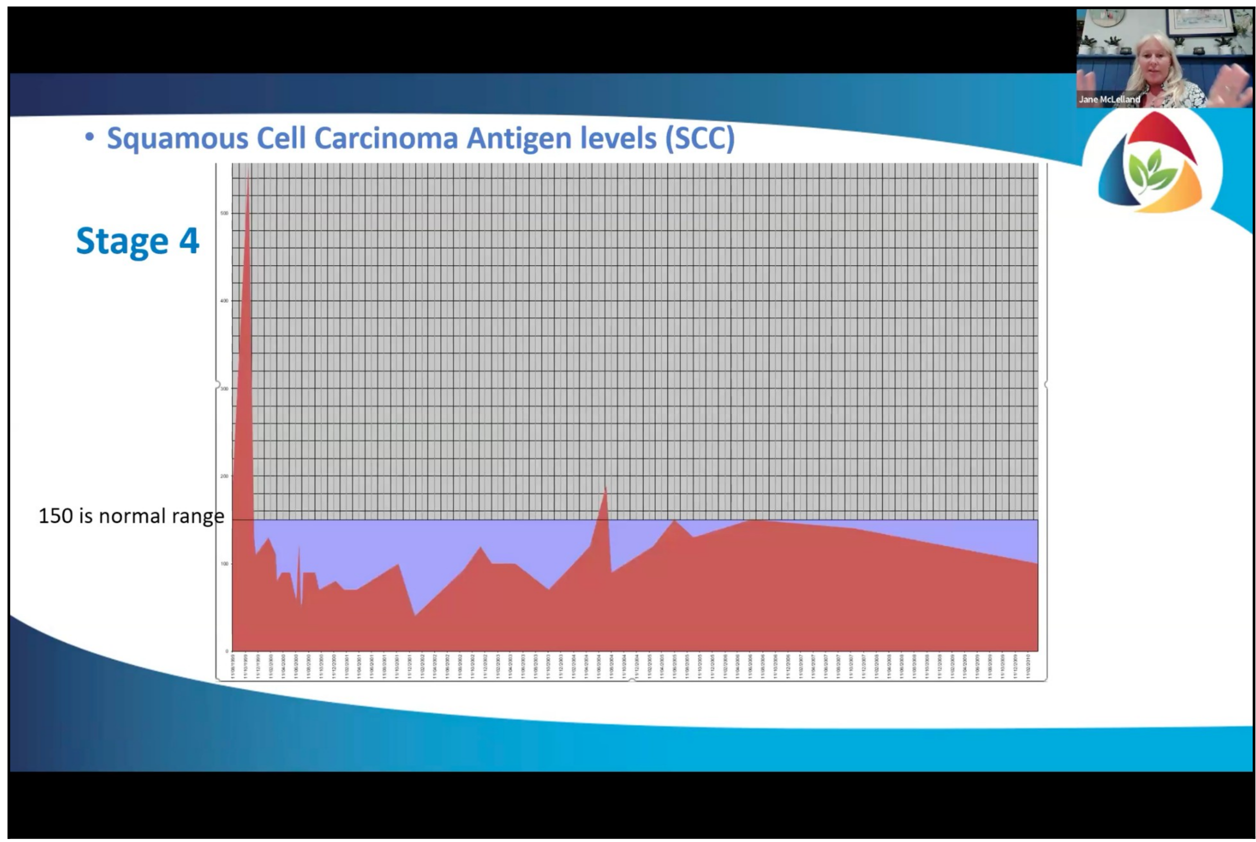
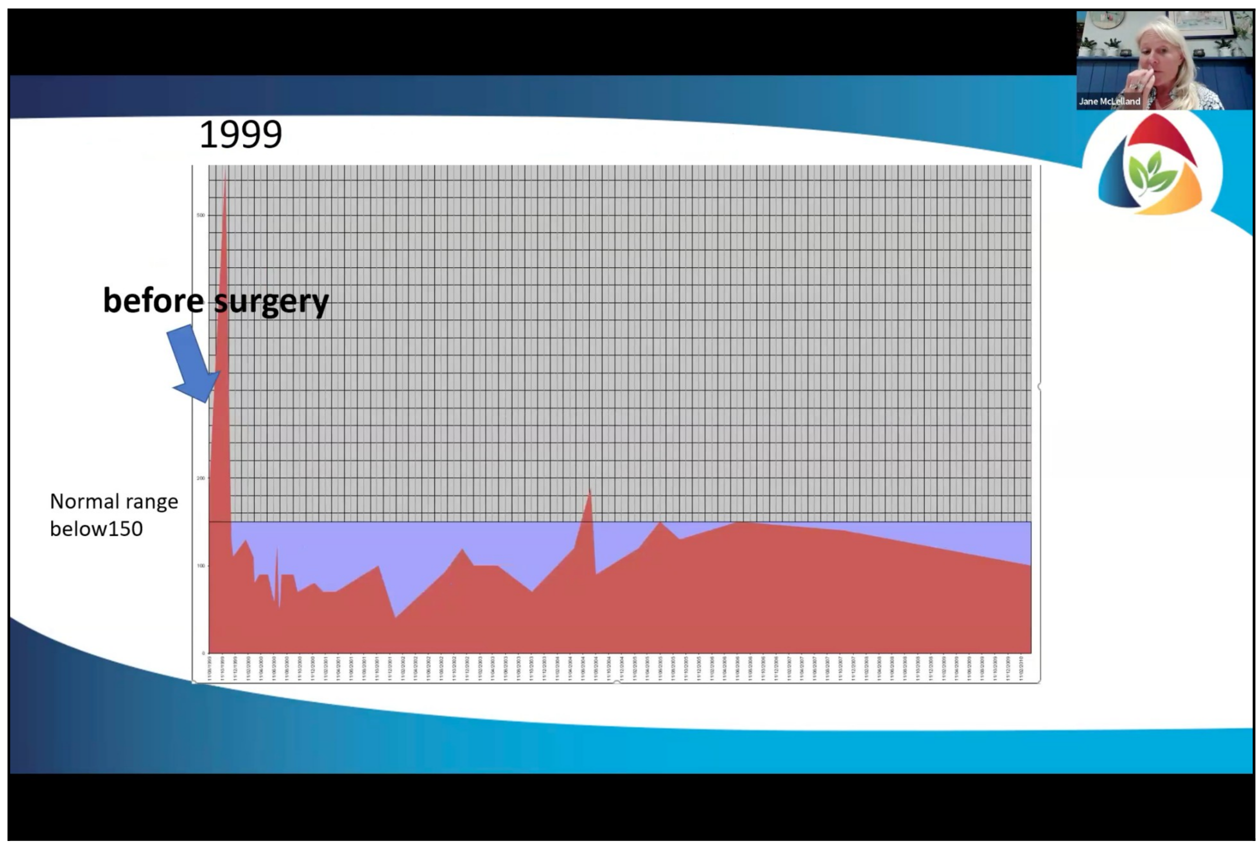
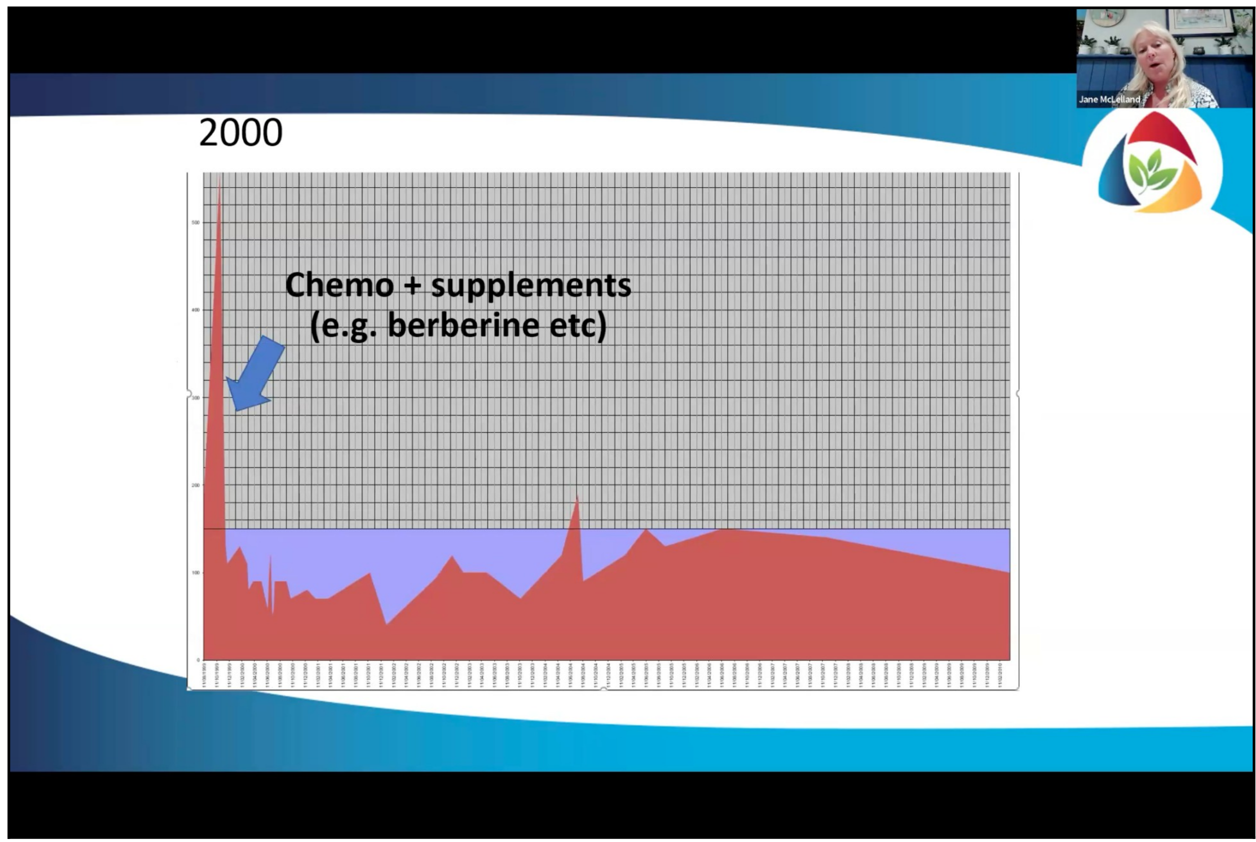


This is looking at the data of my antigen levels and what happened when I was diagnosed with stage IV cervical cancer, first diagnosed in 1994. Then in 1999 it came back in my lungs. Nobody thought to test my antigen levels before I became stage IV. 150 is the normal range. When I was diagnosed, I was about 250 or something.



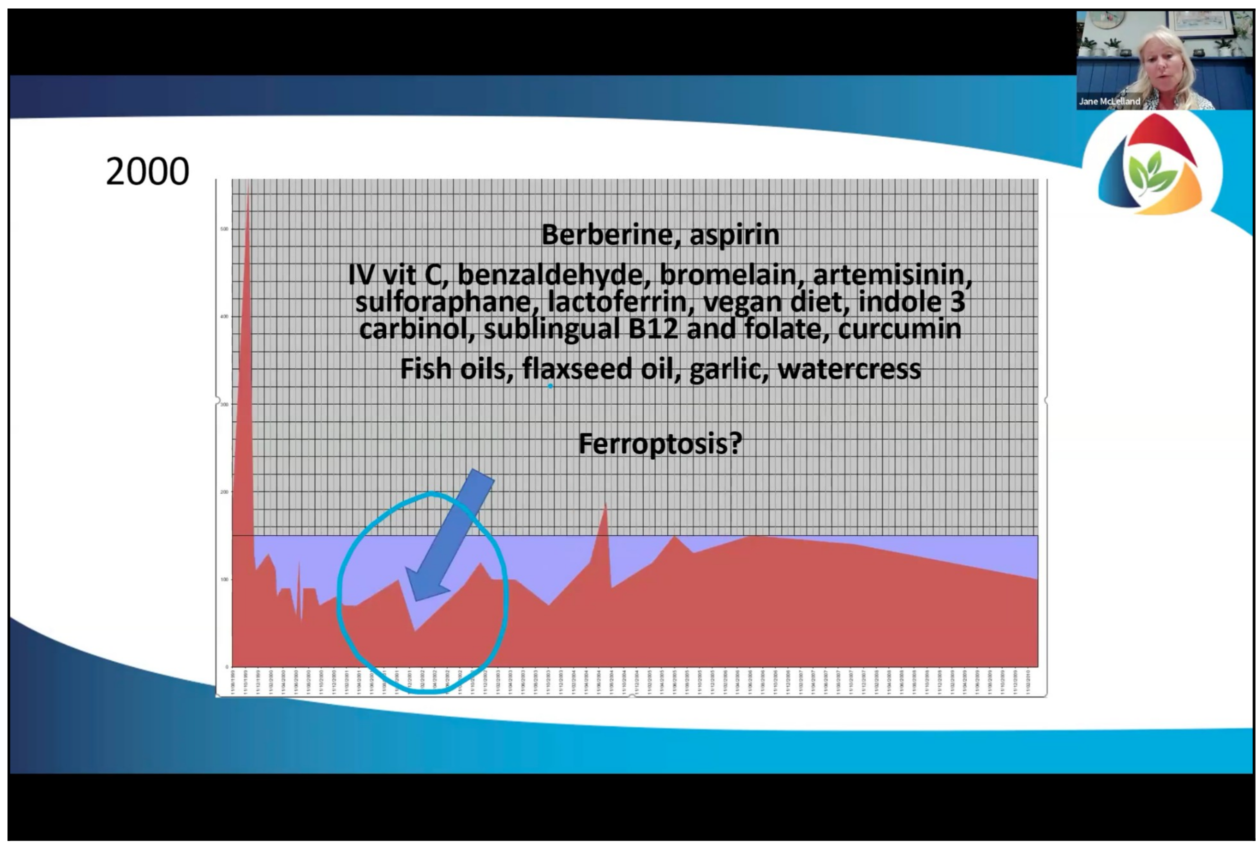
Before surgery, that's where I was, and then immediately after surgery. Surgery is a big problem. It has a massive effect on metastasis. This is one of the things we could really, really improve, by having better cocktails that are given around surgery. We know that there are certain stress factors, catecholamines, all sorts of things that will actually promote the spread of the cancer. Propranolol, statins, are really important pre- and post-op. There has been some fantastic work on that, and also non-steroidal anti-inflammatories. Ketorolac for ovarian cancer, for example, can be hugely beneficial. You can stop the metastasis by about 20% to up to 70%, which is mammoth. We could be really stopping the progression and stopping this flood of people and all of these excess cancer deaths that we're getting now, if we just looked at the timing of when we did things, when we start to give some of these off-label drugs potentially. There are some very good articles. I can find those and send them out if people want to see them afterwards.



Post-chemo, my markers shot right down. This is where I should probably put my graph of berberine and chemo being massively useful.

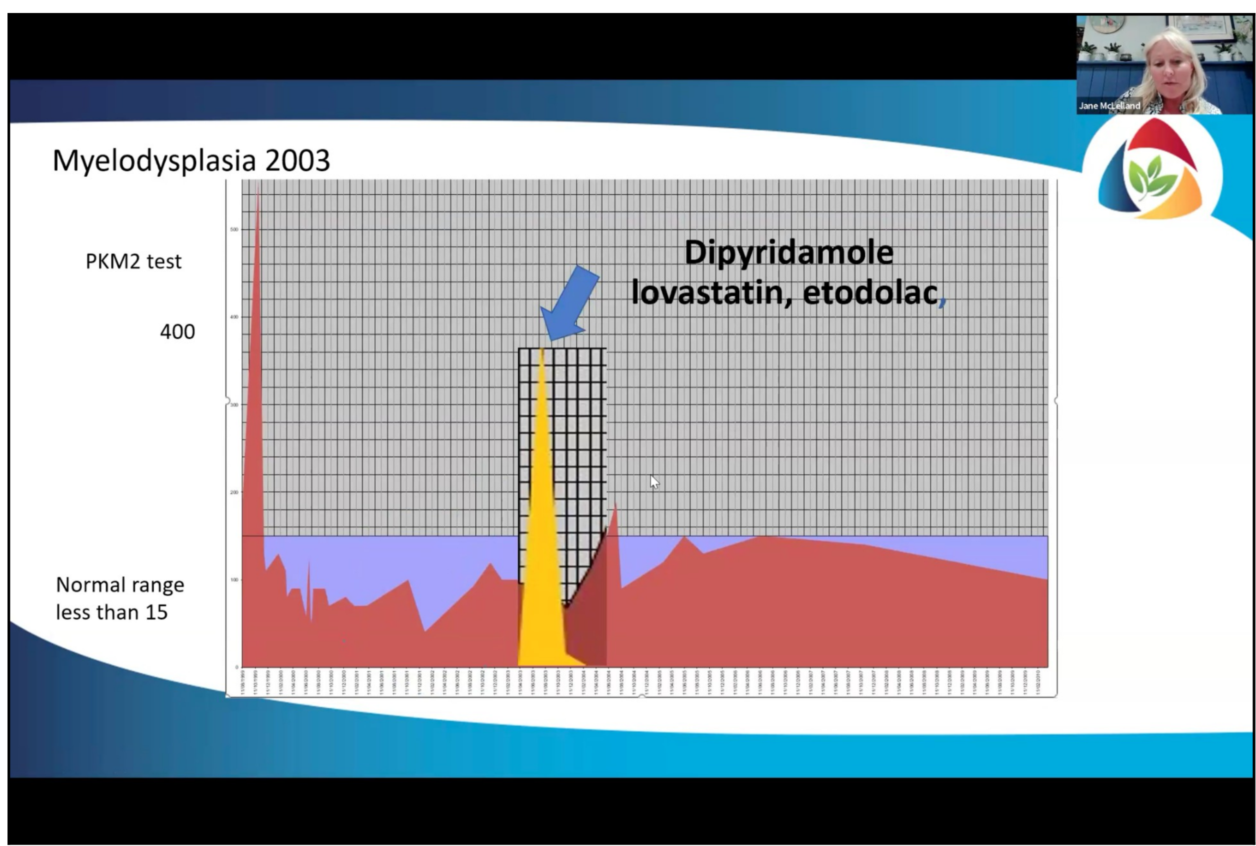
I've got it in my book. I've got one graph for brain cancer, but it does affect other cancers as well. But in brain cancer, if you have berberine, it has a cancer kill rate that is quite high. It's about 80% or something extraordinary. But then if you add carmycene, this was a trial done with carmosine and berberine, then you get a joint cancer kill of about 93%. But the carmycene has about a 40% cancer kill. Berberine is much more effective. Metformin, which is like the drug form of berberine, will have a cancer kill of about 40%. Temozolomide will have a cancer kill rate of about 40%, but the combination of the two shoots it up to about 90%.

I dropped right down having berberine. Berberine was one of the first supplements that I really got into, but it wasn't an oral supplement back then, it was a tincture. I couldn't get it as a supplement, but it's become more and more accepted to be a really, really powerful anti-cancer supplement.



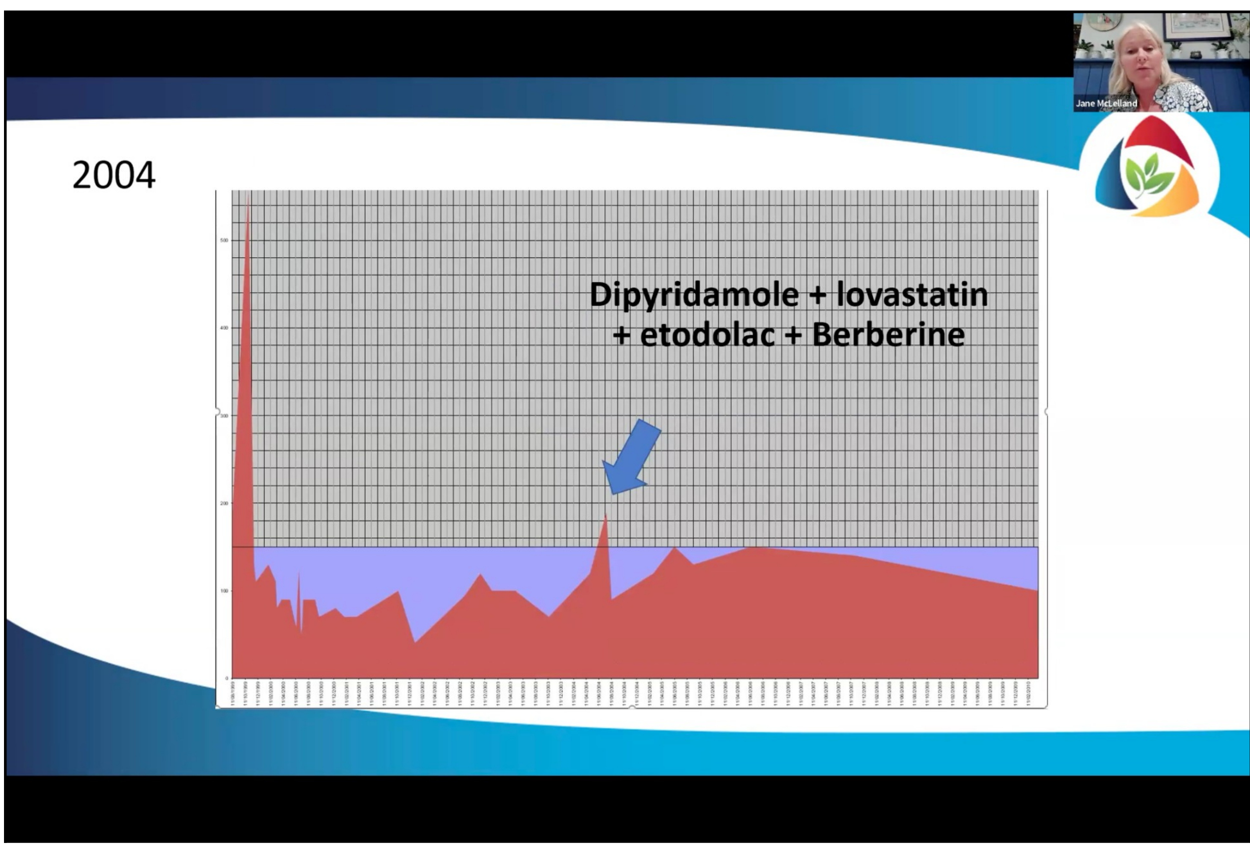
I then bounced around with my markers a little bit for a while, but this is where I had intravenous vitamin C, and my markers dropped way down to the lowest they ever were. Now, during that time, I was doing detox, I was taking artemisinin, I was trying to get rid of parasites and readjust my gut. I was on a fairly restrictive diet at that point, and I took a whole bunch of these things here.

All of these supplements will have an effect and stimulate this ferroptosis process. If you don't know what ferroptosis is, it's a newly discovered way of creating cancer cell death using iron. I'm not saying take iron, but this is the way that is getting some more effective remissions.

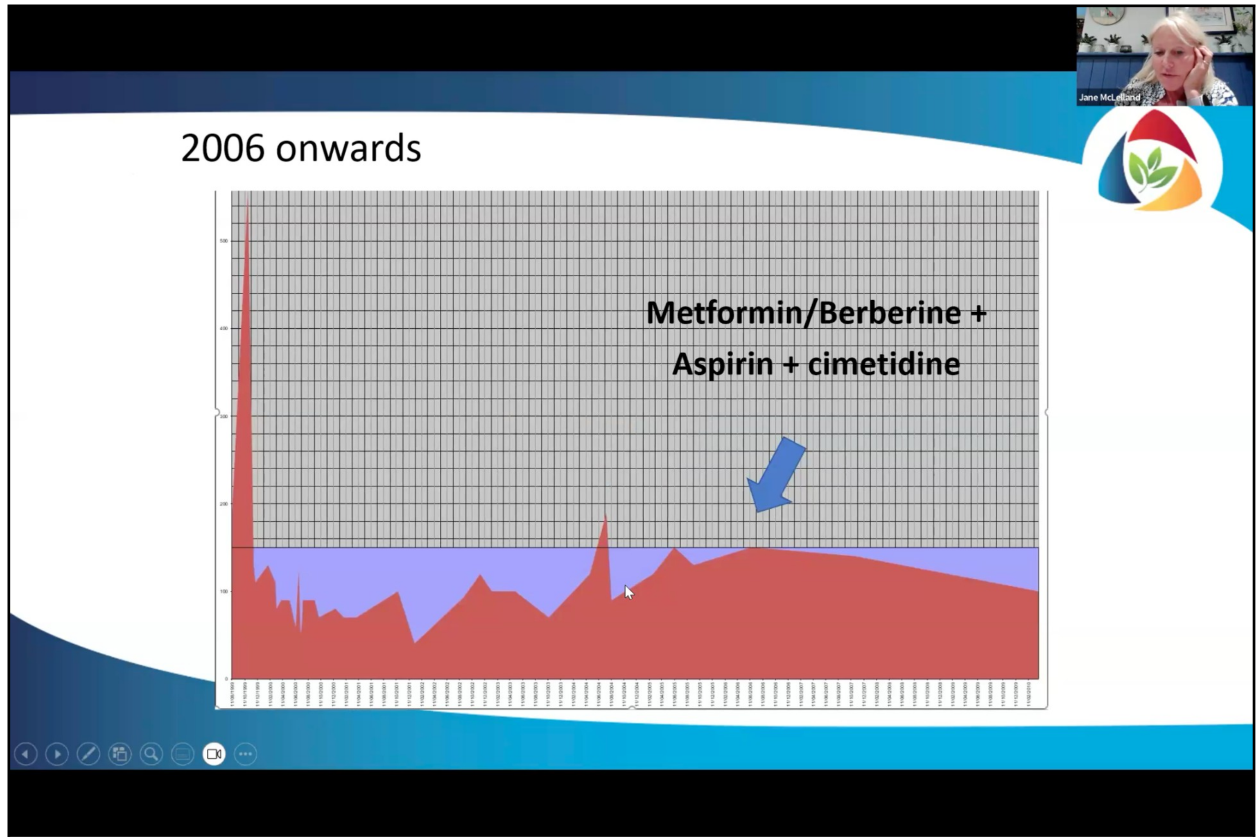


I was tickling around. I thought I was doing okay, using all of these natural approaches.

Then in 2003 I started to feel horribly ill. I got really tired. I was short of breath, and I had some blood tests done. You could see I had myelodysplasia, and I had this PKM2 test done. PKM2 is pyruvate kinase M2. This is only there in cancer. It was way up. The normal level for that should have been way down. Here at 15. I was near enough 400, so at this point, I was panicking, because myelodysplasia leads to AML, and AML is horrifically tricky to treat, particularly if it's a result of the radiotherapy and the chemo, which I'd had as far in massive doses. I'm not really going through my thought process of why I picked these things, but there were specific thought processes which I write about in my book. But the combination of dipyridamole and lovastatin are excellent. I'll talk about that with the Metro Map in a minute. The etodolac is a non-steroidal anti-inflammatory normally used for arthritis, so that combination was hugely effective. It dropped my markers way down within about seven months.



During that period, I took some of the drugs only for about three months, because I didn't know what I was doing. I was my own guinea pig. I was more focused on the natural side at this point. I didn't want to be taking the drugs. Then I was okay for a while. I went off the rails. The next year, I went to Ireland, did a sailing event, drank all the wrong things, and Guinness and Murphys and all sorts of things, was in a mess, and my markers started to shoot up. That's like an exponential climb there, at which point I panicked, and then I started taking my cocktail again. I'd dropped the berberine at that point. I don't know why. I had just forgotten about it. But then I put it back into the cocktail, and whoosh, my markers came straight back down into normal range.



Then they sort of slowly climbed a little bit, bounced around under the normal range. About 2006 or 2007 we had bird flu scares, and I didn't know I had cystic fibrosis at this point. I'm a late diagnosis, but I was really suffering from a lot of infections. I wasn't quite sure why. I just thought it was a result of the chemo and the lung tumors. I just didn't know anything else. I looked into cimetidine, because that was recommended to try and boost your immune system. I’ve just taken it today. I take 400 milligrams in the morning, 400 milligrams at night, and this helps to reverse that TH1-TH2 imbalance that you get. It worked with me from that point onwards. Metformin was also something that I started taking at this time as well.

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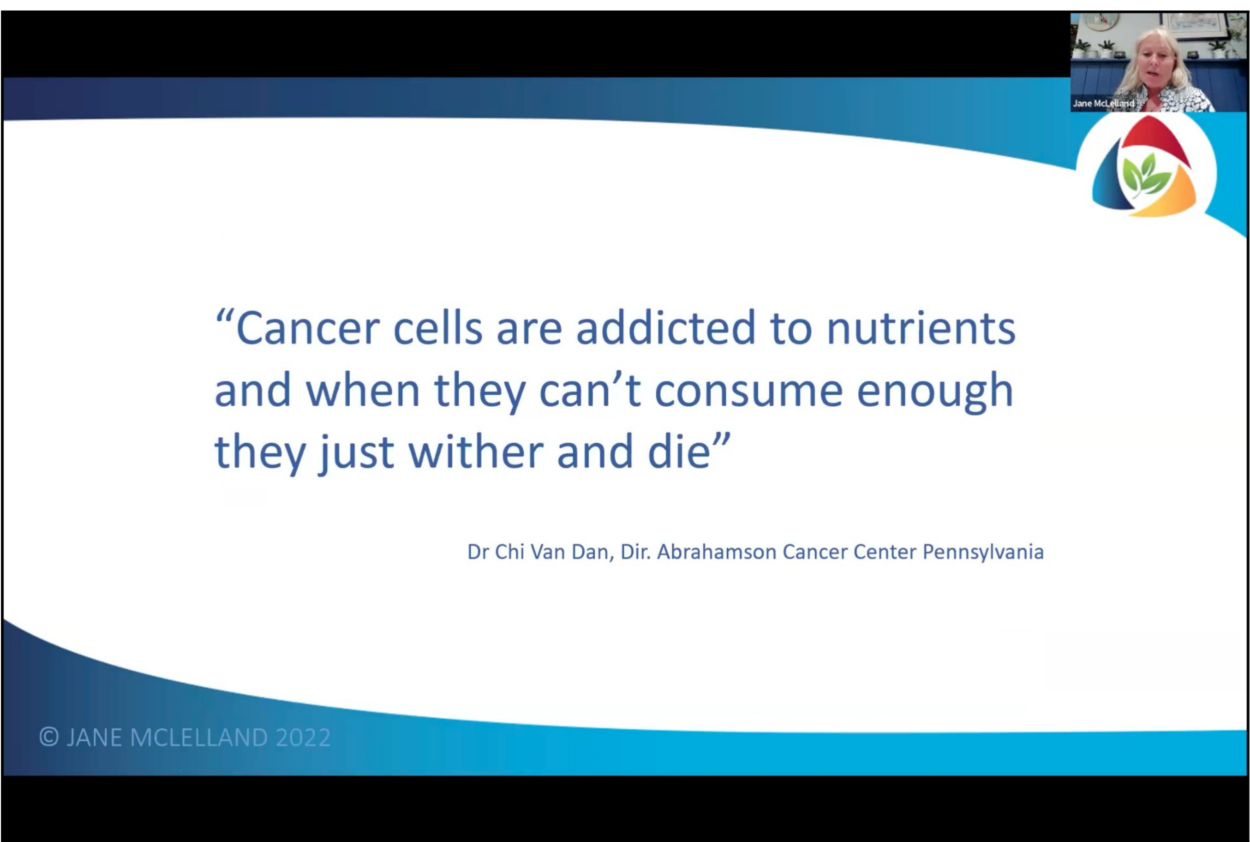
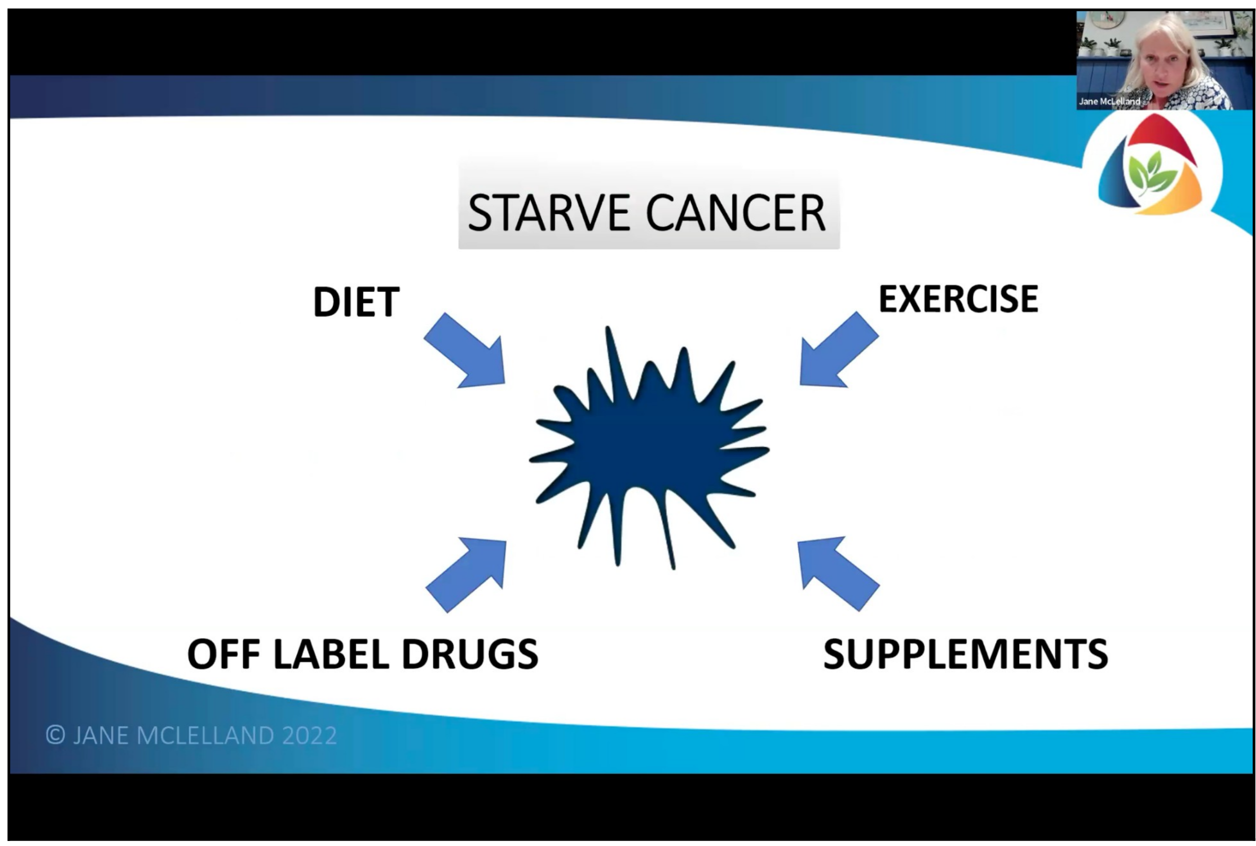


“Cancer cells are addicted to nutrients, and when they can't consume enough, they just wither and die.” This has been noted in many trials that have been done at the Abrahamson Cancer Center in Pennsylvania.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



Starving cancer is crucial for people to actually get long term remission. You can do that in these four ways. Diet on its own doesn't work. Supplements: you have to be lucky if you have a slightly earlier cancer, you may get away with less of the drugs. But really, my advice is to look at all four things if you've got stage IV cancer.

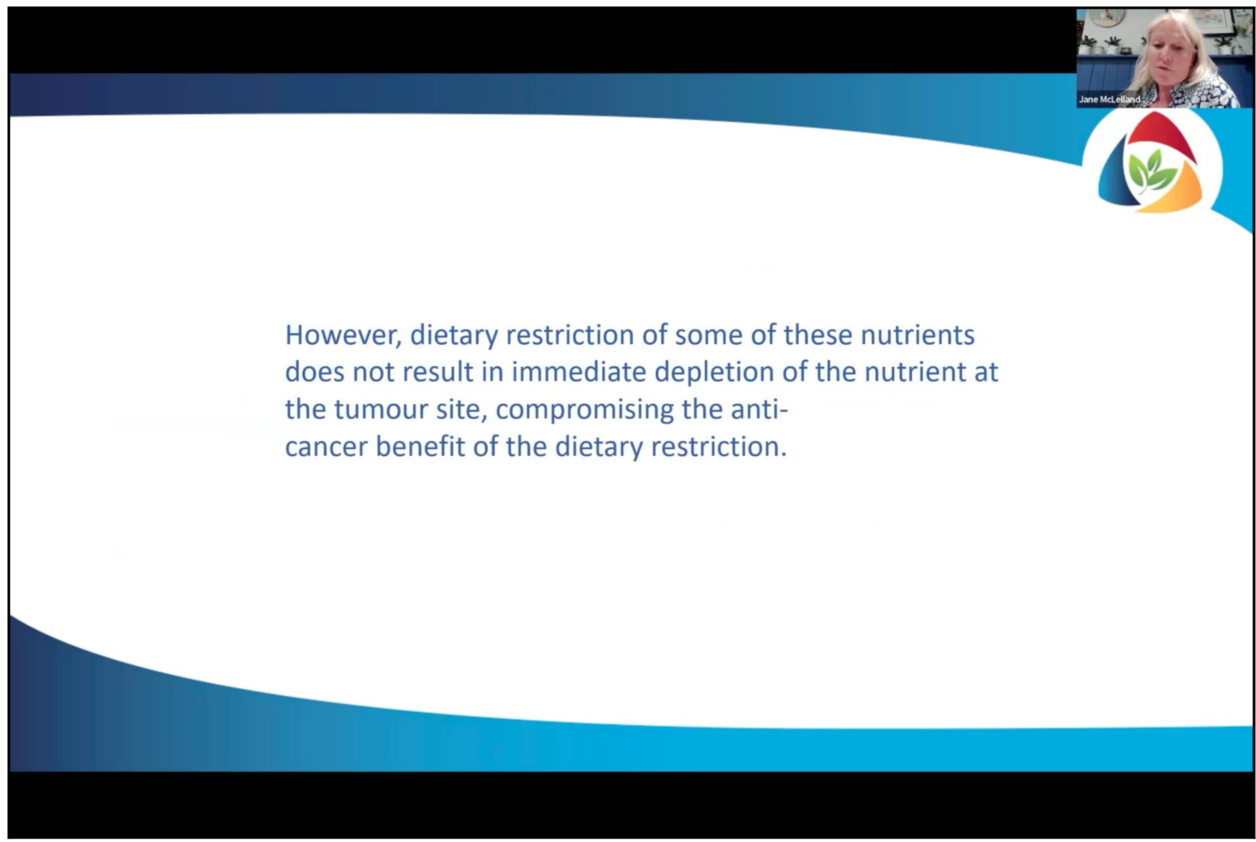
“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

Cancer is always hungry!

The vulnerability of cancer cells to **nutrient deprivation** and their **dependency on specific metabolites** are emerging hallmarks of cancer

e.g. glucose, glutamine, asparagine, aspartate, fatty acids, lactate, serine, arginine, methionine, cysteine
i.e., Tumour starvation..

Cancer is always hungry. It is vulnerable to nutrient deprivation, and it does depend. Certain cancers will depend on specific metabolites, so some will depend more on asparagine, some will be more on arginine. Most cancers are very hungry for both glucose and glutamine, but there are specific amino acids which are unique to different cancers.



Dietary restriction doesn't result in immediate depletion of the nutrient at the tumor site, compromising the anti-cancer benefit. In other words, it doesn't work on its own. It also takes time for these things to work. You have to persevere. I know a lot of people think I'm just pro ketogenic or something. I’m not. The ketogenic diet can work very well at the beginning of a cancer journey, particularly if you are metabolically a bit of a mess. But after about 30 days, it starts to do that alteration. It sort of works its way around. It reroutes itself, and it finds different ways to resist the ketogenic diet.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

Why don’t more doctors prescribe off label drugs?

-Cheap, low toxicity, they enhance treatment efficacy

Legally they are not constrained if acting in the best interest of the patient.

Terminal patients find it hard to access doctors who are able to exercise ‘creative compassion.’

Why don't more doctors prescribe off-label drugs? Because they are mostly cheap, mostly very low toxicity, and they do enhance the treatment efficacy. Legally, doctors can prescribe them happily if they're acting in the best interest of the patient. But I found it hard to access doctors who are able to prescribe on this creative compassion.

My oncologist, back in 2003, was the one who prescribed the lovastatin and the etodolac, because I gave her information to show that the combination of the two was five times better to create a cancer kill than one or the other individually. Back then, she was lovely, one of the good doctors, and she prescribed those for me. She'd been looking into statins herself, and she didn't know which one would be best. Lipophilic statins are, in fact, the best ones for cancer. Atorvastatin. I took lovastatin at 80 milligrams, atorvastatin, simvastatin, and now there's a super statin called pitavastatin, which shows fantastic results in ovarian cancer. We've got somebody with ovarian cancer listening.

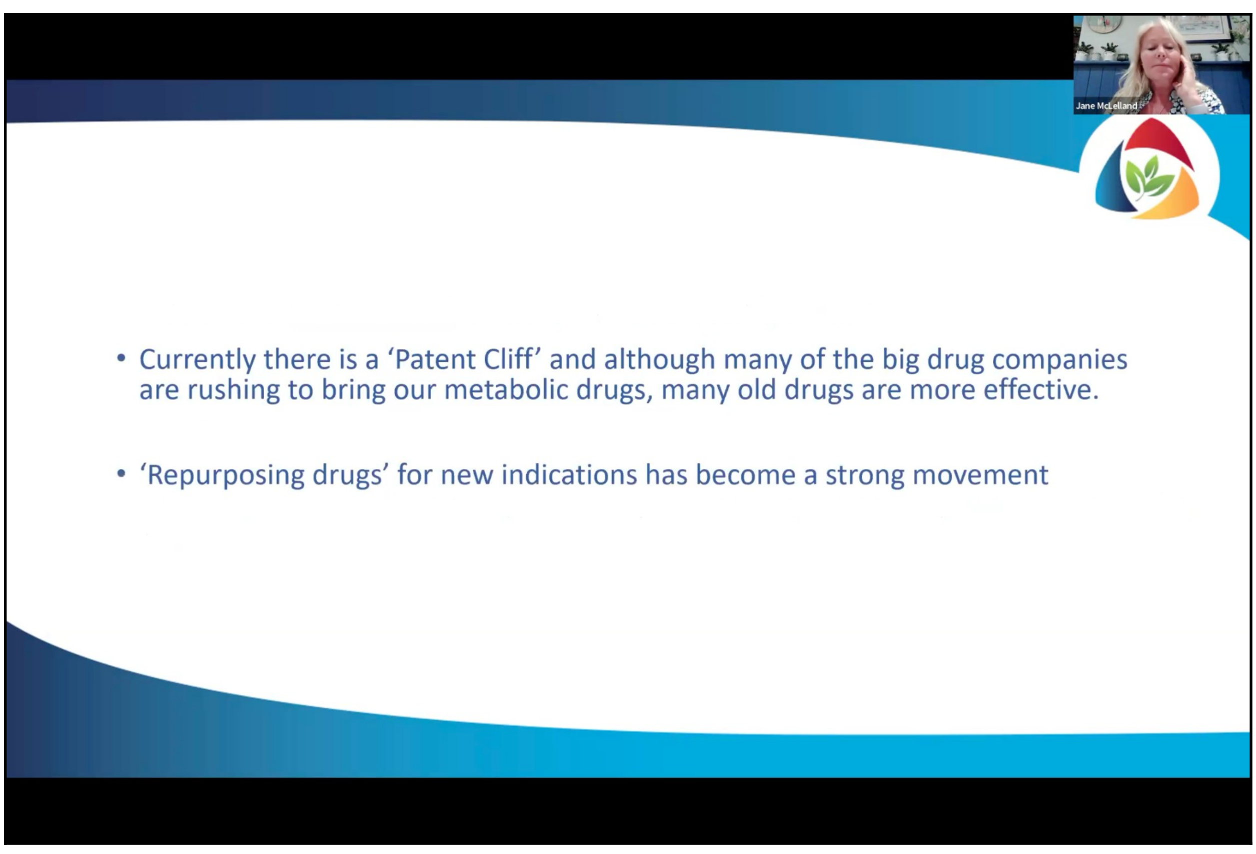
- Are they concerned there are not enough RCTs?

There is strong epidemiological evidence that metformin, statins, doxycycline and mebendazole work.

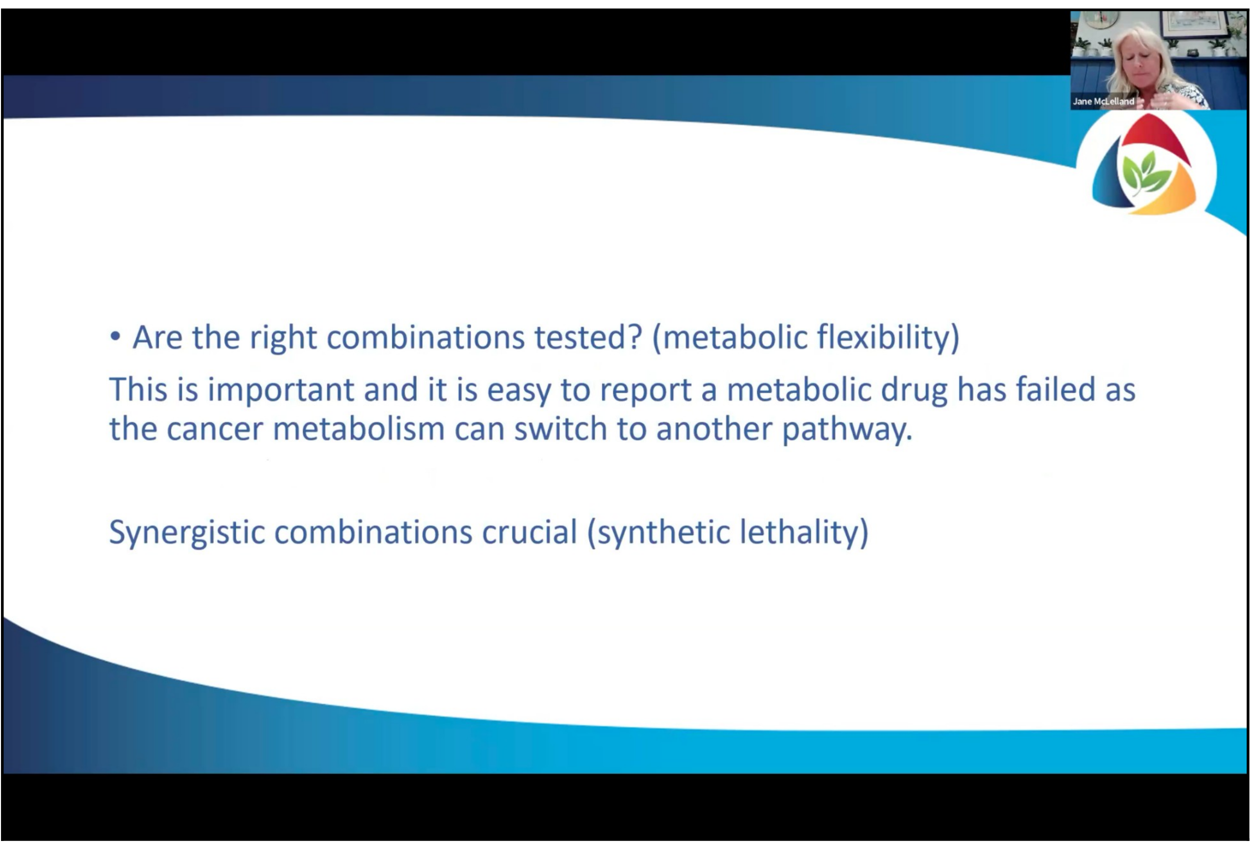
Lack of financial incentive means off label drugs get left on the shelf for RCTs

Doctors are focused on these randomized clinical trials, but just because there isn't a randomized clinical trial doesn't mean the drugs don't work. You need to look at the epidemiological evidence. There is quite a lot. There's enough to substantiate the use of metformin, statins, particularly the COC, the Care Oncology drugs, doxycycline and mebendazol. Of course, there's no financial incentive if you're going to use an off-label drug because they're too cheap, and you don't get your money back on them.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

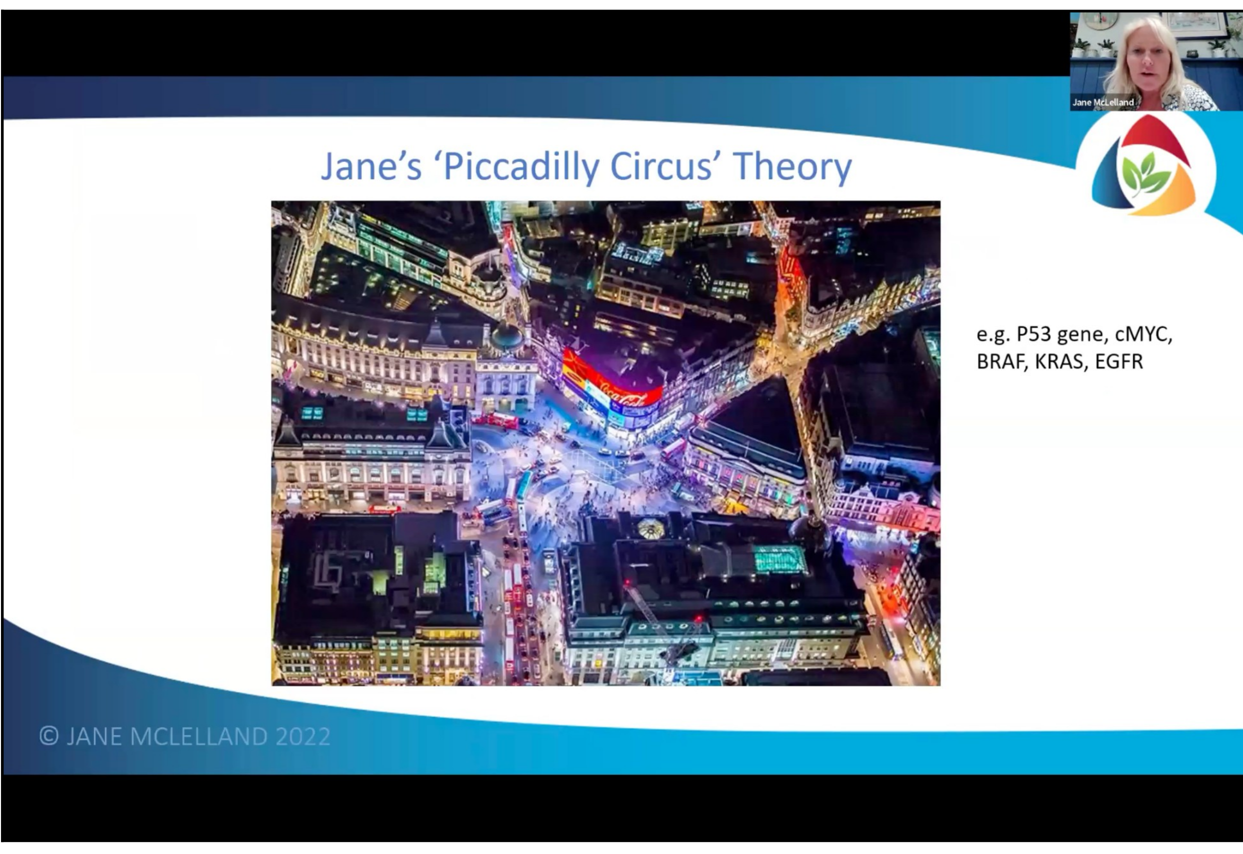


There's also this “patent cliff” with most of the big pharmaceutical companies, so they're getting to the end of their life with their patents. This is why they are trying to develop some metabolic drugs. They haven't really done many so far, but this is why we've now got this repurposing movement, which is fantastic. Hooray.



Combinations: this is what my big thing is. I’m all about combinations and metabolic flexibility. It's really key that people understand this, because if you use a drug or a supplement, it can switch the way that the drug is working, and the cancer will use another pathway. You need synergistic combinations that work together to create what's known as “synthetic lethality”. That would be the ideal.

Jane McLelland
WOO-HOO! I DID IT! I FOUND A CURE!!
THAT'S GREAT, RALPH...NOW WHAT'LL WE DO?
UH... WHAT DO YOU MEAN, FRANK?
WELL, NOW THAT YOU'VE FOUND A CURE, ALL OUR FUNDING WILL BE SENT TO ANOTHER DIVISION
FIND-A-CURE, INC.
WOO-HOO! I MADE A BREAKTHROUGH THAT'LL REQUIRE A LOT MORE RESEARCH!!
MUCH BETTER
©Wiley Ink, inc./Distributed by Universal Uclick via Cartoonstock
• From research labs to the clinic, there are many stumbling blocks...


Jane McLelland
Jane's 'Piccadilly Circus' Theory
e.g. P53 gene, cMYC, BRAF, KRAS, EGFR
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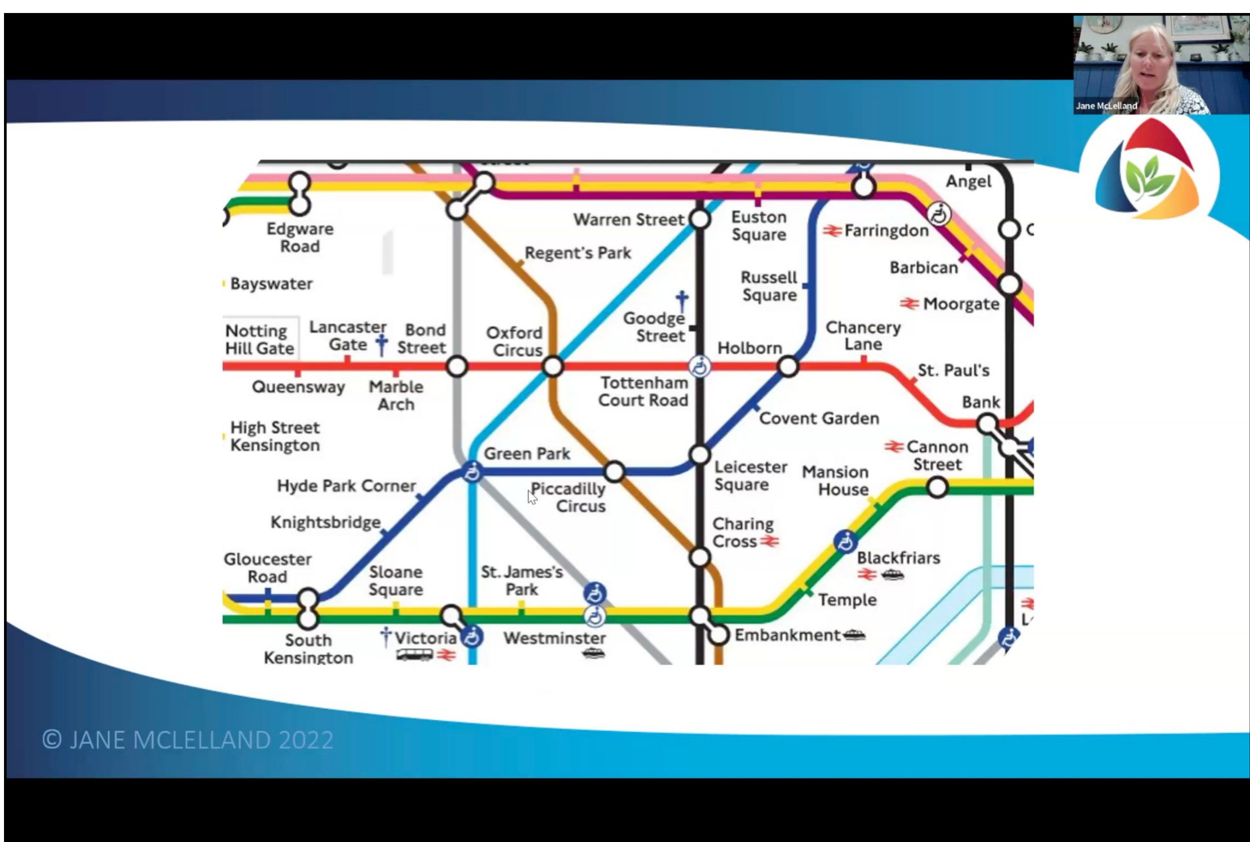
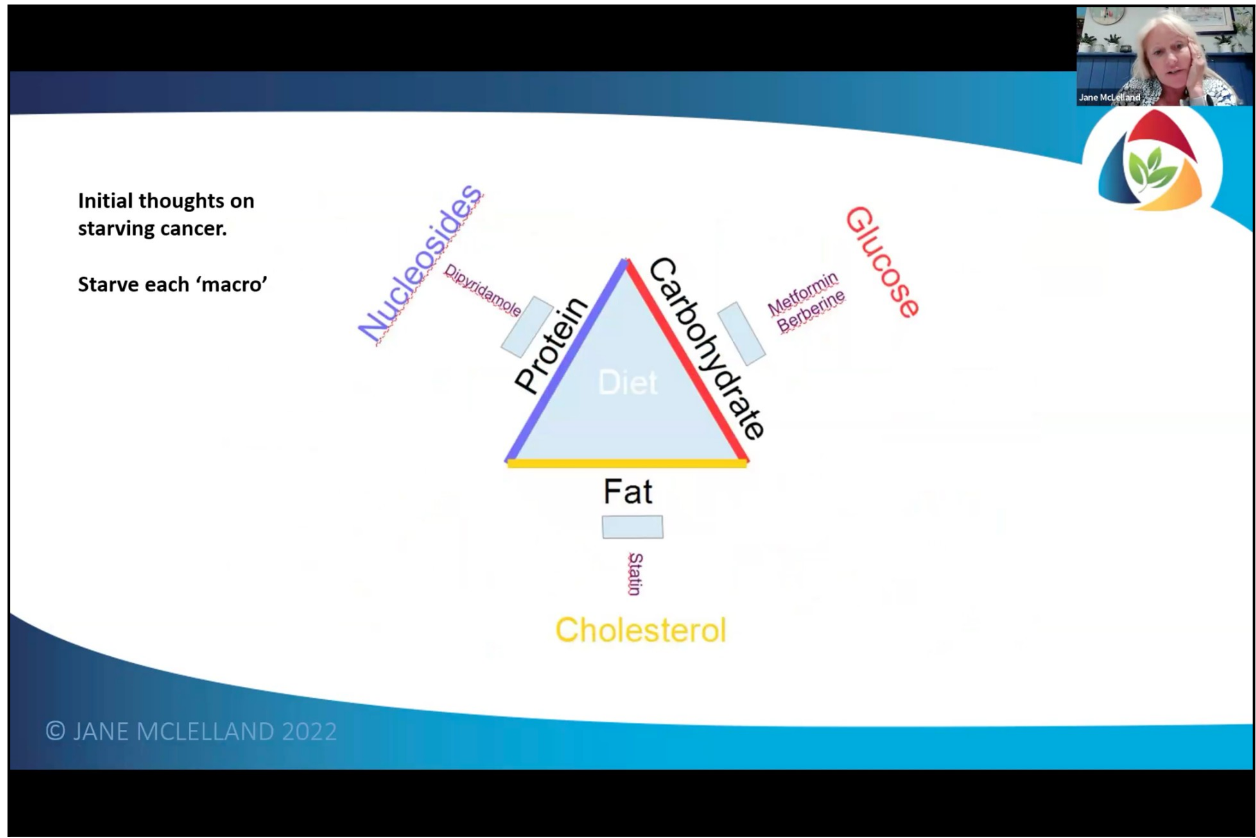
Jane McLelland 23:19
This is my theory, looking down on the cancer cell, on more of the genetic mutations. Cancer is a mix of both genetic and metabolic changes that are happening together. They influence each other. If you look at some of these key gene mutations – these are just a few, a handful of some of the key gene mutations that you get. The P53 gene, for example, if you have the wild type, it's very good at promoting things like ferroptosis, which is cell death. But if you have a mutated P53 it will promote the glycolysis and the glutaminolysis. cMYC as well. That's more the glutaminolysis. The BRAF, the KRAS, EGFR, all very pro the glycolysis. They all have their own specific set of favored pathways inside the cell.

Looking down at Piccadilly Circus – I don't know if anybody else from the UK, but this is a very busy place. If you're looking at these key pathways in the cell, these are the big routes, the P53 the cMYC, the BRAF, can be very big drivers for the cancer.



But what you don't see is underneath. And Piccadilly Circus is here. This is a London Tube app. To explain what I mean about this metabolic flexibility: if you're blocking, say, this route here, which is the Bakerloo route, then the cancer can then just say, “Okay. Well, I can go this long route. I can go all the way around here and eventually get back there.” Or it'll find a shortcut, and then go straight back into Piccadilly Circus using another route.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



This is exactly how the cancer tends to work, and this works with drug resistance as well. If you look at many of the drugs that are used in cancer, common chemotherapies or immunotherapies even, but the targeted drugs, in particular. When you use the targeted drug, you will find there are metabolic pathways that get switched on. If you can work out what those are, and you can look in the literature, you'll find quite a lot of it being reported. You can work out which bits in your map you need to block when you're taking these targeted drugs, so that you know you'll get a much better result. You'll get the drug to work that much longer. This was my initial thoughts way back in about 2012 when I was trying to work out what I'd done.

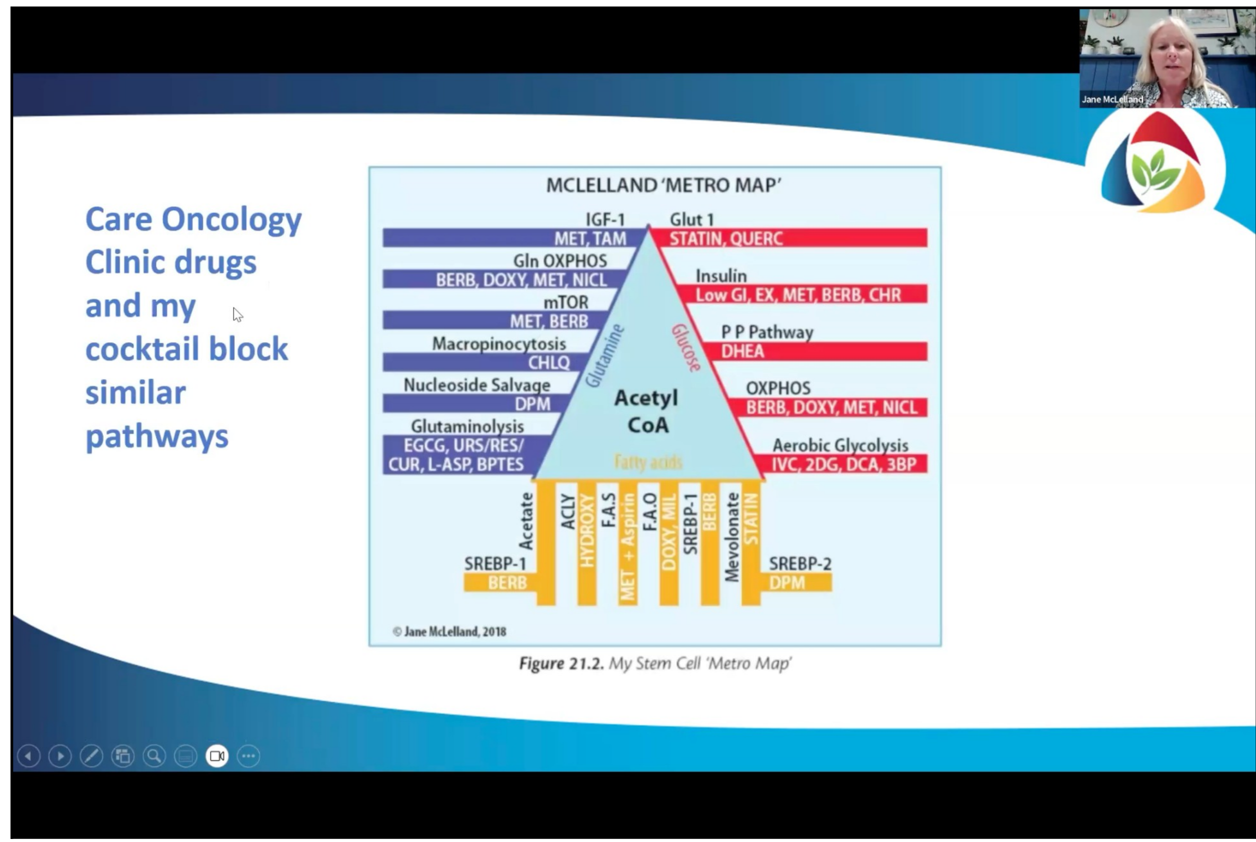
Dipyridamole blocks nucleoside salvage, so it stops the uptake of nucleosides. I didn't know that it also has a big effect on cholesterol as well.

Metformin and berberine both block glucose. They also have many other effects.

Going back a minute, I was blocking fat with a satin.

If you look at this and the Care Oncology drugs that are used – I'm assuming people know who the Care Oncology Clinic are, although, anyway, I won't go into that, but they may not be around for much longer – you've got metformin.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



Up here on the left, it helps to block IGF-1. It also blocks the OXPHOS, which is on both sides, so you've got it there. You've also got metformin where it helps to block mTOR, which is a key protein sensor. It will increase insulin sensitivity, and then down in the fatty acid pathway, it's helping to block that alongside aspirin. Metformin doesn't work on its own there. It has a multiple number of pathways that it'll block. This is very basic. These are the key ones, not comprehensive. This is just a little snapshot, just to give you an idea of the key pathways.

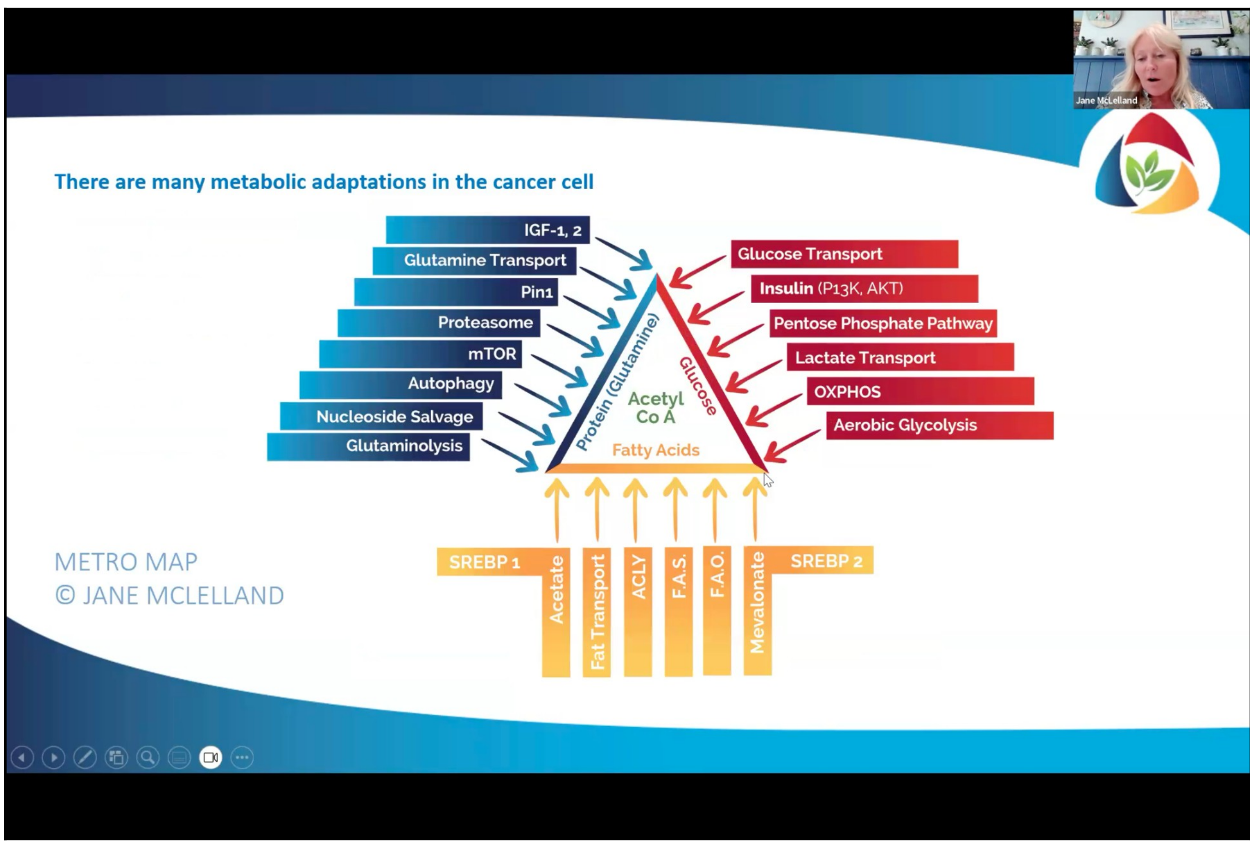
Statins. People are very anti-statin. There's a lot of bad statin press out there about how it's going to reduce your coQ10, and how you'll end up with dementia, etc. Now, coQ10 is a very powerful antioxidant, and if you're having chemo treatments, you don't want coQ10 in your system, particularly if you're trying to simulate ferroptosis. You want to reduce the coQ10 in your system. This is why it's important to look at the settings in the cocktail. If you're going to use metformin, metformin blocks something in the normal Krebs cycle, the OXPHOS cycle, it blocks something called the complex one in the electron transport chain. If it does that, it creates a reverse shunt of glutamine. That reverse shunt then pump out citrate, which then gets converted to cholesterol, and then that's where you need the combination of a statin and metformin. I'm sure Greg, who started up the Care Oncology Clinic, thought about this long and hard, but the synergistic effect of blocking those two pathways was really good. I took the satin and dipyridamole here. I call this my cholesterol corner. These are two key cholesterol pathways. Every cancer cell will have cholesterol knobs on its surface. Is constantly trying to churn out cholesterol to create these new membranes, and I blocked both with the dipyridamole and the methionine. I didn't really fully understand what I was doing back then. I was just had my L

(learner) plates on, but I picked a good duo there. If you look at nucleoside salvage with the dipyridamole, I was stopping the cancer from pulling in little chunks of DNA. But I was also blocking this other pathway, that pentose phosphate pathway, which produces DNA. That's one of the things it does. It also produces glutathione, which is a key antioxidant, but I was using DHEA. If you don't block both of them, then it'll just switch from one to the other.

mTOR and autophagy are two key pathways that feed off each other.

Macropinocytosis: I know somebody has pancreatic cancer. This is a key pathway that pancreatic cancer uses. You need to look into that.

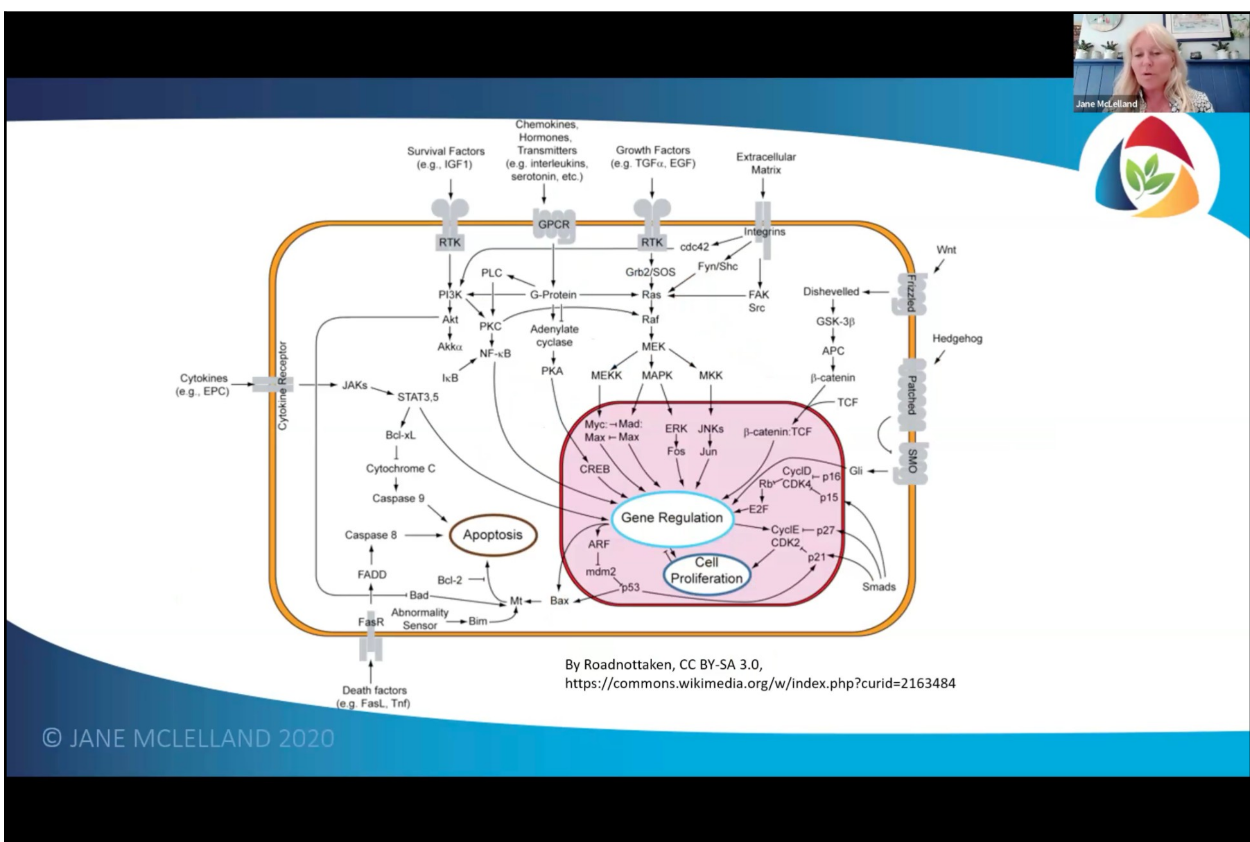
Hydroxychloroquine is something that Steve took a very high level of for about three to six months, gradually reducing it a little bit. He also took metformin, statins, aspirin. He did the full cocktail of things as well, not through my research. He did it himself, but he also had it alongside immunotherapy. This isn't alternative care, it's complementary, and he's still cancer-free. I'll try and remember his surname, so I can put you in touch.



This is my second edition Metro Map again. Glucose will normally go into the muscles, through glute four. But in cancer, it goes through glute one, or mostly group one, but sometimes two and three. It will also use glute five. Prostate cancer, for example, will use a lot of glute five. It likes the fruit, but it doesn't like it. When you look at PET scans, they don't use PET scans in a classic way, normally, for prostate cancer – they will use choline as a tracer, because it's looking at the

fat uptake, because fat is critically important for prostate cancer. These pathways down here, and in fact, statins and dipyridamole, there's good research on that combination working really well for prostate cancer. There are some other things like, I haven't got it well, the fab p5 which is the fatty acid binding protein five, that is really important as well. Berberine will help block the acetate as well. Berberine may be better than metformin for prostate cancer, but that's not to say I wouldn't suggest that you avoid metformin.

That's a general overview of the map. Glutamine is for prostate cancer. The proteins are actually quite important too. You've probably seen that solic acid, curcumin and resveratrol are really good at helping to block the uptake of glutamine with prostate cancer.



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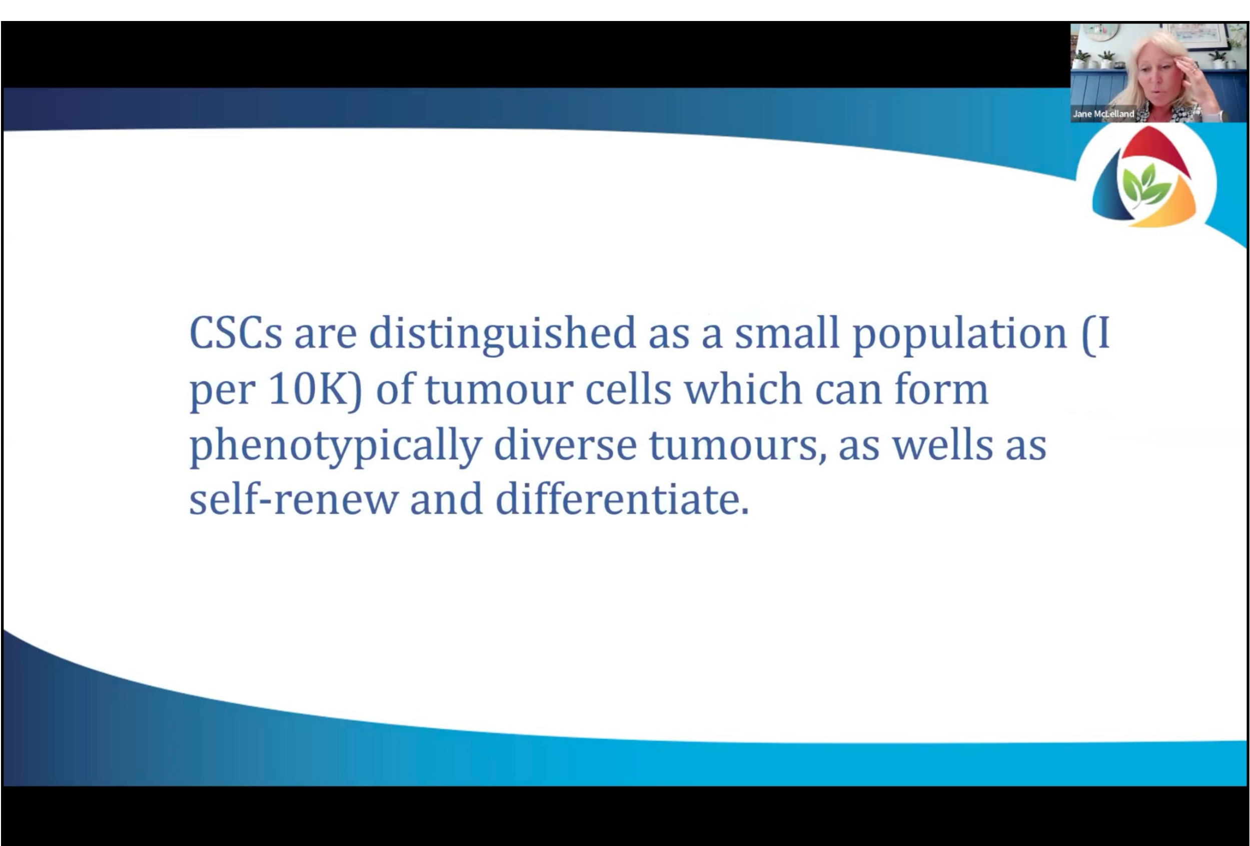
Jane McLelland 33:10
This is what doctors are taught. I'm not going to go through this much, so don't panic. But you can't see any glycolysis in there. There's no mention of autophagy, or the Krebs cycle, nothing. It completely ignores metabolism. This is where we're going wrong. Doctors have no clue about that part of what's going on with the cancer cells. It's got Wnt, and it's got Hedgehog there. I don't quite know where the Notch is on this. They've forgotten that. But there are these pathways. You've got Step3 here, and you've got nuclear factor kappa beta here. But otherwise, I just feel it's just missing some of the key metabolic pathways for doctors to learn. They just don't know it.

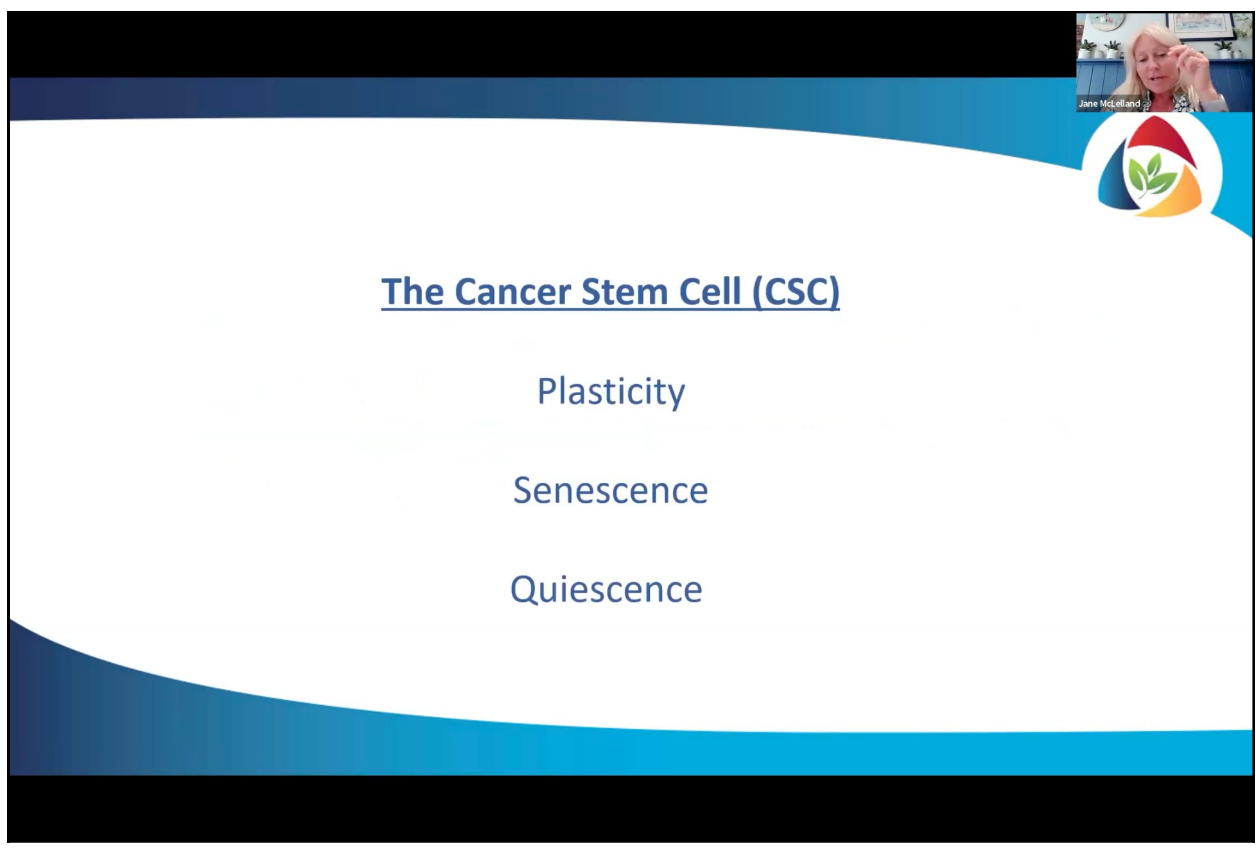
“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

We're just going to talk a little bit about the cancer stem cell, which is a slower dividing part of the cell. If you're going through the progression of cancer, it is where my stem cell is the progression between the different metabolism to going on to the growth factors. It's between the tumor environment stimulates this sudden increase in activity in the cell with the mitochondria, and then once you've actually switched off the immune system, that's the fast dividing cells there, but everything before that is really the cancer stem cell.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

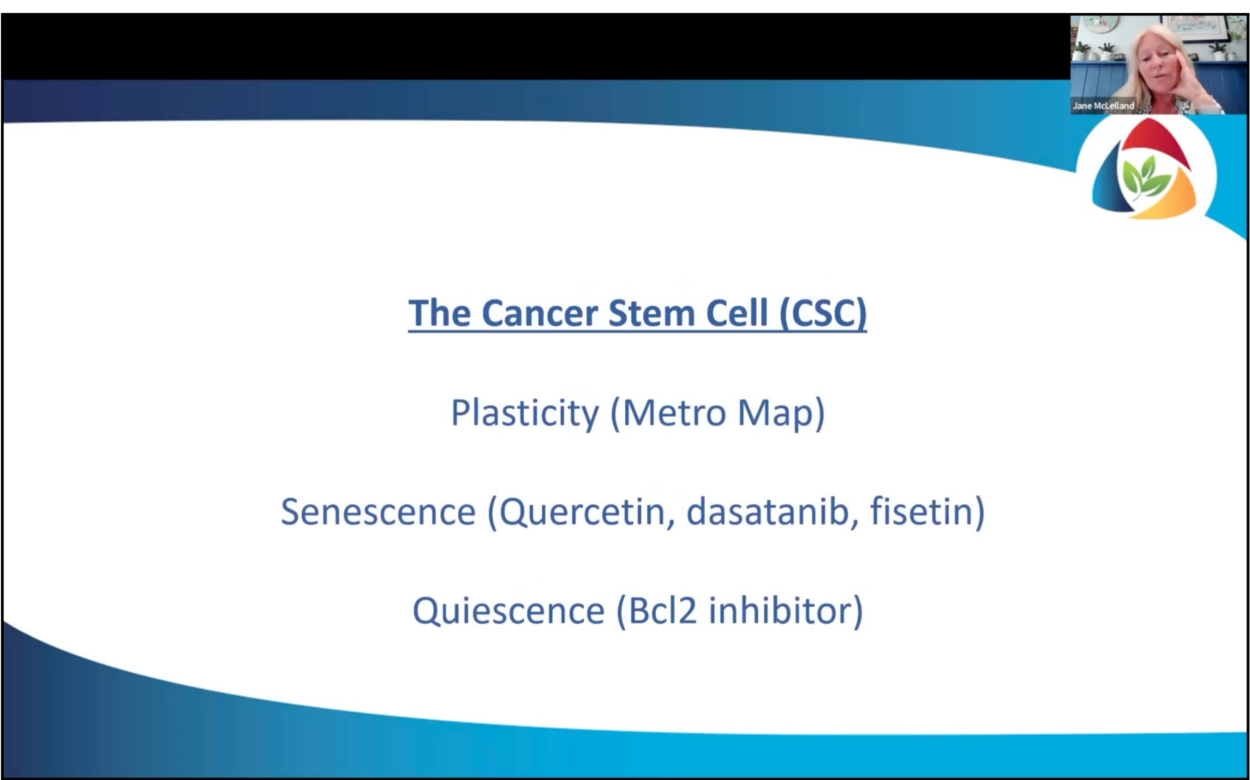


They are a smaller population, so you don't get so many of them. You only get one per 10,000 in the tumor, and they are phenotypically diverse. Otherwise they form all of these different types of tumors. They prefer different fuels, different metabolism.



These are the three key features of stem cells.

Plasticity is the ability to switch and change. Senescence, which is the old cells which just hang around and secrete something called “SASPs”, which stimulate the cancer cell to progress. Then you've got quiescence.



How do you deal with those things?

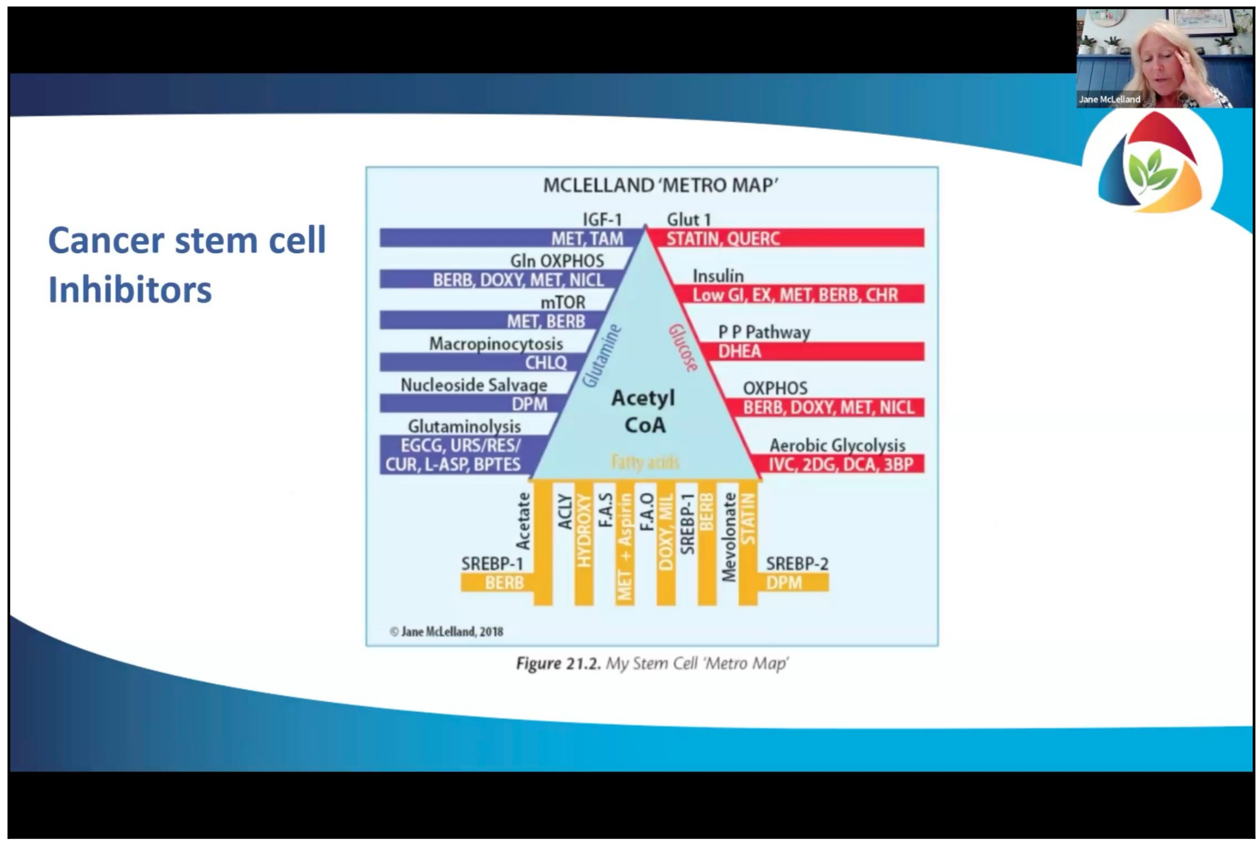
The Metro Map deals with that plasticity.

Senescence. You've got quercetin, dasatanib, and fisetin.

Bcl2 inhibitors will help with the quiescence.

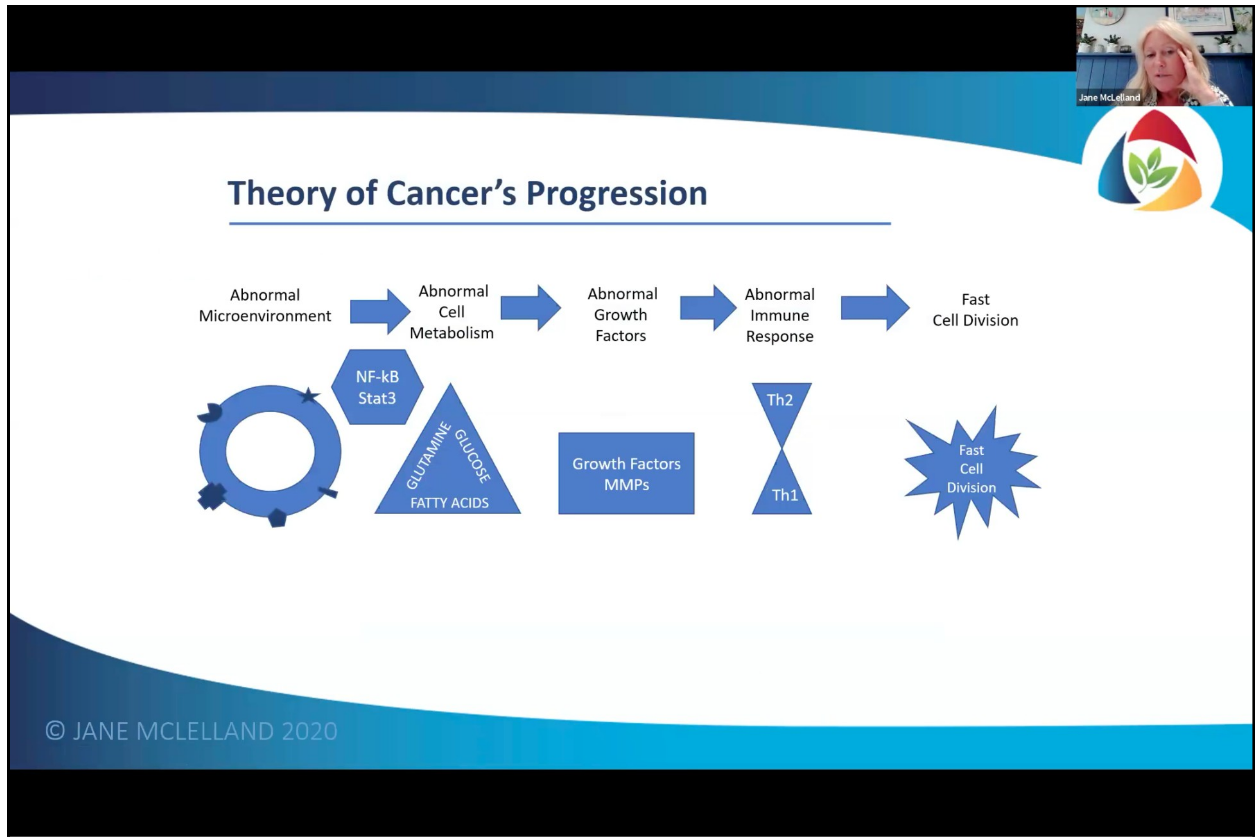
You probably need to have all of those things in your cocktail. You need to have some key inhibitors of the Metro Map. You probably want to have quercetin, which is great anyway, for blocking that glute one and fisetin, which is kind of a natural PARP inhibitor, a brilliant one, and then there are quite a few natural supplements that will inhibit the Bcl2 as well.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



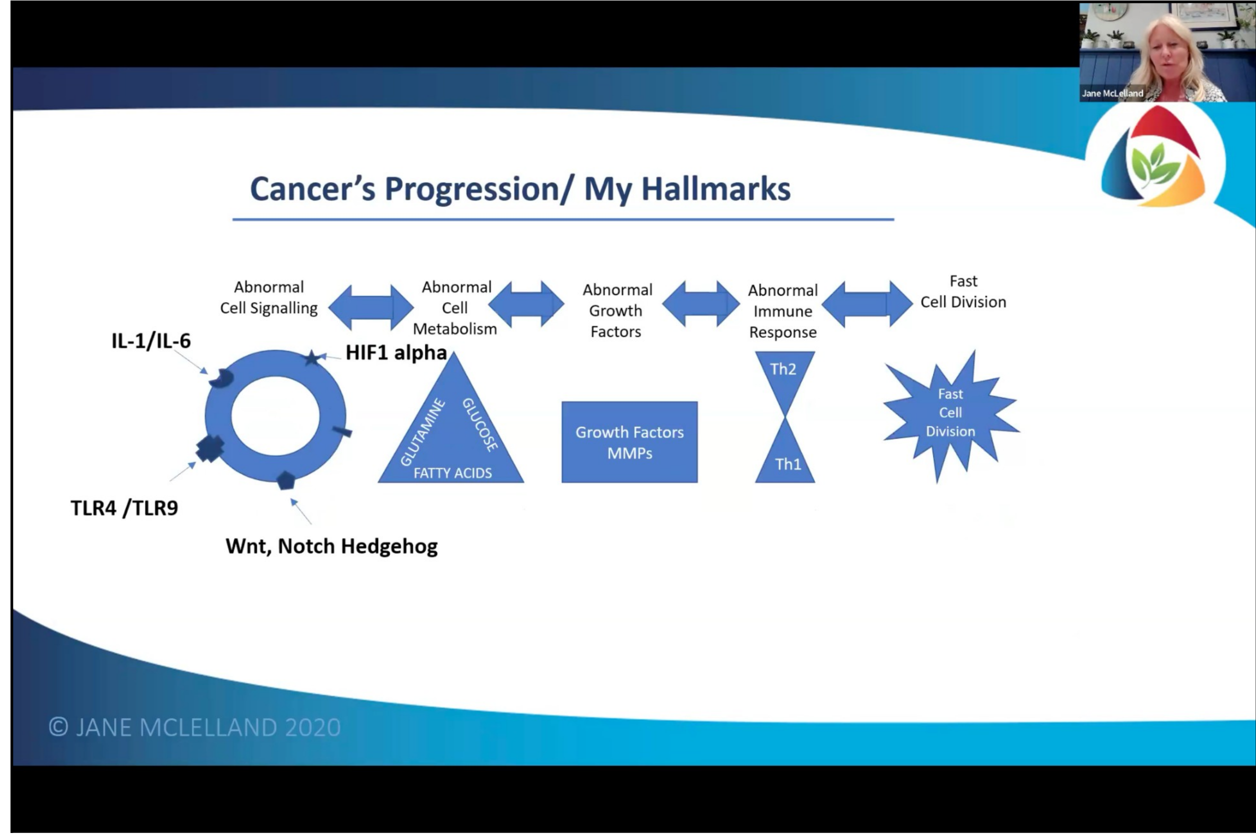
Going back to this, that's the basic chart. You've probably got a fair idea of these things now.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

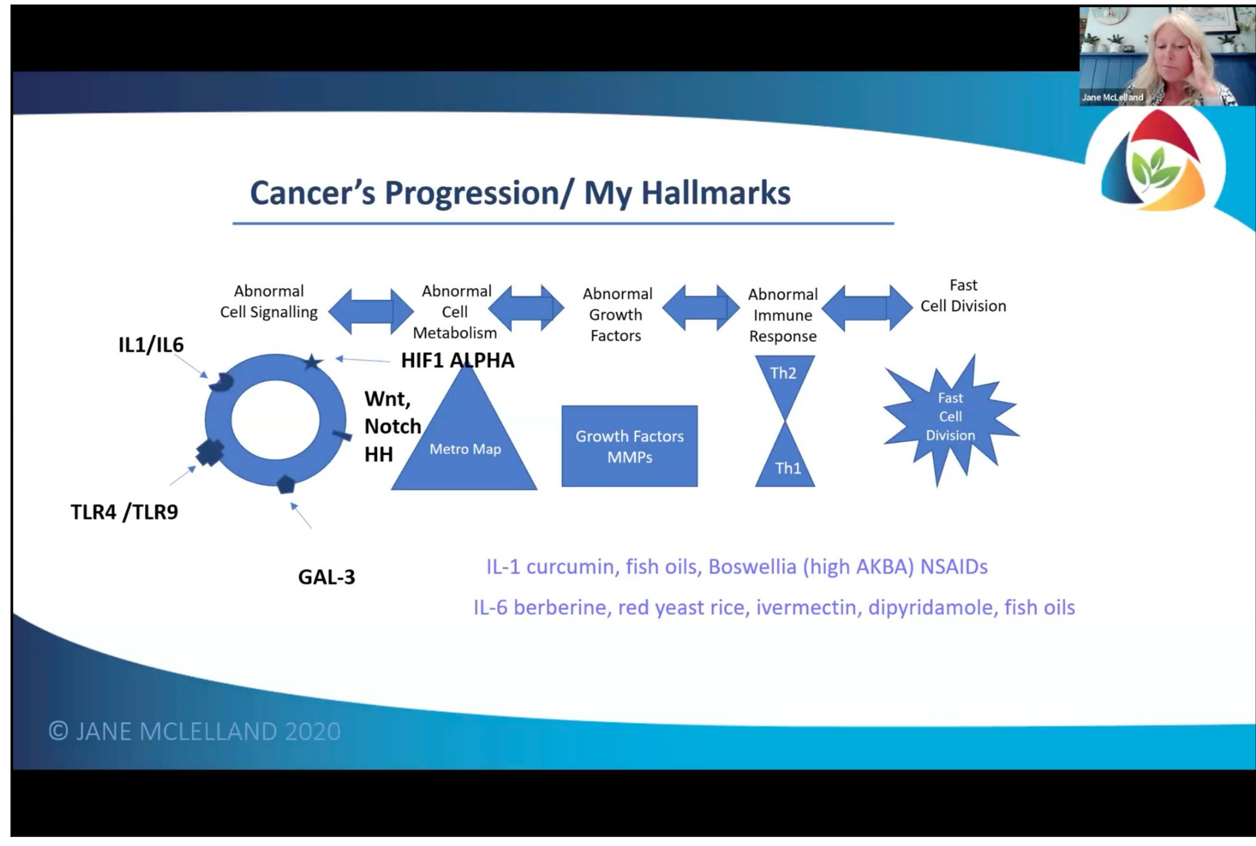


If we look at progression, at the moment we're targeting the fast cell division with chemo and radiotherapy. That's the start of how we used to treat cancer. Then we progressed to trying to hit the growth factors, the VEGF, the vascular endothelial growth factor. Then we moved on. We're now into immunotherapy. It's like a big thing. The next phase, we'll be looking at these RNA vaccines. These will help target some of the epigenetics that's going into the cell. This could be very hopeful. As long as we don't have any spike proteins in there. We don't know what the results of these RNA vaccines will be.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



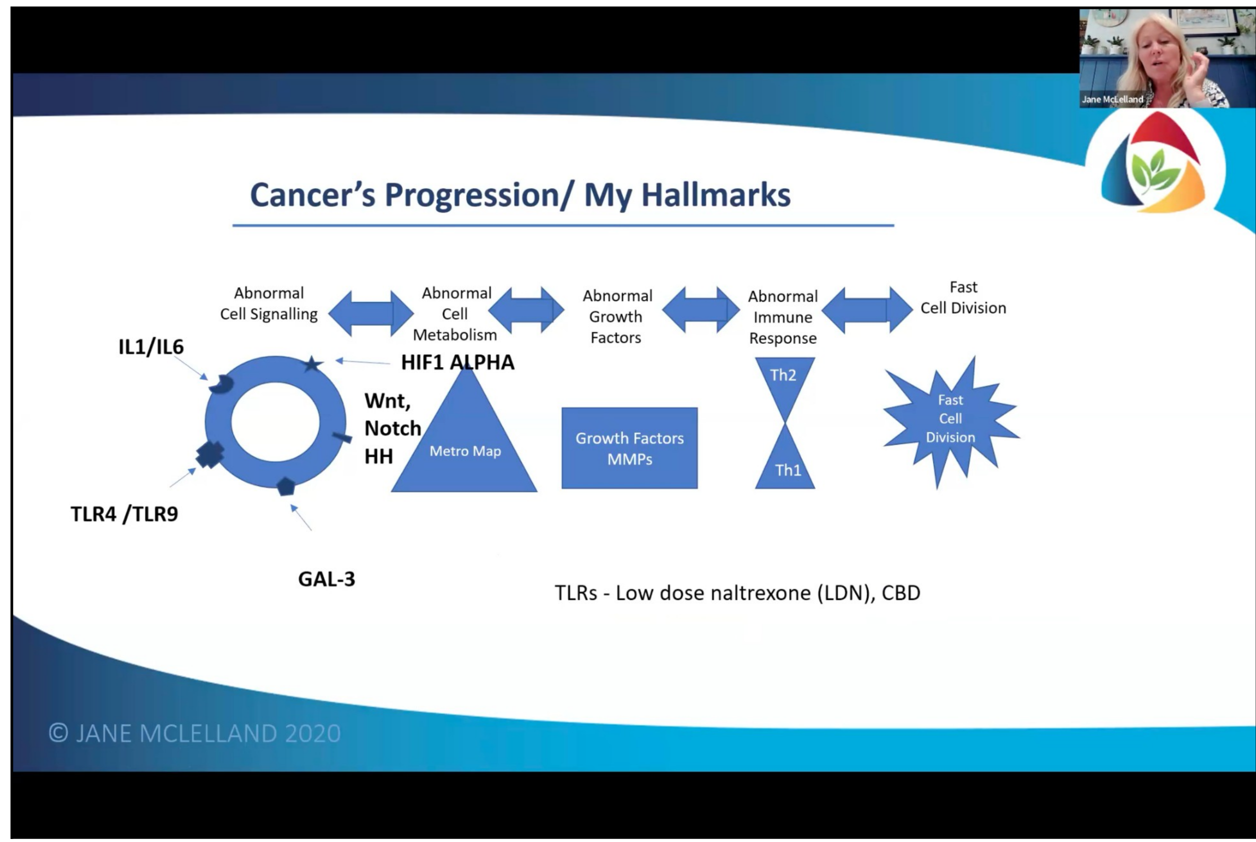
If you look at some of these factors in the tumor microenvironment, you've got this Wnt, Notch, and Hedgehog going on the surface, then you've got these toll-like receptors. These are pathogen receptors. Pathogens are huge. We know that inflammation, IL-1 and IL-6, are inflammatory markers, and the biggest inflammatory cause is really infection, but you've got other things causing it as well, carcinogens. You've got obesity. All of these things will cause low grade inflammation. You've also got HIF1 alpha, which is hypoxia inducible factors, so this will drive cancer as well as a lack of oxygen in the area.



If you're going to target interleukin-1, these are good things that come in fish oils, Boswellia with high AKBA, and non steroidals.

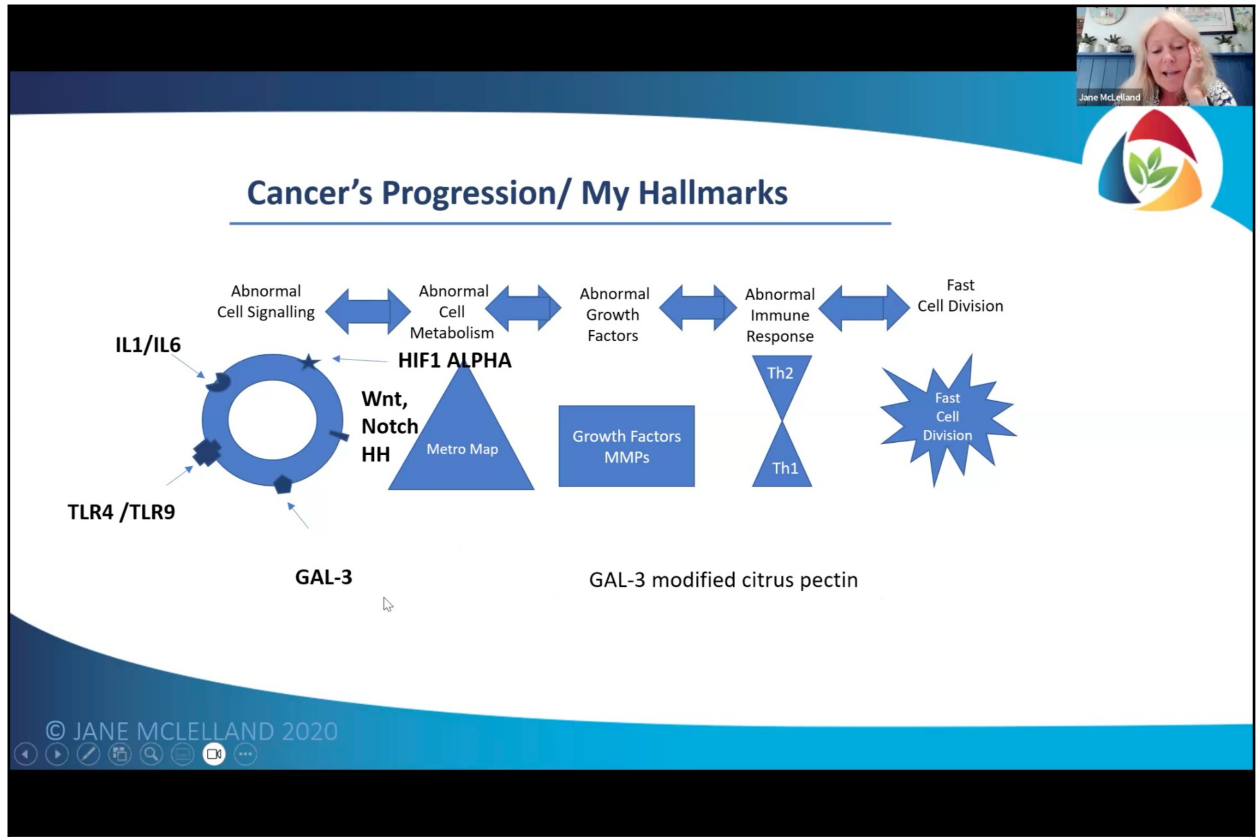
For interleukin-6, berberine and red yeast rice together work really well. Ivermectin, dipyridamole, and fish oils are very useful. There is a lot of hooha over ivermectin, but it is a very useful drug for cancer, and it will help in the Metro Map as well. It helps box some of those pathways.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



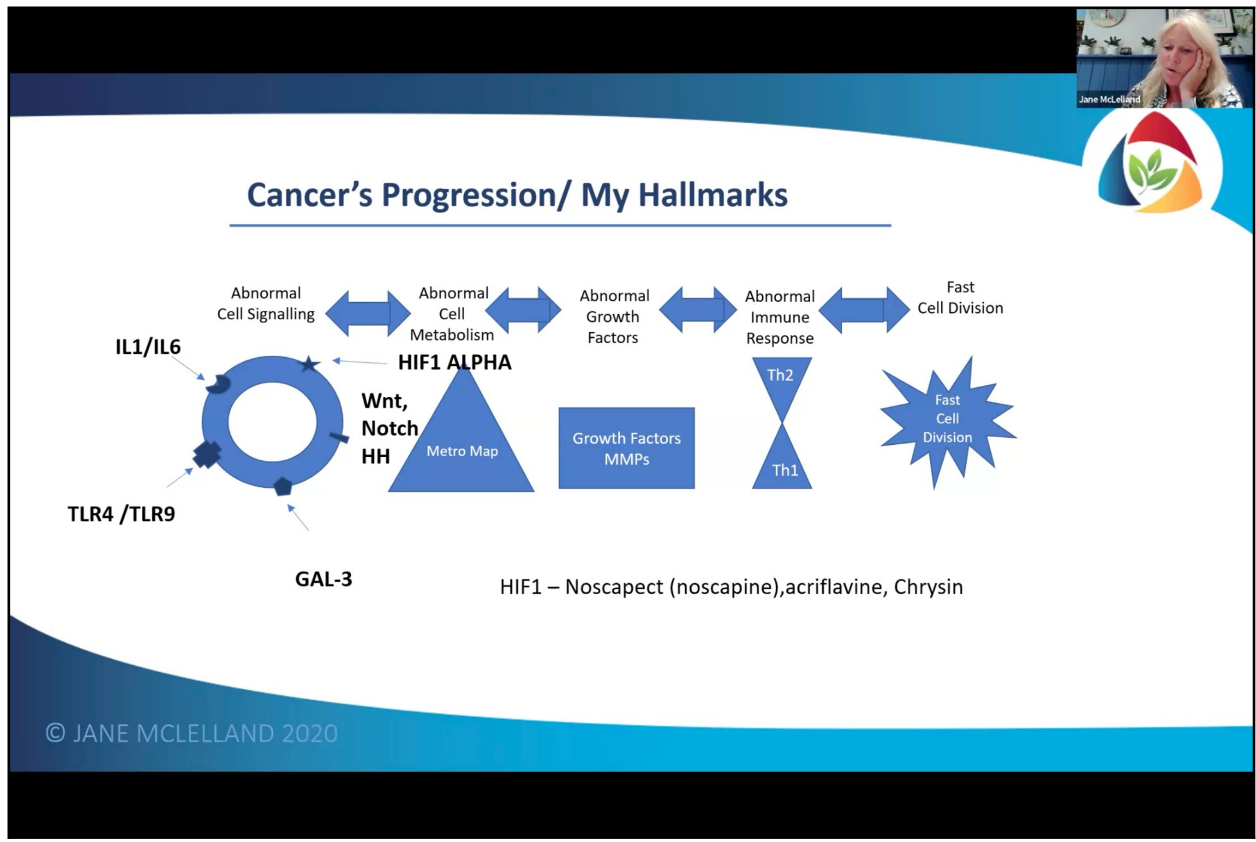
Those toll-like receptors: low dose naltrexone helps on those, and CBD.
The pancreatic person: You've probably seen the research on low dose naltrexone and which they use. I can't remember if it was a combination of LDN and another drug together. But again, I think CBD can be very useful for many cancers because of its effects.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



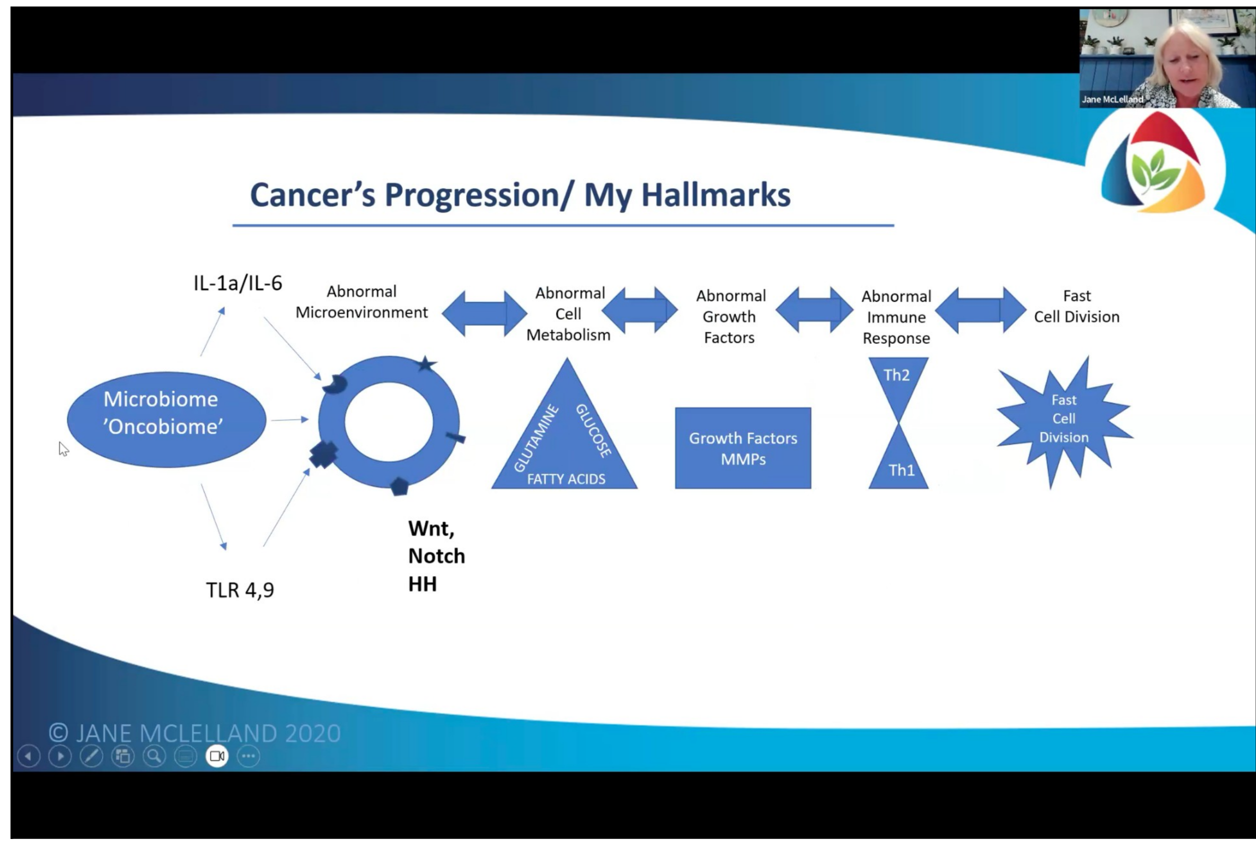
You've also got galactin-3, which you probably know, as we have lots of prostate cancer people here. This is a very upregulated feature. This is a type of pectin, modified citrus pectin. I don't know whether you've talked about that before in this group, but it has very big effects on prostate cancer. Galectins can also affect other cancers as well. You can look that up and see whether it's affected you. Galectin-1 for cervical cancer, and I'm not sure whether the modified citrus pectin works on that, but I did take it anyway back in the day.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

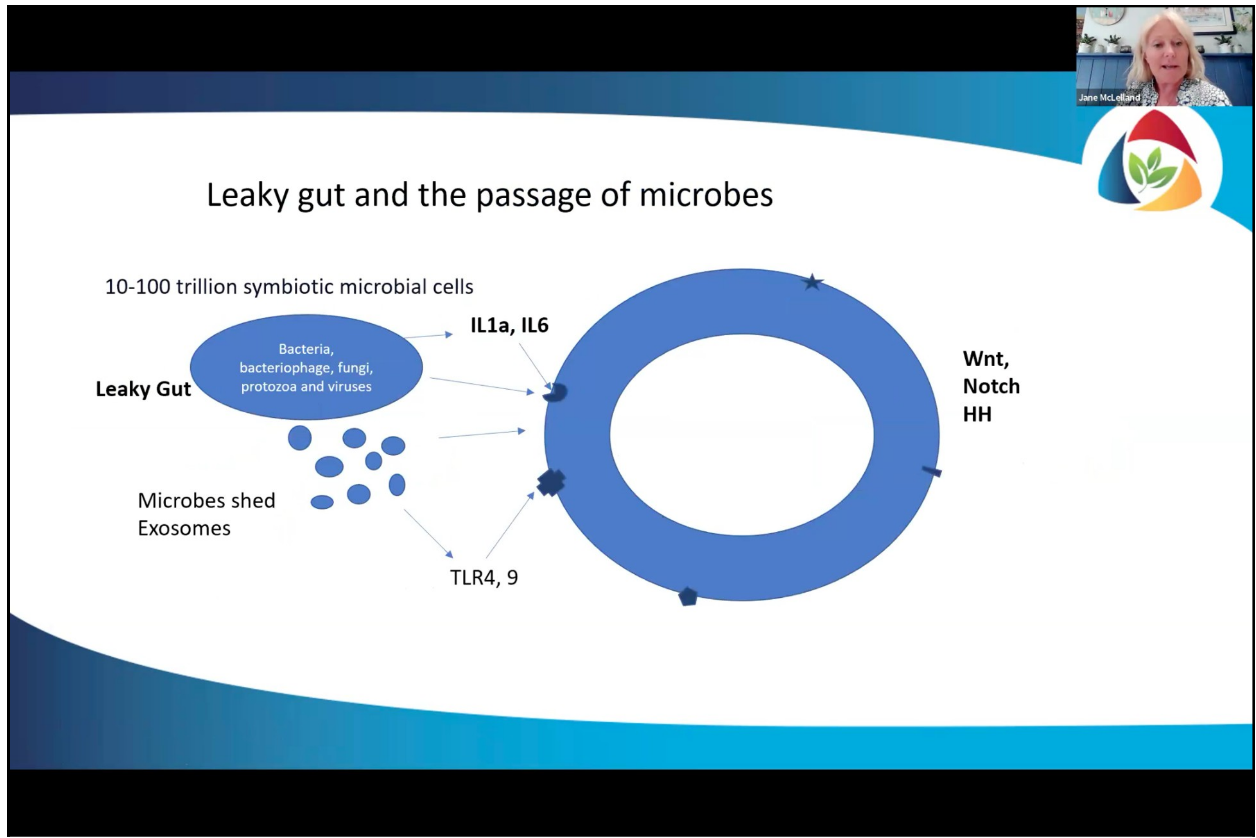


HIF1 Alpha to try and block that factor. We've got noscopine, which is a drug that is a cough medicine. It's used for kids in Holland, but in fact, it's from the opioid poppy. No psychoactive effects with it, though, and that is really useful. But it also works a little bit like mebendazole in terms of blocking the microtubules in the cell. It's a more gentle sort of microtubule disruptor, a bit like vincristine or Vinblastine. It does almost work a little bit like a chemo as well. Acriflavine. To get it orally is quite hard, but if you want to look up acriflavine for cancer, there's some tremendous stuff on it, but we need to have it orally. You can get in Mexico. Chrysin, which is from honey, is very good at helping to block hormonal problems, as well as the HIF1 alpha.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



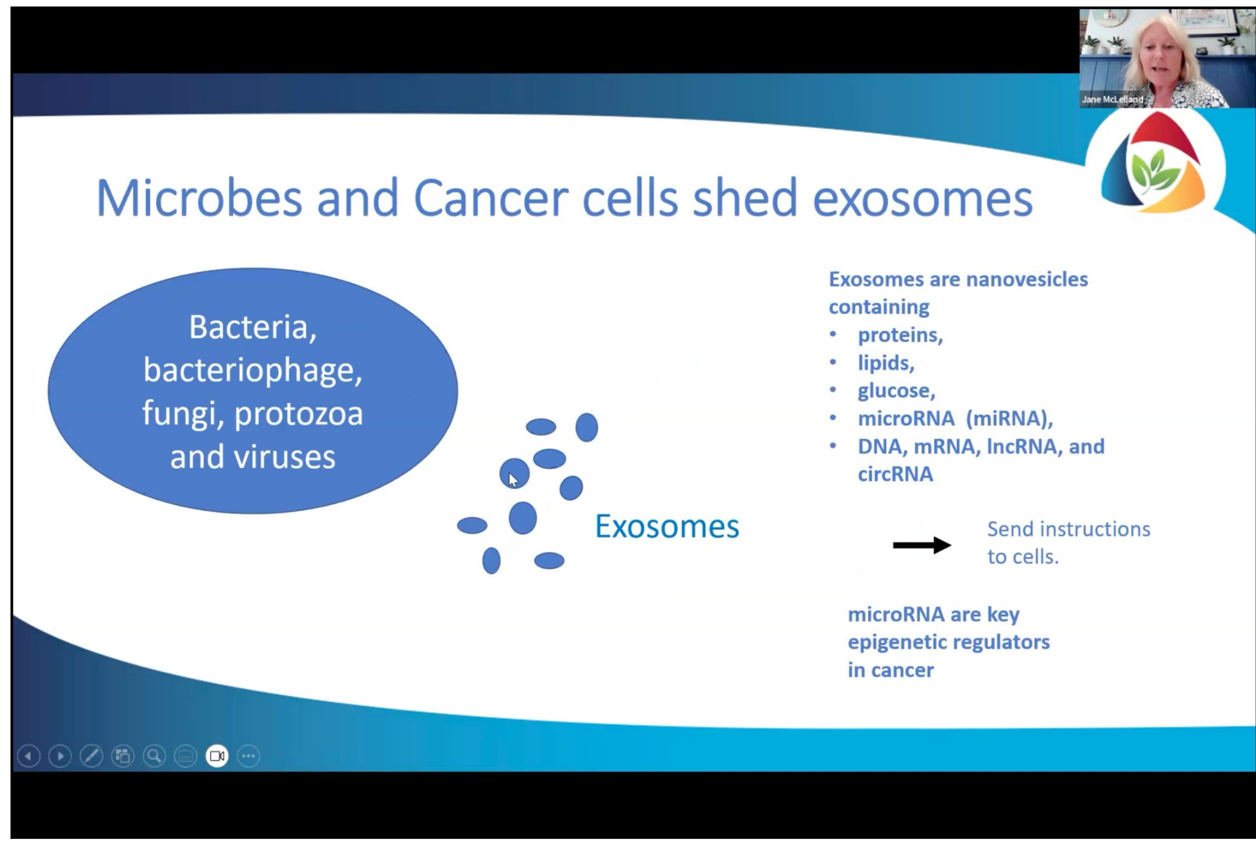
If we go backwards a little bit to what happens with the microbiome: This is an emerging Hallmark, and this is my theory of what happens. It stimulates these toll-like receptors, because you get a leaky gut if you, and most people with cancer at some point will have had gut issues, and you get a leakage of some of these pathogens into the system, so they cause inflammation, and they simulate these toll-like receptors. I suspect they have an influence on Wnt, Notch, and Hedgehog as well.



This is the leaky gut with all of these pathogens.

Then you have something called an “exosome” shed from all of these pathogens. Cancer cells shed these exosomes as well, tiny little nano vesicles which communicate with other cancer cells. The bacteria communicate with other bacteria. You get this big network of communication going on in the system.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



These exosomes contain a lot of these RNA particles, as well as protein, lipid, and glucose. This is where the epigenetics happens. The knowledge from what's happening in the tumor microenvironment going into the cell. I am hopeful that the RNA or the RNA vaccines will do something. Let's wait and see.

To block exosomes, you've got CBD. And another really useful drug for cancer, is amiloride, because this also affects the extracellular pH as well.

Antimicrobials with anti-cancer action:

- Doxycycline (stop for immunotherapy)
- Clofazimine
- Nitazoxanide
- Niclosamide
- Imiquimod

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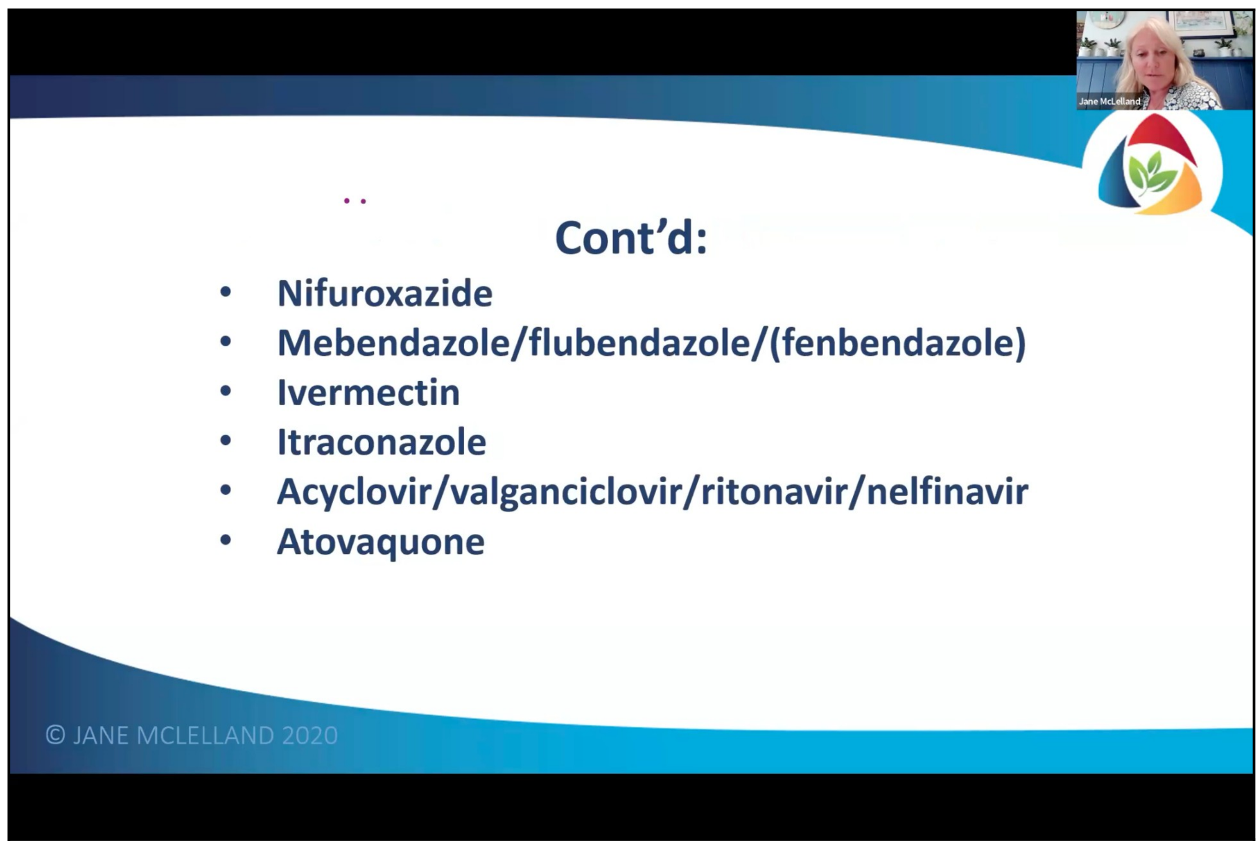
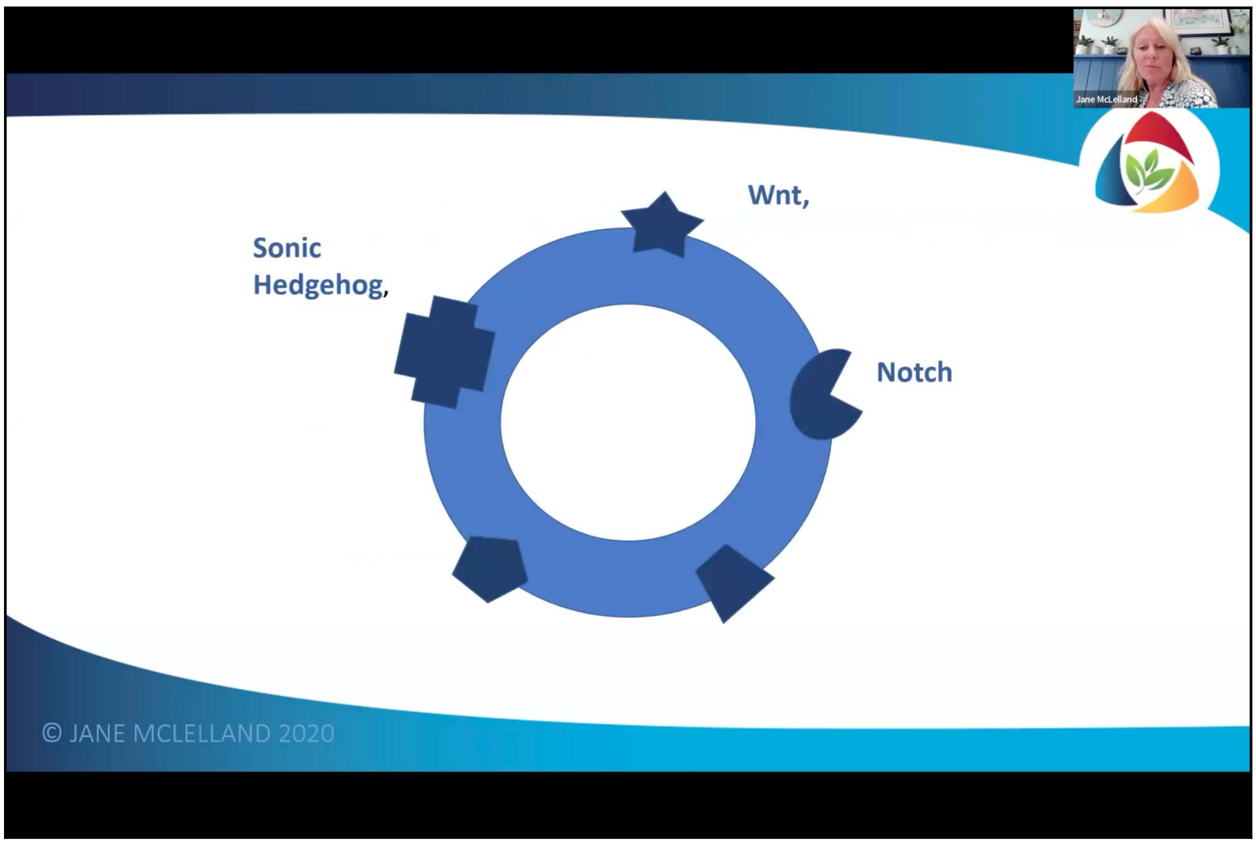
Jane McLelland 43:12
If you look at the antimicrobials with anti-cancer action, you've got a lot from doxycycline, clofazimine, which is used for leprosy, really useful for triple negative breast cancer. In fact, quite a lot of cancers. Nitazoxanide, which helps with chauctosis. Niclosamide is big. It's big, big, big. The two big anti-cancer drugs are niclosamide and mybendazole. A lot of people use fenbendazole, but niclosamide, because it works on the cancer cell membrane, and it helps block many things. It blocks protein uptake, and it helps promote ferroptosis. It has a huge effect, but unfortunately, it needs to get into the system better. That's something I'm working on with a professor in America.



You've got some more of these anti-parasitics as well, which are terrific.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



If you look, we're going back to looking at Sonic Hedgehog, Wnt, Notch on the surface of the cell.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

Jane McLelland

- Doxycycline (stop for immunotherapy)
- Nitazoxanide (Wnt, RANKL)
- Imiquimod (HH)
- Niclosamide (Notch, Wnt, Stat3)
- Nifuroxazide (Jak2)
- Mebendazole/flubendazole/fenbendazole (Hedgehog)
- Ivermectin (Wnt)
- Itraconazole (Wnt/Hedgehog)

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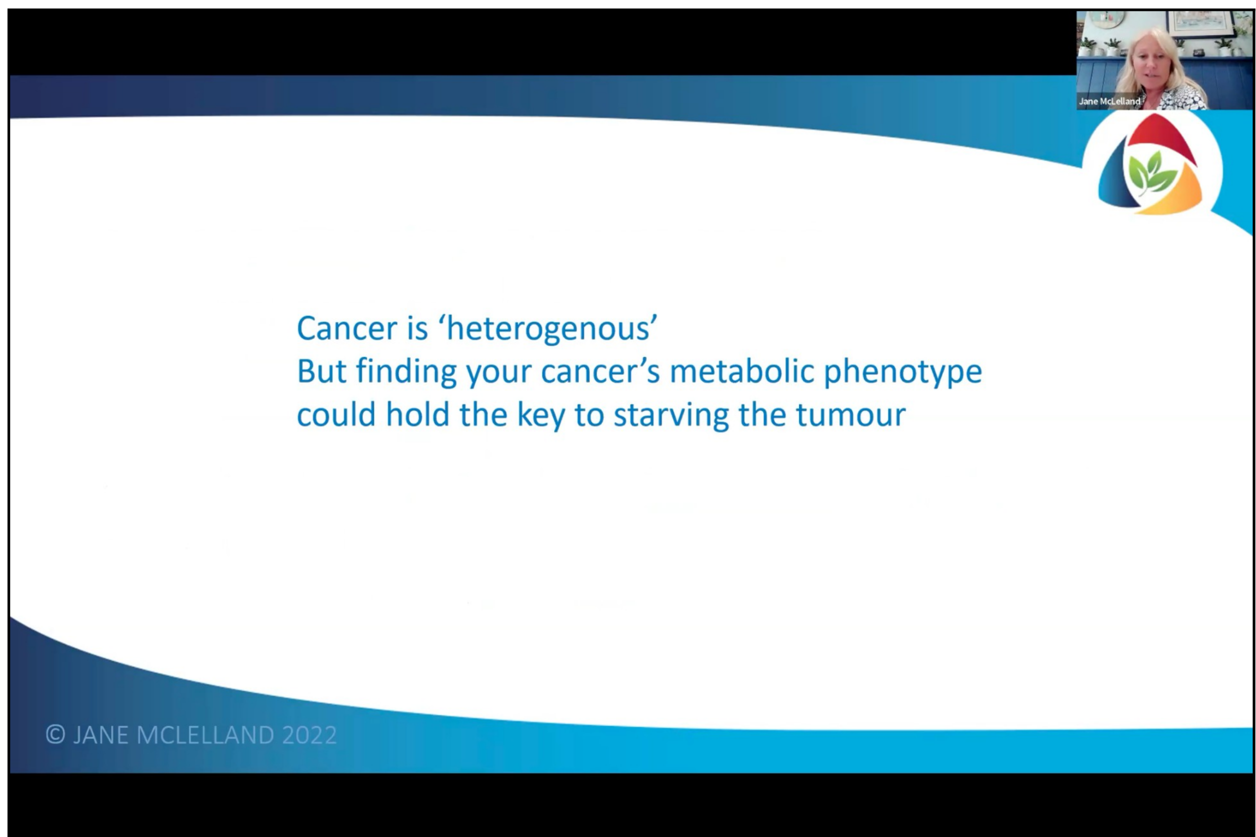
These things were actually blocked. The undruggable targets of Wnt, Notch, and Hedgehog are not undruggable. Nitroxanide helps block Wnt. Imiquimod the Hedgehog. Niclosamide will block Notch, Wnt, and Stat3. Nifuroxazide, Jak2. All of these will block the Hedgehog. Ivermectin will block the Wnt. Itraconazole, the Wnt and the Hedgehog. So a lot of different targets from these antimicrobials on the surface of the cell. It will be really big.

You want to do is work upstream, because that's the way we're going to affect cancer. That's why I'm hopeful that these RNA vaccines will do well, because they are more upstream than a lot of these chemos, etc. Hopefully one day we will phase out chemo or maybe just reduce it. That's what I'd like. It's not to say it's completely redundant, but I do think we overuse it.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]



Cancer is heterogeneous, so you need to find your metabolic phenotype. You need to do a bit of research, having a look to find out which pathways are most upregulated with your cancer.

You can use Perplexity. You can use Consensus. You can use ChatGPT. There are many different easy ways these days to look at the phenotype of your cancer, because different cancers use these different pathways. Maybe glycolytic, cholesterol loving, etc.

Brad Power 46:24
One theme from the chat, and this is always an issue when you get into complementary therapies, and you're talking about molecular pathways – the question is, is there evidence? And of course, the gold standard comes back to randomized clinical trials, and as has been pointed out, sometimes it's evidence-informed, not evidence-based.

There are two questions.
One is, how do you come up with the pathways that you focus on in your model?
And then the second question: what evidence supports the recommendations you're making?

Jane McLelland 47:24
There is epidemiological evidence which goes back to looking at people. If you look at metformin for diabetics, etc., there was a study done in 2013 looking at 15 million diabetics who were taking metformin. I don't know whether it looked at non-diabetics as well, but certainly there was a significant decrease in the number of people who were getting cancer. On statins,

they've looked at it in Finland, but it's looking at the combinations which is key. Because if you're not looking at the right combinations, we’re lacking clinical trials to know whether these things should work or not. So in GBM, for example, Care Oncology did a study looking at their four drugs, mebendazole, the Doxy, the Metformin, and statin together, and they showed that they doubled survival time. They were also looking at triple negative breast cancer. They're looking at a whole range of different cancers across the board. They couldn't publish all the results, but they were seeing a doubling of survival time.

Having said that, I know anecdotally, which is just a terrible evidence base, but you do have a lot of positive progress reports, and they're not always reported in my Facebook group.

I have a very active Facebook group, which everybody's welcome to join if they want. It's called “Jane McLelland off-label drugs for cancer”. There are about 93,000 or 94,000 people there. They will often report their positive progress and then write down what they've used. It's unfortunate that the N-of-one is where we are right now. But if you are stage IV, and you know that they haven't got the answer, what are you going to do? Are you just going to sit there and die, or are you going to have a go and try and look at different ways to actually save your life? This is the issue that we have. Doctors hate it because they want everything to be for the long term good. They're thinking that everybody should be a guinea pig and sacrifice themself for the future good. We need to look at trials in a different way. We should look at trials at looking at better cocktails, and then once we get the cocktail, work out, what we need to cut out. We need more and not less. What they do is they trial one drug. This is not the way to do it. We need to be trialing a cocktail together, to find out which cocktails are the most effective for different types of cancer.

We know that pitavastatin (a statin) for ovarian cancer works really well. You get a 40% increase in survival with statins for ovarian cancer. Pitavastatin is particularly useful, but you need to take it at a high dose. However, if you take ivermectin, you can reduce that dose because it's synergistic. So ivermectin and atorvastatin look to be super, super good for ovarian cancer, but again, that's just part of the cocktail, which would be really good. You have to look at the research, and just because those randomized clinical trials aren't there, doesn't mean to say it doesn't work.

Brad Power 51:12
On diagnostics: you have many pathways, you have many drugs, you have many options. Are there things that you can do from getting tests and diagnostics that predict whether someone will respond to this particular pathway, this particular drug?

Jane McLelland 51:36
I'm not in a lab, so that's not something that I can do. But Chris Apfel, or somebody like him, can take a tumor sample, and look at the different things that will work with it. I want to work a little bit more with Chris about looking at which cocktails might work to see where we go with it, and to actually gather some more evidence. I think it's important.

I like to have my n-of-ones in my Facebook group, and then try and pick through. Whenever somebody writes positive progress about what they've done, and they've got themselves to "no evidence of disease", I like to pick through and think, "Aha. They've used that with that." That's blocked. Then I try to work out the strategy of what they've actually done in order to achieve NED. It's very tricky. I don't know everything, so I can't absolutely guarantee I know what they're doing, but I can get some idea of what they've done, and how it may have worked.

Coming up with some cocktails in the future is something I would love to do; to work with a lab to try and create some cocktails for things like pancreatic cancer. I'm itching to have my own clinic and use some cocktails to try out. Ferroptosis is particularly useful for certain cancers. And pancreatic cancer, for example, tends to respond to ferroptosis treatments much better than many other ones. KRAS colorectal cancer responds super well to ferroptosis treatments, which is using intravenous vitamin C. But because it's intravenous vitamin C, you don't have clinics using that. It's very hard to get your traditional doctors to look at that. They're still absolutely obsessed by the fact that they think that intravenous vitamin C is antioxidant, whereas, in fact, it is reacting with the iron cell, and it's pro-oxidant. It's releasing hydrogen peroxide into the area. So it's not antioxidant. It's a completely different effect.

Brian McCloskey 54:12
Are there basic tests, like blood tests, for example, that would provide clues in terms of any supplements that we should be taking?

I would take that one step further, since you talked about the microbiome: What about microbiome tests?

Jane McLelland 54:34
I think that's key. I did that. I certainly had my gut microbiome tested to see whether I had sufficient bifidobacteria and lactobacilli. And akkermansia is really key in the gut as well. Butyrate will help as well. There are certain things that you definitely need in your gut in order to create the right environment.

In terms of actual tests, I did the PKM2 tests. That's the pyruvate kinase m2, and that is a test that looks at glycolysis. You've got LDH as well, lactate dehydrogenase, which, again, is another glycolytic enzyme. If you're looking at those two, you can get some ideas.

Now looking at glutamine, what do you do? I've looked at this hard to try and find some sort of blood test to see whether you can find out whether the glutamine is a big problem or not. I'm not sure that there is one, sadly, but what you can do is look at your homocysteine levels, because methionine can be very raised sometimes, and that converts into homocysteine, and then that provides cysteine in acetylcysteine to create the antioxidant which will prevent the cancer from being killed off. It's a way of preventing the cancer from being killed off. So methionine is another thing, or homocysteine is another test. I think people should look at that. ESR might give some idea if there's infection going on.

Brian McCloskey 56:26
These would be tests that would come from a microbiome test.

Jane McLelland 56:32
No, these are blood tests.

Brian McCloskey 56:41
Are these listed somewhere so I could get a comprehensive list?

Jane McLelland 56:52
I have got them in my ferroptosis chapter, but otherwise, no. I was going to speak to Life Extension and do a panel with them of tests. Because I don't know anybody who does the PKM2. I was very lucky. I had a Russian lady who had developed this test, and she happened to be living in England at the time. I think she is still here. I'm not sure, but I don't know many people who use the test anymore, but for me, it was very useful, and I think it would be useful for other people as well.

In terms of determining what your cancer is feeding on, we should be doing PET scans to look at glucose, glutamine, choline, kind of like merging them all and seeing which one is actually lit up most, to try and get an idea of what it's doing.

There must be others. I'm going to go to a conference in Bilbao. It's the EACR Metabolic Conference. (The European Association for Cancer Research Cancer Metabolism conference will take place from October 8–10, 2024, in Bilbao, Spain. The conference will bring together experts to discuss the latest advances in cancer metabolism and how to use this knowledge to treat cancer.) Matt van der Heiden will be talking. He's spot on with this kind of thing. This is part of my mission to try and find out those kinds of tests that we can run to really try and fine tune what we're looking at in the cancer patient, because otherwise it is a bit of guesswork.

But you can find key phenotypes that the cancer will use. We know that pancreatic cancer, for example, we use macro pinocytosis. It's also got a big stroma, so we need to get through that. There are lots of different features that we know about different cancers that we can work on that aren't necessarily attacked by the traditional oncologists. They're not necessarily looking at the metabolic features of the cancer.

You can do the research. Use Perplexity. Use consensus. Use ChatGPT, unless I don't favor chat GPT that well over it. Consensus and Perplexity are great for trying to work out the phenotype of different cancers. You can do a lot of research there, and you can find out which of those pathways on the Metro Map are key for you.

Brian McCloskey 59:20
I'll reach out to Rob Knight. He runs a whole gut microbiome center here at UC San Diego. Let's see if maybe he can coalesce the work that you're doing and what he's doing with the microbiome.

Jane McLelland 59:37
William Li uses fecal transplants and does all sorts of things before giving people immunotherapy. The difference in the result is quite phenomenal. It's a big thing.

Brad Power 1:00:09
Some specific questions I see about GBM, pancreatic cancer, we've got ovarian cancer represented. There's some comments about prostate cancer, and the comment being that some of these drugs are hormone-driven, and so how does that interact with the metabolic analysis you're doing?

Jane McLelland 1:00:38
If we're looking at hormones, if you roll back from the ultimate, you get more testosterone and prostate cancer, you get more estrogen and breast cancer. But actually the triggers for that, when you look back and you sort of run it backwards, you've got inflammation which drives it, and the malonate pathway, which produces these hormones, also is involved. That's another reason why statins could be very useful for the prostate and breast and the fat side of it, because of the hormonal implications.

I cover more of that in my online course, blocking the hormones, but it's obviously going to drive a lot of the growth factors. That's what it does. Estrogen stimulates growth. And testosterone.

Brad Power 1:01:42
There are a number of questions about access to doctors in the US who follow your practice and your models. Someone pointed out that you have a list of doctors in the US that can be found.

Any comments on access to your work?

You said you're running a course with a discount.

Jane McLelland 1:02:12
I've got a teachable course which effectively breaks down all of those modules, from the tumor microenvironment through to the fast dividing cells, to looking at things like the CASP phase cascade and how to increase the death of the cancer. That is a whole load of info. That's available.

If you want to get hold of doctors: if you go to my website, which is howtostarvecancer.com, go to slash doctors slash, then you've got a list of people who are happy to provide off label drugs. Will LaValley is there.

Brad Power 1:03:01
I was going to call on Will.

From your perspective, do you have any comments you would have, since you're using similar ideas. Anything you would add to the discussion?

Will LaValley 1:03:17
I think the concept of a broad spectrum of therapeutic agents is really important. The understanding, and I think in pointing out the example of metformin targeting multiple molecular vulnerabilities as a component of a range of therapeutic agents, the concept that I find most useful is that of molecular network targeting. The way Jane presents it is brilliant. It is understandable, it's digestible. The point that we're dealing with preclinical trial data is a big issue. It's inherently important to transparently discuss that we're using preclinical trial data in general, and the people, the doctors and their patients, have the options to consider it in addition to what the conventional treatment is. There's a rationale for using these options. And the challenge – exactly what Jane was talking about – is what's the best approach? What are the best combinations? How do you know? And what way do you know how much dosing and who's going to tolerate what? So it's inherently a molecular network approach using synergistic combinations. The construct of overlapping, concurrent therapeutic interventions. Jane has done more than anyone in presenting this information so that physicians as well as their patients and their family members have an understanding of this.

Brad Power 1:05:24
Thanks for that endorsement.

Rick Davis 1:05:39
What strikes me most listening to the presentation is that Jane does not really distinguish between stage four cancers. Not all stage four cancers are equal, and there are many people in stage four who live for many, many years. Are they incurable? Yes. Are they terminal? Well, a lot of people may die from something else. We see that. We work with those people, particularly in prostate cancer. It very much depends on the phenotype of the prostate cancer. So when statements are made like, well, take Metformin or take statins, it may not apply to all cancers. For example, for years, there's been a movement in prostate cancer to take aspirin, statins and metformin. It started with Snuffy Myers back in the 1990s, but now there's a preponderance of evidence around Metformin. It doesn't make much of a difference. I put a link to a meta study in the chat window. It doesn't make much of a difference unless you have diabetes. Not that I wouldn't take it or suggest it if I needed it, because it really doesn't do much harm unless you take it in too large doses. Oftentimes we'll tell guys to cut back what they're taking if they get a bunch of diarrhea, but it probably doesn't do much good, sort of like chicken soup. It may help and it can't hurt.

Jane McLelland 1:07:34
Is this study looking at Metformin on its own?

Rick Davis 1:07:42
Yes.

Jane McLelland 1:07:45
They did a study which came out on Medscape, and said, “Metformin bombs in the LANDMARK cancer trial for breast cancer.” But when you looked at the details, it didn't bomb. It was shown to be very useful for HER2. There's a lot of backlash against this, because people, big pharma, don't want these cheap drugs.

Rick Davis 1:08:16
I agree with you, but like you just said, it did great in HER2. You have to be more specific. Yes, it may be great HER2, but it's only specific for certain things. This was a meta study. I don't know how many were in the meta study. It's further back in the chat window, but you know what? I'm going to copy it and put it right at the bottom right now. You can go to it.

Jane McLelland 1:08:43
I can have a look. That's fine. It's the same with the breast cancer one, but it was looking at Metformin on its own. If you use Metformin, it blocks the electron transport chain in the ox FOSS pathway. That's one of its key things, that it does. Hexokinase, too. What does it do? You actually get something called glucose, glutamine, carboxylation. So the Krebs cycle goes backwards. Instead of going round this way, it suddenly goes oops, and then it will pump out the citrate instead of doing the normal Krebs cycle, because of the interference of the Metformin, but the cancer can learn to switch around in different ways. But the key is that you just need things that will make it work. You don't necessarily need a lot of things to make it work, but you just need the right combination and different cancers so if you have a P10 loss with prostate cancer, you're going to be more glycolytic. Your cancer, your specific phenotype of your cancer, and how it works, may be more aggressive, depending on the genetics as well, and that's something else I haven't really talked about, but those genetic features will make a difference as to how responsive you are.

Rick Davis 1:10:02
You're making my point, Jane, which is that this presentation talks in generalities, when, in fact, what we've got to do is almost look at this like it was a precision medicine, and start at the bottom and find out what microbiomes you have. Do a next generation sequencing, find out what mutations you have, and then we build on what you are, with what you have.

Jane McLelland 1:10:31
It gets expensive, tricky. All these tests assess, redress stuff can be phenomenally difficult for people to keep looking at all this stuff all the time. We have to try and make it accessible for people to do, and you've got to cut down some of that noise, otherwise you'll never get any.

Rick Davis 1:10:52
It's more expensive to buy all these drugs, even if they're supplements, when they don't work, than to know what will work and then buy the specific supplements for your particular case. So when we make these, because we have guys come into our and women come into some of our virtual groups, and they say, “This one says, get this.” And this one says, “Get that.” The issue is, is it going to work for you? It's not “one size fits all”.

Jane McLelland 1:11:22
The point is that it has to be more personalized. This is why I would have you use Perplexity or Consensus AI to look into.

You will get specific information as to whether it's a drug that's been used in mice, or whether it's been used in humans, or whatever. You'll get more information. The best evidence is in humans, but we don't have that with most of the off label drugs, and I don't think we will ever have it. This is a problem. It's a big problem. How do we overcome that, and how do we get to the point where we say, “This is a cocktail that will really work?” All we have to do is just keep going and build up the numbers, build up the data, and have people like Glenn Sabin or whoever, or xCures whoever it is, building that data, that platform, to say, X number of people who have used this cocktail, and it's worked for this number of people. That's what we need to do. I don't think we will ever have the information that we really want. It's a problem because of the financial incentives to do these tests. They're just not there unless we have some very, very generous philanthropists who put some money forward for us to do it. We're always going to have this argument, “Oh, there aren’t enough RCTs.” It's going to be my endless argument about it that we will have to say that we'll have to look at the best evidence that we can get and work out the potential metabolic pathways that you specifically have depending on your phenotype of the cancer, the genetics that you have, those mutations, what they are actually going to do to you, to your cancer, and then look at the best combination that we can from that perspective.

This has been great. I knew I'd get the question on RCT. There's no evidence. In fact, that was classic, that was expected. But, you know, there is evidence.

I encourage people to use some of the really good AI that's available now to have a look at their particular phenotypes for their own cancers, because it's very useful, and they will come across. If you haven't done much research into off label drugs for your cancer, start doing a little bit of hunting around and become maybe a little bit more comfortable with it, because it worries me that some people don't start. A lot of mistakes that people make with cancer is that they don't look at the extra information that's out there; they just focus on what the doctor said. They don't worry about their diet, they don't worry about exercise, they don't worry about all of these other things, the stress, etc, all of that, all of that needs to be taken into consideration. It all has a bearing on how well you're going to do with your treatment. Maintain an open mind, and I'll leave it at that.

Brad Power 1:15:05
Mechanically, I'm very curious, because I'm very comfortable using AI and Perplexity and so on. What do you input? Do you take your medical record, do you take your test results? What do you input into Perplexity to say, “What's the customized treatment just for me?”

Jane McLelland 1:15:22

You have to be quite specific about it. I won't necessarily get the right answer straight away, because you can say, “What are the key metabolic features of prostate cancer?” It won't necessarily tell you the right answer. Then you have to break it down to, “Is arginine involved in the metabolism for driving prostate cancer?” Then you have to work. You have to be more specific about the types of amino acids, etc. Then you have to go into those pathways.

My job is to educate people what those pathways are. That's my metro map, because otherwise, how do you know what you're asking? How do you know to get the information without knowing what those pathways are? That's key. You can put those individually into Perplexity, and you can say things like, “What off label drugs, or what repurposed drugs might be useful for blocking particular pathways?” I've learned some new ones since doing it. It's a useful tool.

Brad Power 1:16:37
So you're having a conversation, is what I hear. You're asking a series of questions about a variety of pathways. But the question is, how do you know whether that's relevant to me? What are the things that might be showing up in your blood work and your test results in your history that you would then say, “This is me querying that pathway.”

Jane McLelland 1:16:59
If you know that you have always eaten a high meat diet, you're probably going to be feeding more on the amino acids. That's the way it would prefer to feed, if you have a lot of glutamine running around the system. So your diet will have some bearing on it. Obesity as well. That has a bearing on it. Your insulin resistance. All of those tests are very useful as well to see whether they may have a bearing on your cancer into what you should be doing about it. If you're very insulin resistant you should maybe look at the ketogenic diet for a while, but I don't generally say that to people with prostate cancer, because I think the meat and the dairy is so key in terms of cutting those out. There's a lot of evidence behind that.

I talk about Michelle Montagniak, who was one of the first ones to promote the keto diet, which was meat and lots of dairy and all of that. He died of prostate cancer, and yet he was trying to work at improving his heart parameters, but he didn't end up helping himself, because he died of prostate cancer because of the meat and the saturated fat and everything else he was having. That’s his little story.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

CHAT DISCUSSION

00:06:37 Tracey Nicole’s Iphone 7 (3): Hello everyone! I’m diagnosed with stage 3aHGSC ovarian cancer

00:14:44 Helen: Hello, I’m diagnosed with Mesonephric-like adenocarcinoma ovarian cancer. There is no protocol for it. It’s a rare form of OC, making up less than 1% of OCs, so no protocol. They use the protocol for High Grade Serous OC. Thank you, Jane for joining us.

00:18:07 Rick Davis: Stage 4 in practice is incurable but not necessarily terminal or final. Depends on the cancer.

00:18:26 Robb Owen: Have you looked into micronutrient deficiencies and their role in DNA mutations during nucleotide synthesis? Specifically zinc and selenium. These micronutrient deficiencies along with genistein play a significant role in aerobic glycolysis, ROS and oxidative stress.

00:23:12 Rick Davis: Are your comments T4 cancer specific? There is plenty of data around Metformin and prostate cancer, mostly showing it has no impact, especially for T4.

00:26:06 allen morris: I saw a molecule listed called NF-kB. Is that a molecule that either drives cancer or is protective against? And is that something that is druggable in a positive or inhibitory way with your go to agents or lifestyle changes?

00:27:00 Rick Davis: Prostate cancer is not especially glucose driven - how does this effect your recommendations?

00:28:52 Roger Royse: PCKS9 inhibitors (Repatha) have recently also been shown to help similar to statins

00:33:50 Helen: Why do say COC may not be around much longer? Do you mean the one in the USA, which you no longer support. Or the one in London or all of them?

00:34:20 Robb Owen: Zinc regulates NF-κB, and its deficiency can lead to increased inflammation, potentially promoting oncogenic pathways [13].

00:36:50 Brian McCloskey: What kind of diagnostics would you recommend to understand and monitor cancer drivers that support the "metro map" cocktail?

00:37:23 Tracey Nicole’s Iphone 7 (3): What was the name of the super statin that was showing great promise for ovarian cancer? I am currently using lovastatin but it seems I may need to change. Thank you so much.

00:43:46 Ellen Miller: Where in the US could a stage 4 person go for medical guidance and treatment that include the things you are talking about?

00:44:32 Ellen Miller: I’m on folfiri chemo; are supplements and/off-label drugs advised, or not, while on chemo.

00:49:51 Rick Davis: Metformin and prostate cancer mortality: a meta-analysis https://pubmed.ncbi.nlm.nih.gov/26537119/ Conclusion - a benefit of metformin in men with diabetes and prostate cancer.

00:50:23 allen morris: Do any physicians in mainstream Academic Medical Centers in the USA endorse your approach? And if so can you share who they are? So, that one could co-pilot with your list of physicians.

00:50:47 Katya Tsaioun: Reacted to "Do any physicians in..." with 👍

00:51:07 Rick Davis: Is there empirical evidence for your recommendations?

00:52:03 Helen: Replying to "Do any physicians in..."

Have you seen the list of professionals recommended on her website, in diff countries?
00:52:19 allen morris: Replying to "Do any physicians in..."

No
00:53:06 Rick Davis: Dr. Morris - I think yu cn answer your queston by looking at Ms. McC's list of recommended doctors https://www.howtostarvecancer.com/doctors/ There is no representation from Centers of Excellence, many of which have integrative windows.
00:53:40 Helen: Replying to "Do any physicians in..."

https://www.howtostarvecancer.com/doctors/
00:54:52 allen morris: Replying to "Do any physicians in..."

Looks like mainly integrative centers. No academic medical centers
00:55:36 Rick Davis: Replying to "Do any physicians in..."

Dr. Morris - I think you can answer your question by looking at Ms. McC's list of recommended doctors https://www.howtostarvecancer.com/doctors/ There is no representation from Centers of Excellence, many of which have integrative centers.
00:55:42 allen morris: What was your diagnosis and how was the diagnosis made?
00:56:41 allen morris: Reacted to "Dr. Morris - I think..." with 👍
00:56:48 Rick Davis: Not all Stage 4s are equal
00:59:45 Katya Tsaioun, PhD: None of the recommended doctors are in Boston area, a “frontier” of cancer research. State licensing laws are, thus, limiting patients’ access to such therapies. Any workarounds?
01:00:26 allen morris: Do you have any long term survivors who have used your protocol, are PDAC pancreactic cancer stage Stage 4, and documented by pathologist diagnosis?\
01:00:51 Helen: Replying to "What was your diagno..."

I have her book, but also listened to many interviews with her on several podcasts - if she doesn’t get around to answering your question about her different cancer - you can hear about them on the podcasts.
01:05:33 allen morris: Has your protocol resulted in any long term survivors who have GBM?
01:05:35 Katya Tsaioun, PhD: MGH is refusing to do PET scans. Say they don’t get any actionable information, MRI is all they need
01:07:01 aimee donovan: MCRC with Kras G12D mutation here ..
01:08:10 Vanessa Hugo: Replying to "Has your protocol re..."

I know of at least one CPL member. I won't disclose her identity, but I believe she's 3.5 yrs out so far and still stable. She did a clinical trial (Selinexor) + Jane's metro map approach, but did not do Optune
01:08:43 Katka: Please cover breast cancer too - stage 4
01:09:05 allen morris: Replying to "Has your protocol re..."

Would love to know greater details?
01:09:26 Vanessa Hugo: Replying to "Has your protocol re..."

Send me an email! vanessaleehugo@gmail.com
01:09:46 allen morris: Replying to "Has your protocol re..."

allmorris@yahoo.com
01:10:18 Katka: I have your book and the online course... can i work out my course of action from that? From just book it is very hard. Lots of info. Also researching the specific cancer feeding and signalling is hard - many terms are not in your book
01:11:13 Dr. Chris Apfel: Thank you, Jane, great and comprehensive overview and I am looking forward to our next meeting with Dr. LaValley and you.
Need to go. Thank you again.
01:12:16 Helen: Replying to "Has your protocol re..."

Many folks showing results are on her FB page
https://www.facebook.com/groups/off.label.drugsforcancer
01:13:26 allen morris: Reacted to "Stage 4 in practice ..." with 👍
01:15:13 Rick Davis: https://pubmed.ncbi.nlm.nih.gov/26537119/
01:20:06 Rick Davis: What she is saying is not what she's selling!
01:21:31 Rick Davis: Folks - please make sure you have next generation somatic sequencing for your cancer. That's where it starts.
01:21:52 Helen: Replying to "Folks - please make ..."

How do I do that in France?
01:21:59 allen morris: Reacted to "Folks - please make ..." with 👍
01:25:02 Eric Hall: Thank you Jane for sharing all of this. I have done much of this myself and hearing your presentation really confirms this is a the path for me.
01:25:05 Vanessa Hugo: Thank you. Appreciate your work.

“Starving Cancer - Beyond the Metro Map” (Jane McLelland) [#113]

Follow-up Next Steps

- Schedule a follow up discussion on diagnostics and predictive testing for metabolic therapies
- Reach out to microbiome researchers like Rob Knight to collaborate on integrating microbiome work with metabolic research