CELL SUPPRESSION THEORY

Synopsis

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A new theory of cancer

All established cancer theories share a common ideological flaw — the **presumption** that cancer originates from '**cell malfunction**', implying that **cell damage** propels the development of the disease.

The issue at hand, is that none of these theories can successfully pinpoint the precise damage, or combination of damage, required to account for cancer's odd behaviour. As a result, all theories remain **unproven**, leaving the **origin of cancer shrouded in uncertainty**.

What if cell malfunction isn't the driving mechanism?

Presented below is a revolutionary paradigm that stands as one of the few unexplored paths of inquiry into the cause(s) of cancer. Mark introduces the notion that '**cell suppression**' serves as the underlying mechanism driving tumour growth and development, activated and controlled by **opportunistic intracellular pathogens**.

The distinguishing feature of this groundbreaking cancer theory, is its capacity to explain **every fundamental hallmark** of the disease, signifying that the causative factor(s) have been identified. Through this novel framework, not only does **cancer make sense**, but an **effective treatment approach** may at last be defined.

CELL SUPPRESSION THEORY
SYNOPSIS

Up to 70% of cancer patients in the US report using some form of complimentary or alternative therapy[1]. Between 40-50% do so in the UK[2], a significant amount. Given the less than favourable success rate with conventional treatments, many attempt to navigate the wild west of alternatives looking for an answer to this terrible disease, with little to no guidance. What's desperately needed is a simple and clear evidence-based framework that not only provides this guidance, but goes one step further to help patients easily and quickly identify the most effective therapeutic options for their cancer type. This would be the holy grail of cancer survival guides.

Well, this synopsis does just that. Summarising the significance of the *Cell Suppression Theory* and the unique insight it provides regarding the origin of the disease, this document introduces you to this much-needed evidence-based framework which is outlined in greater detail within the book '*THE CANCER RESOLUTION?*'. This multi-award-winning book enables you to better navigate this sea of alternative information, providing the critical insight needed to identify the treatment approach that will most likely improve your chances of survival. This is achieved through critical analysis of cancer theory and the presentation of a new theory that promises to revolutionise how we treat the disease.

The benefit of the book is in how it translates this newfound knowledge into layman's terms bringing the science of cancer into the public domain for cancer patients to easily understand. This book will empower you to take control of your treatment journey in a way that allows you to work with your oncologist as opposed to feeling like a helpless bystander. It provides hope and the confidence you need to engineer your recovery back to full health.

The benefit of this synopsis is in how it is able to summarise the key concepts presented in the book. It enables you to quickly grasp the value of the framework being discussed, and the value of the new theory being presented, both of which when combined and understood, have the potential to greatly improve your chances of survival.

Preamble:

In the drive to increase our understanding of cancer and improve survival outcomes, new cancer theories are being developed all the time. Currently there are at least nine, with four arising within the last 20 years. Why is this relevant? The development of so many theories highlights a critical issue that is pivotal to your ongoing survival as a cancer patient – and that is: **the origin of cancer**

1 *https://www.cancertherapyadvisor.com/news/survey-most-cancer-patients-use-complementary-or-alternative-medicine/*

2 *https://pmc.ncbi.nlm.nih.gov/articles/PMC4952625/*

remains unknown. In other words, no current theory has yet been proven to be correct – if it had, a cure would have been developed by now. This is noteworthy because it challenges the dominant narrative and throws into question the undesirable chemotherapy and radiotherapy treatments offered by most oncologists as the standard of care. It also opens the door to cancer patients taking control of their own treatment journey, with the potential to greatly improve their own survival outcome.

The key revelation here that should have us all excited, is not just that so many theories exist, it's that treatments are developed from cancer theory – each theory offers different treatment options based upon the mechanism claimed to be driving the disease. Why is this important? Over the last 60 years the medical establishment has focused nearly all of its attention and resources on one theory, to the exclusion of all others, namely the *Somatic Mutation Theory* – the notion that cancer is a genetic disease. This means there is an area of untapped potential waiting to be uncovered in the theories who's treatments have not yet been vigorously tested. To provide context and confidence in the framework I am mapping out for you here, it would be helpful to understand the scientific process of how theories arise and inform treatment development, here's a simple 7-step overview – step 6 is the most important:

1. Studies analyse tissue to identify key features of a disease
2. Several hypotheses are formed from this initial data
3. This provides justification for pursuing further research
4. Supporting evidence for a hypothesis results in the development of a theory.
5. The theory justifies and guides the development, and testing, of potential treatments in animals and then humans.
6. If treatments associated with any particular theory fail to make a significant impact against the disease, the theory is refined through more investigation before new treatments are again developed and tested. **Long-term failure to develop an effective treatment indicates the theory is incorrect**. This results in new hypotheses and new theories being developed to remedy this failure to resolve the disease. And so the process continues until these new theories enable the development of an effective treatment.
7. An effective treatment capable of curing a disease proves the associated theory correct.

In regards to cancer, we are currently at stage 6 in this process – specifically the stage of developing new theories. Let's dissect what this means. As I alluded to a moment ago, we don't seem to be testing any treatments associated with these additional theories, so we don't actually know if their associated treatments are effective or not – what is clear, is that they have the potential to be. Unfortunately, the natural inquisitive and progressive nature of science seems to have stalled and become stuck at the 'test the established theory' stage, where only treatments associated with the *Somatic Mutation Theory* are being tested in human clinical trials – hence why only treatments associated with this theory are offered to cancer patients by their oncologists.

For example, metabolic therapies that stem from the *Metabolic Theory* of cancer, have not been tested in large scale human clinical trials, so we cannot confirm or deny the effectiveness of this treatment approach – the significance of this will become apparent momentarily. In other words, as we haven't got round to testing them yet, any number of these other theories could be harbouring treatments that are actually effective against cancer. In this respect, it seems that science has let us down because rather than testing all theories equally to discover which treatments are the most effective, we are only testing one theory, under the false pretence that a consensus of support for this theory among scientists, means that this theory is likely correct – which is a dangerous unscientific assumption that risks hindering progress.

The scientific method demands that scientists are objective, meaning they should be guided by evidence and should test all plausible theories to the same degree and with the same urgency. They should not be guided by assumptions based on a consensus of thought that leads them to focus on one theory above all others. Science is about evidence, not consensus of opinion. We must also consider that it's not just opinion that's driving the consensus. Scientists pitch for funding, the vast majority of funding is in understanding cancer genetics as opposed to studying any other theory. If 90% of all investment is targeted towards genetic research, you will generate a consensus in that field of study by default, because that's where the money is, and usually where the greatest return on investment is made.

In light of this unfortunate state of affairs, learning about these additional cancer theories should be the new craze in patient circles, because they offer untapped potential for the discovery of treatments that can make a positive difference. Moreover, theories operate in a science-based capacity with supporting evidence justifying any protocol being proposed by them, so we know that the treatments associated with these theories are developed through a methodical scientific process – providing us with a level of confidence in the reasoning behind their use.

Scientists will caution that we shouldn't bound headlong into adopting these potential solutions before they are tested in human clinical trials – which I whole-heartily agree with. However, the dilemma for cancer patients is that scientists seem unwilling to test them for us to learn of their potential benefit. So, while we don't have the luxury of time to wait to see when this will happen, we are left with only two options:

1. Go along with standard of care (the controversial and undesirable treatments that have been tested and lack significant efficacy), or,
2. Take matters into our own hands by using the science available to us that can help us make an informed decision on which of these treatment approaches shows the greatest potential for combatting cancer.

The notion that this is dangerous due to its experimental nature should not be used to dissuade us from considering this line of inquiry – because even mainstream oncologists are experimenting with the limited range of treatments they are allowed to provide, by virtue of the fact they are not guaranteed to work. In this respect both oncologists and patients inhabit the same space when it comes

to attempting to treat the disease, both are forced to experiment as no effective cure exists. Hence why up to 70% of patients turn to complimentary and alternative treatments, and why oncologists offer patients the opportunity to take part in experimental clinical trials.

In this fog of uncertainty that both patients and oncologists inhabit, I could spend time bemoaning the unscientific nature of focusing all of our attention on the *Somatic Mutation Theory* – but I won't, because while it is frustrating that resources are not being spread between theories evenly, all is not lost, as we can still draw a valid conclusion from the abundant data gained from testing this one theory, data that can help us clarify the way forward:

- Despite a disproportionate level of investment and research ploughed into attempting to prove the *Somatic Mutation Theory* correct over the last 60 years, no cure or effective treatment has been forthcoming for the vast majority of cancers.

With this in mind, I refer back to the 7 step scientific process mentioned a moment ago – referencing a key sentence from step 6 in that list provides us with important insight: "*Long-term failure to develop an effective treatment indicates the theory is incorrect.*" It's very telling that we are now at the stage of developing new theories to compensate for this lack of progress. The failure of the *Somatic Mutation Theory* to enable the development of effective treatments over such a long period of time, is a reflection that the established theory could indeed be wrong – a message perfectly illustrated by Professor Paul Davies during one of his presentations.

In his 2013 presentation of the *Atavistic Theory* at the *New Scientist live* event in London[3], Professor Davies provided some chilling insight. Firstly he confirmed that despite all the drugs developed so far, they only improved life-extension for later stage disease by 4.1 weeks on average. Now updated, the latest figure is 2 months of life extension[4] from all the drugs created – nothing to shout about. Secondly, he explained why so little progress has been made. Citing research performed by the *National Cancer Institute* in America, Professor Davies announced that up to 80% of a million cancer studies analysed, cannot be replicated. In other words, they are wrong. Let that just sink in for a moment as you contemplate accepting the treatments offered to you by your oncologist, and oncologists of clear conscience please take note. Up to 80% of the studies that form the foundational evidence in support of the dominant cancer theory – the notion that cancer is a genetic disease – are incorrect. Translated: the dominant view of cancer, as well as the treatments provided to patients, are largely based on false, flawed, and incorrect science.

Is it any wonder that effective treatments have not materialised when the rationale for their use is flawed to begin with? In light of this revelation, the problem appears to be that many medical professionals seem reluctant to accept the possibility that the theory they've invested so much time, effort and money in, could be wrong – and so nothing much changes, while patients continue to be

3 *https://www.youtube.com/watch?v=yoQYh0qPtz8*

4 *doi:10.1001/jamaoto.2014.1570*

offered undesirable treatments that provide no guarantee of successful resolution. The crazy aspect of all of this, is that medical professionals get cancer too, so they are inadvertently restricting their own chances of survival by unwittingly adopting the limited vision of what cancer is and the flawed treatment approach associated with it. The error in such dogmatic thinking and devotion to this one theory, seems evident when explained in this context.

So, where does this leave cancer patients intent on taking responsibility for their own health, and wishing to research different theories in the hope the treatments associated with them may offer salvation from this terrible disease? Firstly, while we shouldn't dismiss the *Somatic Mutation Theory* and conventional treatments entirely, this does give us pause to reconsider, or at least the justification to question, the advice and treatments offered by our oncologists. Having said that, I would always advocate for surgery where applicable and if desirable, as early diagnosis and surgery form the majority of the success stories against cancer. Secondly, without any significant clinical data to support the treatments associated with these other theories, we have to rely on the next best metric at our disposal, and that is to determine the accuracy of any given theory. This is the key measurement that can inform our decision-making process.

In essence, the more accurate the theory, the greater the likelihood that a key mechanism is being targeted, which in turn means the theory's associated treatments are likely to be the most effective. In other words, you can potentially improve your survival prospects by identifying the most accurate cancer theory and then adopting the treatments associated with it. Or to put it more bluntly, your survival largely depends on which theory of cancer you choose to subscribe to. In this respect, if you choose to place responsibility for your recovery in the hands of most mainstream oncologists, you are inadvertently subscribing to the *Somatic Mutation Theory*, because this is the theory that they also subscribe to. With this in mind, we've now established that patients have four choices when it comes to their therapeutic options. To clarify, I would always advocate for surgery where applicable and if desirable – so the options below are not indicating that you should forego surgery in early stage disease – these options are more relevant to later stage disease where we find that conventional chemotherapies are far less effective than surgery:

- **Option A**: Subscribe to the *Somatic Mutation Theory* and undertake the mainstream treatments that have been tested in clinical trials, treatments that have a proven track record of only extending life by approximately 2 months on average.

  Potential benefit: While some cancers appear to respond much better than others, the potential benefit is low and risky. This is reflected in the low bar set for treatment success which is primarily measured based on 5 year survival, as opposed to long-term disease free survival, or overall cancer mortality.
- **Option B**: Learn about a number of other cancer theories that have largely been ignored; based on the prospect that several of these under-funded and untested theories may actually be more accurate, meaning that the treatments associated with them may show greater efficacy against the

disease. And of course, there is always the outlandish prospect that one of these theories may have identified the underlying cause already.

Potential benefit: High, due to the utilisation of a robust evidence-based framework that enables the identification of the most accurate theories, and by extension, the treatment approach most likely to be effective.

- **Option C**: Do your own research without utilising the knowledge base and supporting evidence that comes from studying cancer theory. Instead, rely more on pre-clinical and anecdotal evidence to inform treatment decisions in the hope that what seems to have worked for others, or in rats, will work for you.

  Potential benefit: Low to medium, but extremely risky due to the lack of a robust evidence-based framework that can guide decision-making.
- **Option D**: A combination of option A and C.

  Potential benefit: Better than option A alone if evidence-based.

The case for choosing Option B:

Determining the accuracy of each theory is the key to identifying the treatment approach that has the greatest potential to improve survival outcomes. It can help us clarify which features of the disease to focus on targeting. So, how do we determine the accuracy of a cancer theory to enable us to make an informed decision? Thankfully we have a robust metric for doing this. The accuracy of all cancer theories can be measured against the number of hallmarks that each can explain. There are currently 10 officially recognised hallmarks of cancer put forward by Hanahan and Weinberg. These are the features that define the disease. For example, failure of cell death mechanisms (apoptosis), unbridled cell growth, blood vessel growth (angiogenesis), and immune evasion are just four hallmarks shared between all solid cancers that are required to be explained by any given theory. The goal is to explain all 10. The more hallmarks that can be explained, the more accurate the theory is deemed to be. The more accurate the theory, the greater the likelihood its associated treatments will be effective due to the greater probability that a key mechanism driving the disease is being targeted.

This all sounds plausible and very scientific, but analysing different cancer theories against each hallmark to determine their accuracy is a daunting prospect for any scientist, let alone a member of the public, after all it took me eight years of research to arrive at my current position. So, to save you precious time and a probable migraine, I've done the hard work for you and already assessed the accuracy of the most relevant theories that offer the greatest potential to improve your survival outcome.

Just to be clear, I'm not asking that you blindly trust my assessment of the accuracy of each relevant theory, far from it, there's no place for trust in science, which is why my book contains over 800 references. With that in mind I encourage you to always do your own due diligence and to assess every claim I make on its own merit. My goal has been to make this process as easy as possible by including the quotes and links to the evidence in support of my reasoning so that you can

judge my conclusions for yourself based on the evidence.

Taking my assessment at face value for the purpose of this synopsis, when it is applied to a number of leading theories, an interesting picture emerges that can help us make better informed treatment decisions moving forward. Significantly, the established *Somatic Mutation Theory* (SMT) that informs the treatments offered by most conventional oncologists, struggles to explain more than 2 of these 10 hallmarks, indicating that genetic mutations are not driving the disease. This suggests that conventional treatments are likely targeting the wrong mechanism, and goes some way to explain why no cure has been forthcoming. Both the *Cancer Stem Cell* (CSC) and *Tissue Organisation Field Theories* (TOFT) appear to explain between 4 and 5 hallmarks, while the *Metabolic Theory* (MT) seems capable of explaining at least 7, arguably more. At a glance, this suggests that cancer is more likely a metabolic disease influenced by damaged terrain and corrupted stem cells. Worth noting, is that the treatments developed from the *Somatic Mutation Theory* can worsen and stimulate all of the mechanisms thought to drive cancer that are explained by the other three more accurate theories.

All of a sudden a new framework of how best to approach the treatment of cancer starts to emerge, one where three theories align and become front runners in the formulation of a potential protocol that utilises metabolic therapies, with a view to targeting cancer stem cells and repairing the cellular terrain. This clarity helps us to understand why we should question the dominant approach. With this in mind, its important to further critically analyse the *Somatic Mutation Theory* to reaffirm why it may not provide the most beneficial treatment strategy.

REASONS TO RECONSIDER the Somatic Mutation Theory:

While proponents of the *Somatic Mutation Theory* claim that DNA mutations drive the disease, often unscientifically citing this as fact, an overwhelming body of evidence indicates that this established interpretation of cancer is **incorrect**. This is extremely concerning given that *Standard of Care* treatments are developed from the *Somatic Mutation Theory.* With no cure forthcoming after six decades of research, billions invested, and treatments that only extend life by two months on average[5], has the medical establishment lost its way?

Here follows some of the most significant shortcomings of the *Somatic Mutation Theory* worth taking into consideration when determining the treatment path you wish to take:

1. **Random DNA mutations do not explain the consistency of cancer:** Before we were able to analyse all of the 21,000 DNA genes in cancer cells to draw a valid conclusion, the *Somatic Mutation Theory* proposed that a predictable pattern of DNA mutations were driving each cancer type, much like how specific keys on a piano are used to play a specific tune. However, by 2013 scientists unexpectedly discovered that mutations were utterly random, even between the same cells within the same tumour[6]. How could such randomness be causing the consistency of the disease? This is akin to

5 *For later stage cancers* – doi:10.1001/jamaoto.2014.1570

6 *https://www.cancernetwork.com/view/heterogeneity-and-cancer*

pressing random combinations of keys on the piano and expecting to hear the same tune every time.

2. **P53 and RAS:** Of approximately 13,000 tumour samples analysed through the *The Cancer Genome Atlas* database, the p53 gene and RAS gene were found to be mutated in 35% and 18% respectively[7]. P53 is responsible for instigating cell death mechanisms, and RAS for cell growth control. Mutations in both can prevent cell death and trigger cell growth forming a tumour. Surely, in order to claim DNA mutations are responsible, these two key genes need to be mutated in 100% of cancers? But this isn't the case.
3. **There are insufficient numbers of driver mutations:**
   In accordance with the SMT at least 8 DNA mutations are required to explain cancer's hallmarks. However, studies show that many cancers do not contain enough driver mutations to explain the disease[8]. The average driver mutation rate was found to be 1.7, not the 8 required.
4. **Cancer develops without mutations:** Some cancers show no driver mutations at all[9]. In fact a recent 2024 study analysing 10,478 cancer genomes found that approximately **45% of tumours did not have identifiable driver mutations** in the genes they studied, meaning these cancers were associated with **no detected driver mutations[10].** How can cancer be genetic, if it forms without the required genetic mutations?
5. **Mutations in healthy tissue, but no cancer:** Despite alleged cancer-causing mutations being present in healthy tissue, cancer does not form[11].
6. **Failed DNA transfer experiments:** Mutated DNA from cancerous tumours were transferred to healthy cells. The aim was to show that this cancerous DNA would drive abnormal growth in these healthy cells. However, abnormal growth does not occur[12], directly challenging the notion that the mutations present in tumours are driving tumour growth.
7. **The Tissue Organisational Field Theory (TOFT) challenges the SMT:** Studies show that if you take cancer cells and place them within the vicinity of healthy cells, they revert back to being normal again[13]. This occurs despite the mutations remaining within the now healthy tissue, indicating that those mutations were not driving the tumour growth.
8. **Failure to explain cancer's hallmarks:** In light of the lack of association between DNA mutations and cancer development, it's not surprising to discover that the *Somatic Mutation Theory* struggles to explain more than 2 of the 10 hallmarks required to be explained. This is simply due to the fact that the required mutations are not present, so cannot account for each

7 *https://portal.gdc.cancer.gov/analysis_page?app=MutationFrequencyApp*

8 *https://www.pnas.org/doi/10.1073/pnas.1803155115*

9 *www.ncbi.nlm.nih.gov/pmc/articles/PMC3933226/*

10 *https://www.nature.com/articles/s41588-024-01785-9*

11 *https://pmc.ncbi.nlm.nih.gov/articles/PMC8765002/*

12 *https://aacrjournals.org/cancerres/article/63/11/2733/510012/Mouse-Embryos-Cloned-from-Brain-Tumors1*

13 *https://www.sciencedirect.com/science/article/abs/pii/S0079610716300888*

hallmark. For instance, how can the failure of the cell death mechanism be explained using DNA mutations, when the required cell death genes are not consistently mutated in all cancers? This failure to account for these crucial hallmarks, which are the measure of accuracy of any theory, emphasises that cancer is likely not genetic in origin.

These are just a number of key contentions with the *Somatic Mutation Theory* that throw its validity and that of its treatments, into doubt. There are more. This suggests that cancer is not a genetic disease and in turn highlights the danger in only treating cancer from this genetic perspective. While chemotherapies can provide a benefit, there is clear evidence to warrant looking into other theories and the treatments associated with them. I'm not definitively stating that cancer isn't a genetic disease, or that there isn't a genetic component, but given these contentions and the clear shortcomings of our current chemotherapy approach, it makes sense to consider other view points and options, especially when we have the ability to assess the accuracy of other theories worthy of our attention.

I'm not against conventional chemotherapies either, they can reduce tumour size to enable removal of the tumour via surgery. Low dose chemotherapy as proposed by Dr Robert Gatenby, offers the potential for improved management of the disease. Rather, I'm highlighting the elephant in the room: that many are guilty of failing to approach the problem of cancer from an objective problem-solving perspective, and from first principles. Unwarranted devotion to this theory has hindered progress in both understanding the disease and developing effective treatments. Patients and oncologists need to be aware of this if we are to elicit change that can benefit us all.

Making history, pioneering a new understanding & generating hope:

In light of the shortcomings of the *Somatic Mutation Theory*, and despite the improved accuracy of these other theories, there remains the sense we are still missing a major piece of the cancer puzzle. It would seem that something miraculous and extraordinary is required to break the deadlock. Well, the prayers of many may have been answered, because in 2023 the *Cell Suppression Theory* was published. While this theory justifies the development and testing of new and existing drugs in clinical trials, the theory can immediately benefit patients without the need to wait decades for trials to be conducted. This is because the treatments associated with the theory are available to be used immediately, and can be adopted to work along-side current treatment protocols. Furthermore, abundant evidence already exists to justify their use.

While the antagonist at the heart of this new theory has been considered before, the proposed mechanism by which it potentially generates cancer is new, and reflects a significant departure from conventional thinking, the kind of out-of-the-box thinking that could turn this killer disease into something no more dangerous than the common cold.

This is a bold claim, but not one that is made lightly, for the *Cell Suppression Theory* appears to be the most accurate cancer theory currently available by virtue of the fact it is the first theory capable of explaining all 10 hallmarks of cancer. This

indicates that a new mechanism driving cancer has been identified, and that if targeted effectively, the disease can be resolved. This means that the *Cell Suppression Theory,* published within the book '*THE CANCER RESOLUTION?*', opens the door to additional treatment options that have the potential to greatly improve the survival outcome for all cancer patients. This is exciting because the simple manner in which the science is explained means that the theory's basic principles can be grasped by the layperson very quickly, without the need to research key points in depth – thus empowering patients and offering renewed hope.

To be clear, the *Cell Suppression Theory* is not just another theory, it's in a league of its own. This is because most cancer theories are developed from the same, potentially flawed, premise – the notion that cancer is a result of '**cell malfunction**'. This one universal assumption underpins the foundational premise behind most cancer theories – and highlights the potential error in thinking that underlies them all – what if cancer isn't the result of cell malfunction? Where do mainstream theories stand then? Such a reality would explain why there are so many cancer theories in development (at least nine), and why most fall short in fully explaining the disease. Based on this understanding, you could argue that only one dominant theory exists – the 'Cell Malfunction Theory', and all other theories are just sub-theories competing to identify which feature of the cell has malfunctioned. The *Cell Suppression Theory* on the other hand, offers a different paradigm entirely, that cancer is the result of '**cell suppression**', not malfunction; no other theory falls into this category.

This unique paradigm shift is why it's so important, and potentially so beneficial, to invest time in understanding cancer theory, in particular the revolutionary *Cell Suppression Theory* discussed in my book. Being the most accurate theory indicates its potential to help patients finally combat the disease. Under this unique paradigm our cells are not seen to be defective or working against us. They haven't developed a mind of their own, become evil, or defied millions of years of evolutionary programming to develop autonomy that threatens patient survival as well as their own – as the cell malfunction paradigm asserts. Rather, our cells are doing what they've been designed to do, and that's to protect us. The problem is, they've been hijacked under compromised conditions leading to the suppression of key pathways that go on to inadvertently create the symptoms that we refer to as cancer. The question proposed by the *Cell Suppression Theory*, and then answered, is: why, and through what mechanism are our cells being suppressed, and how does this suppression paradoxically lead to uncontrolled cell growth?

The rest of this synopsis answers those crucial questions and provides a simple summary of my theory; offering insight into the problem-solving process that led me to this unique conclusion, including the pivotal features that set it apart.

A UNIQUE and exciting PROPOSITION:

The framework of using the accuracy of different cancer theories to determine the most effective treatment approach is a game-changer because it

legitimately guides us through a science-based process that can establish the best course of action to take.

Based on the premise that accuracy equates to an increased probability of treatment success, patients would do well to consider the *Cell Suppression Theory*, *Metabolic Theory*, *Cancer Stem Cell Theory and Tissue Organisation Field Theory* as foundational theories that can inform treatment decisions. The significance of the *Cell Suppression Theory* (CST) is not just the fact it appears to be the most accurate, rather it is that it incorporates all three of these other theories and their treatment approaches to generate a truly holistic therapeutic solution to cancer. In addition, it considers the role of the microbiome and mental health. This is why the CST offers the greatest potential for an improved therapeutic outcome and has the potential to benefit all cancer patients.

The problem-solving process that led me to develop a revolutionary cancer theory:

I realised quite early on in my quest to understand cancer, that the key to identifying the origin of any disease lies in consistency. The randomness of DNA damage, its lack of consistency, coupled with other major contentions found to contradict the *Somatic Mutation Theory* led me away from this concept and open to studying other theories. Out of all of these other theories the *Metabolic Theory* stood out for its identification of a significant consistency found to be present in all cancers. That consistency is called the *Warburg effect*. The Warburg effect describes the abnormal metabolism of cancer cells – the abnormal use of energy pathways. This is significant because through the process of the Warburg effect, the *Metabolic Theory* explains at least 7 of the 10 hallmarks, possibly more, making it the most accurate mainstream theory. The Warburg effect (aka abnormal metabolism) features as one of the 10 official hallmarks, such is its significance to the development of the disease. Allow me to expand on this.

Cells utilise two pathways for creating energy. One called **OXPHOS**, which combines glucose with oxygen, and one called **glycolysis**, which ferments glucose and doesn't require oxygen. The OXPHOS energy pathway resides within organelles called mitochondria, which are similar to bacteria, only they are key components of the cell. When these mitochondria are damaged, or starved of the oxygen they need, they can't generate energy, so the backup energy system of glycolysis takes over. This is where the cell itself (not the mitochondria) ferments glucose for energy. Once the oxygen supply is restored, mitochondria can again produce energy via the OXPHOS pathway by combining glucose with oxygen once more. When this happens, the backup energy system of glycolysis (glucose fermentation), reduces or is stopped. Glycolysis supports mitochondrial-based energy production (OXPHOS), and can operate as a backup.

OXPHOS is a more efficient way of generating energy, whereas glycolysis is a wasteful way of creating energy because it produces less energy for each glucose molecule processed, and it produces lactic acid, an unfavourable corrosive by-product – hence why OXPHOS is the preferred energy state of most healthy cells.

But what does this have to do with developing a new theory that's potentially identified the origin of the disease? Cancer cells primarily use

glycolysis (glucose fermentation) for energy even when oxygen is available. This is abnormal because mitochondria should be generating energy under these conditions. Scientists have been attempting to understand why cancer cells use this inefficient energy state. This is important because evidence suggests that cancer develops as a direct result of this energy shift to the Warburg effect. This is partly because of the corrosive nature of the lactic acid produced, which increases chronic inflammation, blood vessel growth, and partial suppression of the immune system. And partly because glycolysis is a proliferative energy state in and of itself – enabling abnormal cell growth if it is in use for prolonged periods. In other words, this energy shift is a pivotal feature of cancer, and sits at the heart of tumour growth. It didn't take me long to realise that identifying the cause of this energy shift should be the focus of my problem-solving quest, because whatever is causing the Warburg effect, is likely to be the driving mechanism behind the disease.

Proponents of the *Metabolic Theory* have recognised the significance of the Warburg effect in regards to cancer's origin. They have also recognised, as I have, the need to explain cancer's consistencies, and that this consistent metabolic feature of cancer hints at a singular origin. As OXPHOS is not producing energy in cancer cells despite sufficient oxygen being available, proponents of the *Metabolic Theory* have drawn the conclusion that this OXPHOS pathway must be damaged and no longer working. This is the consistent and singular origin of cancer proposed by the theory. Irreversible damage to OXPHOS allegedly forces cells to rely on their backup energy system (glycolysis) in order to survive. But as OXPHOS isn't repaired, glycolysis is sustained for longer than it should be. This results in a build up of corrosive lactic acid, blood vessel growth, and cells that start growing out of control due to the chronic inflammation that results. This abnormal energy shift to glycolysis even in the presence of oxygen was discovered by Otto Warburg in the 1920s, and termed the Warburg effect in his honor. It is from the Warburg effect that the rest of the symptoms (hallmarks) of cancer seem to develop, according to the *Metabolic Theory*. In other words, defective mitochondria, or defective OXPHOS, is the origin of cancer according to the *Metabolic Theory*, because this defect triggers the Warburg effect that goes on to generate the disease. The solution in this regard would be to apply metabolic therapies that starve the cancer of the glucose and glutamine that it thrives on, and to utilise treatments such as Hyperbaric Oxygen Therapy to reinvigorate mitochondria.

If defective OXPHOS is the origin of cancer why did I continue my research and go on to write a new theory? As good as the *Metabolic Theory* is, I came across a number of contentions that challenge the notion that mitochondria are dysfunctional. I needed to resolve this before I could agree completely with the conclusions of the *Metabolic Theory*. This doesn't mean that abnormal metabolism isn't a key feature of the disease, it absolutely is, and explaining the Warburg effect is still pivotal to identifying cancer's origin. It just suggested to me that something else may be responsible for the Warburg effect and so I continued my research. I cover these contentions in detail within my book '*THE CANCER RESOLUTION?*', so I will mention just some of them here to provide insight into my reasoning:

Metabolic Theory Contentions:

1. **OXPHOS appears to be operational**: Contradicting the *Metabolic Theory*, a number of studies show that OXPHOS appears to be operational, that it is required to be operational for malignancy, and can be upregulated under certain conditions[14] [15] [16] [17]. When OXPHOS has been targeted using drugs, a loss of energy occurs, and cancer cells are inhibited.
2. **The Tissue Organisation Field Theory**: This theory shows that cancer cells revert back to a normal healthy state when they are surrounded by healthy cells[18]. This reversion to a normal state of being indicates that OXPHOS is not irreversibly damaged.
3. **Oncocytomas, senescent cells, and children with Barth syndrome**: All are characterised by defective mitochondria where OXPHOS is also defective – the characteristics that mimic the conditions said to cause cancer. Yet cancer rarely forms[19] [20] [21], suggesting that defective OXPHOS results in cells that don't proliferate, as opposed to cells that do. Oncocytomas and senescent cells do not proliferate despite this defect.
4. **Prostate cancer is not initiated by the Warburg effect:** This is a major contention because prostate cancer is the most prevalent cancer among men, yet the Warburg effect doesn't occur until much later in the disease. Early prostate cancer is not detectable via an FDG-PET scan[22] which is able to detect cancer cells using the Warburg-effect (glycolysis) due to the increased uptake of glucose that occurs.
5. **The required mitochondrial DNA mutations are not present**: Mutations in mitochondrial DNA that are required to explain OXPHOS dysfunction are not present[23]. (mitochondria have their own DNA separate from the DNA found within the nucleus)
6. **Apoptosis is triggered with plant compounds**: The cell death mechanism referred to as *apoptosis*, is said to no longer work because mitochondria are defective – hence why a tumour forms. However, cancer cells trigger apoptosis via the mitochondrial cell death pathway when they are exposed to the natural compounds sulfurophane, bromelain[24] and even honey[25], highlighting that apoptosis is not defective, and indicating a suppressive mechanism is at play.

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14 *https://pmc.ncbi.nlm.nih.gov/articles/PMC3234981/*

15 *https://pmc.ncbi.nlm.nih.gov/articles/PMC2857128/*

16 *https://pmc.ncbi.nlm.nih.gov/articles/PMC5085139/*

17 *https://academic.oup.com/toxsci/article/200/2/369/7658967*

18 *https://www.sciencedirect.com/science/article/abs/pii/S0079610716300888*

19 *https://pmc.ncbi.nlm.nih.gov/articles/PMC5739687/*

20 *https://pmc.ncbi.nlm.nih.gov/articles/PMC9246372/*

21 *https://www.frontiersin.org/journals/cell-and-developmental-biology/articles/10.3389/fcell.2015.00043/full*

22 *https://pmc.ncbi.nlm.nih.gov/articles/PMC5474672/*

23 *www.frontiersin.org/articles/10.3389/fcell.2015.00043/full*

24 *www.ncbi.nlm.nih.gov/pmc/articles/PMC3633552/*

25 *https://www.sciencedirect.com/science/article/abs/pii/S0278691510007404*

These contentions led me to continue my research all the while keeping in mind the significance of explaining the Warburg effect.

The rise of the Cell Suppression Theory – new insight:

It wasn't long before I stumbled across my lightbulb moment. Upon reading a paper discussing how pathogens (disease-causing micro-organisms) invade cells, and how cells respond, I saw the phrase *'a Warburg-like metabolic response is triggered…'*. The significance of this was not lost on me. I realised the paper was indicating that infection can trigger the same Warburg effect found in cancer. It highlighted that the Warburg effect is an anti-infection response.

My immediate thought was: what if the pathogen succeeds in sustaining itself within the cell, known as an intracellular infection, does the Warburg effect persist? Yes. It turns out that the Warburg effect remains until the pathogen is eliminated. I realised this could explain the Warburg effect in cancer. My attention then turned to determining whether intracellular pathogens can and do suppress cell death mechanisms. Again the answer was yes, they effectively hijack cellular machinery to improve their chances of surviving within the cell by preventing the cell from committing cell death. This was the turning point in my thinking, because at first glance, an intracellular infection of this sort could theoretically generate a tumour by preventing cell death, and sustaining the proliferative state of the Warburg effect. This nicely aligned with my previous thoughts on cell mechanisms being suppressed rather than irreversibly damaged.

I was always intrigued by studies where plant-based compounds would cause cancer cells to commit cell death. This was intriguing because this mechanism should no longer be operational according to other theories. It is said that cancer arises because apoptosis is defective and cannot occur. However, these plant-based studies challenge this cell malfunction narrative. Reconsidering this evidence through the lens of suppression, with pathogens at its core, enabled me to provide the first coherent explanation for why plant compounds selectively triggered apoptosis in cancer cells. The answer: their antimicrobial properties kill the pathogen that has hijacked the cell to generate the disease. Killing the pathogen relinquishes control back to mitochondria allowing the cell to trigger the cell death mechanism in the normal way, which it does due to the significant damage it has sustained.

No better is this illustrated than with honey and silver. Silver selectively kills cancer cells by triggering the cell death mechanism – but silver is not required or utilised by the body, it is not broken down by cells for nutritional purposes. Its only property possible of explaining this reaction relates to its well-documented ability to kill pathogens – silver is highly anti-microbial. This is why the water tanks of the International Space Station are lined with silver, and why silver is incorporated into medical devices and can be used to aid wound healing. By killing the pathogen suppressing the cell death mechanism, silver allows the cell to regain control and initiate the process.

Honey is even more intriguing. The shift to the Warburg effect in cancer forces the cell to consume approximately 18 times the amount of glucose to that of

a healthy cell using OXPHOS, this is because glycolysis generates less energy per glucose molecule processed – so a cancer cell using glycolysis for energy, needs to utilise much greater quantities of glucose in order to generate the energy required to operate the cell. In other words, cancer cells feed on glucose – numerous studies confirm this. Honey, which is primarily sugar (50% fructose and 30% glucose), should fuel cancer growth, however it paradoxically kills it through the mechanism of apoptosis – a mechanism said to be broken. Cancer cells in a petri dish are grown on glucose so why does the glucose in honey encourage cancer cells to self-destruct? Aside from its potential cancer-feeding properties, honey is highly antimicrobial, and like silver, is used in wound healing to control for infection. Could the antimicrobial properties of honey explain its ability to trigger apoptosis and kill the cancer cell, when in effect, its high glucose content should be fuelling the growth of the disease?

Viewing cancer through the lens of infection enables us to start making sense of the disease. We now know that infection triggers the all important Warburg effect, but how exactly do pathogens explain this process, what is actually happening? This is where it gets interesting and is best described through Dr Robert Naviaux's *Cell Danger Response* model (CDR). His model describes how cells react to danger (toxins, infection etc…). Upon detection of danger the cell cycles through three phases:

- **CDR-1** inflammation, with a focus on combatting pathogens or removing toxins – danger removal.
- **CDR-2** proliferation, with a focus on tissue repair.
- **CDR-3** differentiation, returning the cell back to a calm homeostatic state.

Under this model, I propose that cancer arises because the cell is stuck between phases CDR-1 (the pathogen isn't eliminated), and CDR-2 (the need to repair chronic damage). This results in a persistent Warburg effect, and cells that are locked into a proliferative state of repair, with an inability to commit cell death as this defence mechanism is also being suppressed by the pathogen – this explains the development of a tumour.

Now here's the most interesting part described by Dr Naviaux's model. Upon the initial threat of danger, or in this case, infection, cells enter the CDR-1 phase. This is where mitochondria **intentionally** suppress OXPHOS. The emphasis here is that this process is intentional. Mitochondria suppress OXPHOS in order to divert oxygen use away from generating energy, to combatting the pathogen by creating *Reactive Oxygen Species* (ROS) – ROS can be viewed as bullets from a gun. These oxygen free radicals are essentially used to damage the pathogen – this is a key attack mechanism also enacted by immune cells.

With no energy being created through the now temporarily suppressed OXPHOS pathway, glycolysis is upregulated (the backup energy pathway of glucose fermentation is upregulated) to supply the energy needed to combat the pathogen, even though oxygen is available. These are the conditions referred to as the Warburg effect. The question is, did Otto Warburg mistakenly view this intentional suppression of OXPHOS as a defect in OXPHOS operation? Did he misinterpret the reason for this energy shift all those years ago?

Upon pathogen elimination cell repair mechanisms will complete, allowing

the cell danger response to cycle through CDR-2, the repair phase, to CDR-3 and ultimately revert back to homeostasis, where normal operation resumes – in this scenario OXPHOS becomes operational again. However, under conditions where the pathogen is not eliminated, the cell will remain in this CDR-1 phase where OXPHOS continues to be suppressed and glycolysis is upregulated to help combat the pathogen.

As the battle is sustained for far longer than any normal infection, high levels of ROS are produced leading to damage to many cell components including mitochondria and DNA within the nucleus. Lactic acid accumulates too, causing excess damage to surrounding tissue, which then initiates cell repair signalling. This chronic inflammatory state increases the need and signalling for repair mechanisms to be instigated. And so the cell becomes stuck between the proliferative cell repair phase of CDR-2 and the pathogen elimination phase of CDR-1. As a result, excess cell growth occurs leading to a tumour. This process nicely explains how the novel intracellular infection I propose, can instigate the development of a tumour.

To cut a long story short, after I started viewing cancer through the lens of infection, particularly fungal infection, I was able to explain all of the remaining hallmarks. In fact, during my research I identified at least 20 additional features associated with the disease, many of which remain unexplained by other theories. I then went on to explain all 20 and more besides, indicating that while all roads to cancer pass through mitochondria, they all appear to lead back to the influence of fungal pathogens. All this is explained in detail within my book, supported by peer-reviewed evidence. In fact, to quickly summarise, the following table shows that many of the features found to occur in cancer, also occur as a direct result of fungal infection. The left column in the following table depicts key features that we see occurring in cancer. The right column depicts key features that arise from fungal infection. That so many common features of cancer align with the symptoms generated by fungal infection is striking and very telling:

Fungal infection/cancer comparison table:

Cancer characteristics	**Link to fungal infection**
The Warburg-effect occurs	The Warburg effect is an anti-infection response
Apoptosis is inhibited	Fungi inhibit apoptosis
Cell proliferation is upregulated	Fungal infection triggers cell proliferation
Succinic Acid is produced via mSLP	Fungal infection triggers Succinic Acid production via mSLP
TLR 2 activation	Fungal infection activates TLR 2
NfkB activation	Fungal infection activates NFkB
PI3K/ATK/mTOR activation (growth pathways)	Fungal infection activates PI3K/ATK/mTOR
MAPK activation (growth pathways)	Fungal infection activates MAPK
E-cadherin is downregulated	Fungi suppress E-cadherin to facilitate invasion

CDH1, APC, HER2, p53, BRAC1 cancer related genes	All of these genes facilitate intracellular infection when mutated
CYP1B1 is activated only in tumours	CYP1B1 forms part of an anti-fungal response pathway
Nagalase production in tumours is a marker of tumour development & progression	Fungi produce Nagalase to evade macrophages by suppressing *Macrophage Activation Factor*
M2 Macrophage phenotype (cell repair) is dominant in cancer	Fungal pathogens instigate an M2 Macrophage phenotype to evade immune elimination
Th1 response is suppressed (intracellular immune response)	Fungal pathogens suppress the Th1 response to evade intracellular detection
Th2 response is upregulated (extracellular immune response – parasite elimination)	Fungal pathogens encourage the Th2 response to evade intracellular detection
PD-L1 upregulation hides cancer cells from immune cells	Fungal pathogens upregulate PD-L1 on the cell surface to evade immune detection
MMP-9 is upregulated – associated with inflammation and metastasis	Fungal pathogens upregulate MMP-9 to facilitate infection and modulate inflammation
Galectin-3 is upregulated – enabling metastasis	Galectin-3 is an anti-fungal protein triggered to target and eliminate fungal pathogens
Lipid droplet accumulation occurs	Lipid droplet accumulation occurs during infection to protect Polyunsaturated fatty acids from peroxide destruction during the targeting of the pathogens using Reactive Oxygen Species
Effective drugs	Anti-fungal drugs show efficacy against a broad range of cancers – the anti-fungal drug Itraconazole is one of the best performing off-label drugs against cancer
Effective off-labels - Metformin, Lovastatin, Mebendazole, Ivermectin, Doxycycline etc...	Most off-label drugs effective against cancer are also anti-fungal.
Terminally diagnosed pancreatic cancer patient[26]	Cured by use of Itraconazole – an anti-fungal drug
Aykut et al (2019) pancreatic cancer study[27]	Malassezia fungi were found to drive tumour growth • Anti-fungal therapy reduced tumour by 40% and stopped it from growing. • Re-introduction of Malassezia fungi re-established tumour growth
The Mayo Clinic admits fungal infection and cancer appear one and the same – Dr Vikram MD[28]	Fungal infections create tumours that mimic cancer – impossible to tell apart

When viewed in this format, it is clear that the influence of fungal pathogens can

26 *www.ncbi.nlm.nih.gov/pubmed/25670260#*

27 *https://pmc.ncbi.nlm.nih.gov/articles/PMC6858566/*

28 *https://www.youtube.com/watch?v=7P56JbKCtZM*

explain all of these features associated with cancer, whereas mitochondrial dysfunction cannot. There are many more correlations. And yes, fungi are the primary pathogen that I propose are driving the disease. So the question is: are cancer cells upregulating all of these pathways for their own survival because they've developed a mind of their own, or, is it the fungal pathogen present within the cell, that generates all of these conditions to facilitate its ongoing survival?

The tumour-associated-microbiome:

For the *Cell Suppression Theory* to hold weight, fungal pathogens would need to be present within tumours, just like for the *Somatic Mutation Theory* to be correct we would need to see that the p53 gene and RAS gene are mutated in all cancers – which they aren't. Up until recently, and even now, oncologists claim that tumours are sterile, free of micro-organisms – that was the prevailing assertion. However, emerging evidence from Ravid Straussman et al (2022)[29], has uprooted conventional thinking by confirming that out of 35 cancer types tested, pathogens both bacterial and fungal, exist in all of them. In fact, each tumour harbours its own unique population of micro-organisms termed the *Tumour-Associated-Microbiome.*

Moreover, the latest evidence is uncovering that most areas of the body – once thought to also be sterile, such as the brain – are also colonised by micro-organisms. This lends further support to the *Cell Suppression Theory*, in that the mechanism (the pathogen driving it), is primed and ready to initiate the Warburg effect and the hallmarks of cancer when the opportune moment arises. By opportune moment I'm referring to chronic inflammatory conditions that exhaust the immune system and make epithelial cells vulnerable to pathogen invasion. This enabled me to explain the initiation of cancer, how it begins. With this in mind, is it any wonder that 85% of all cancers arise from epithelial cells, the type of cells that are first in line to come in to contact with fungal pathogens? These cells form a protective barrier against infection. Surely, if cancer was caused by random DNA mutations, a result of bad luck, it would arise randomly in all different cell types, not so consistently in one cell type, the cells that are most commonly at threat of fungal invasion.

Finally, and significantly, even the main scientist working on the *Metabolic Theory*, Professor Thomas Seyfried, has recently recognised not only that tumours contain intracellular pathogens, but that these pathogens can, and do, trigger the Warburg effect as proposed by my *Cell Suppression Theory*. In his own words he states: "*these microbes are facilitators of fermentation metabolism…*".

You can listen to Professor Seyfried's comments regarding how micro-organisms facilitate the Warburg effect on the *Finding Genius Podcast* and on *Professor Seyfried's own YouTube channel* by clicking these two links. This discussion about micro-organisms starts at around the 16 minute mark. It would seem that even proponents of the *Metabolic Theory* are now acknowledging the relevance of my *Cell Suppression Theory* and the undeniable influence of these microbes in cancer.

29 *https://pmc.ncbi.nlm.nih.gov/articles/PMC9567272/*

What does this mean for treatment?

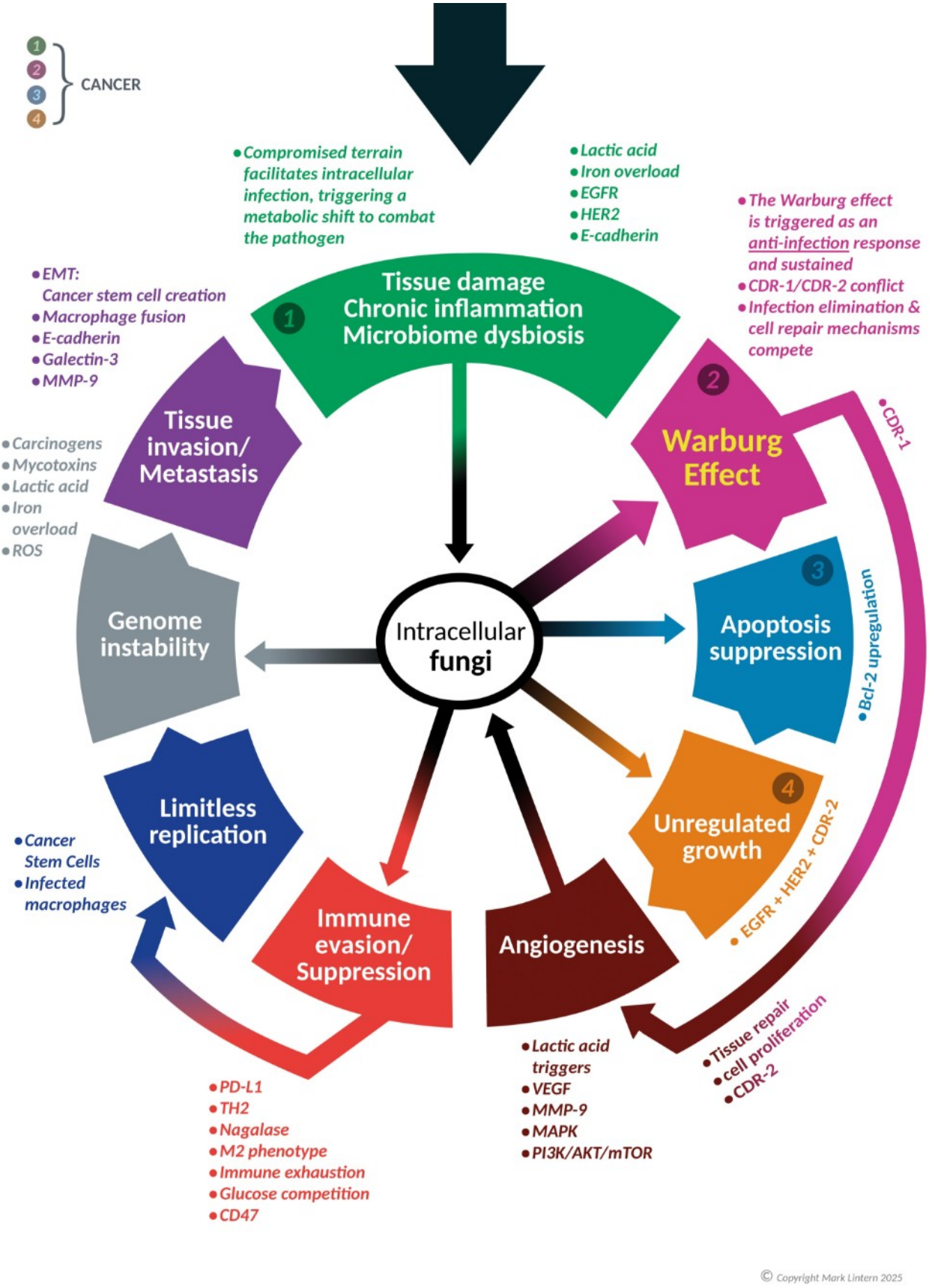
When viewed through this pathogen lens, all of cancer's seemingly odd behaviour make sense. Under this paradigm a new understanding can be reached that explains many of the aspects of the disease that remain unexplained, elucidating why current treatment approaches aren't working as well as we'd hope. Through this new understanding we have the justification for utilising additional treatments based on an anti-fungal approach. Indeed, studies into both anti-fungal and anti-parasitic drugs are showing efficacy against a broad range of cancers through an unexplained mechanism. A mechanism that the *Cell Suppression Theory* can provide a plausible explanation for. As we see with most off-label drugs that show efficacy, such as the anti-parasitic drugs mentioned, they all possess potent anti-fungal properties. Incidentally the metabolic approach of starving cancer of glucose and glutamine would also be beneficial because glucose and glutamine are the fungal pathogens primary food source. When we consider this, could the benefit we are seeing with the use of these other therapies be due to them inadvertently targeting the fungal pathogen that the *Cell Suppression Theory* identifies as the origin of the disease? In this respect, all that maybe needed to defeat cancer is to consciously adapt current treatment protocols by including additional treatments that can target fungi.

CONCLUSION:

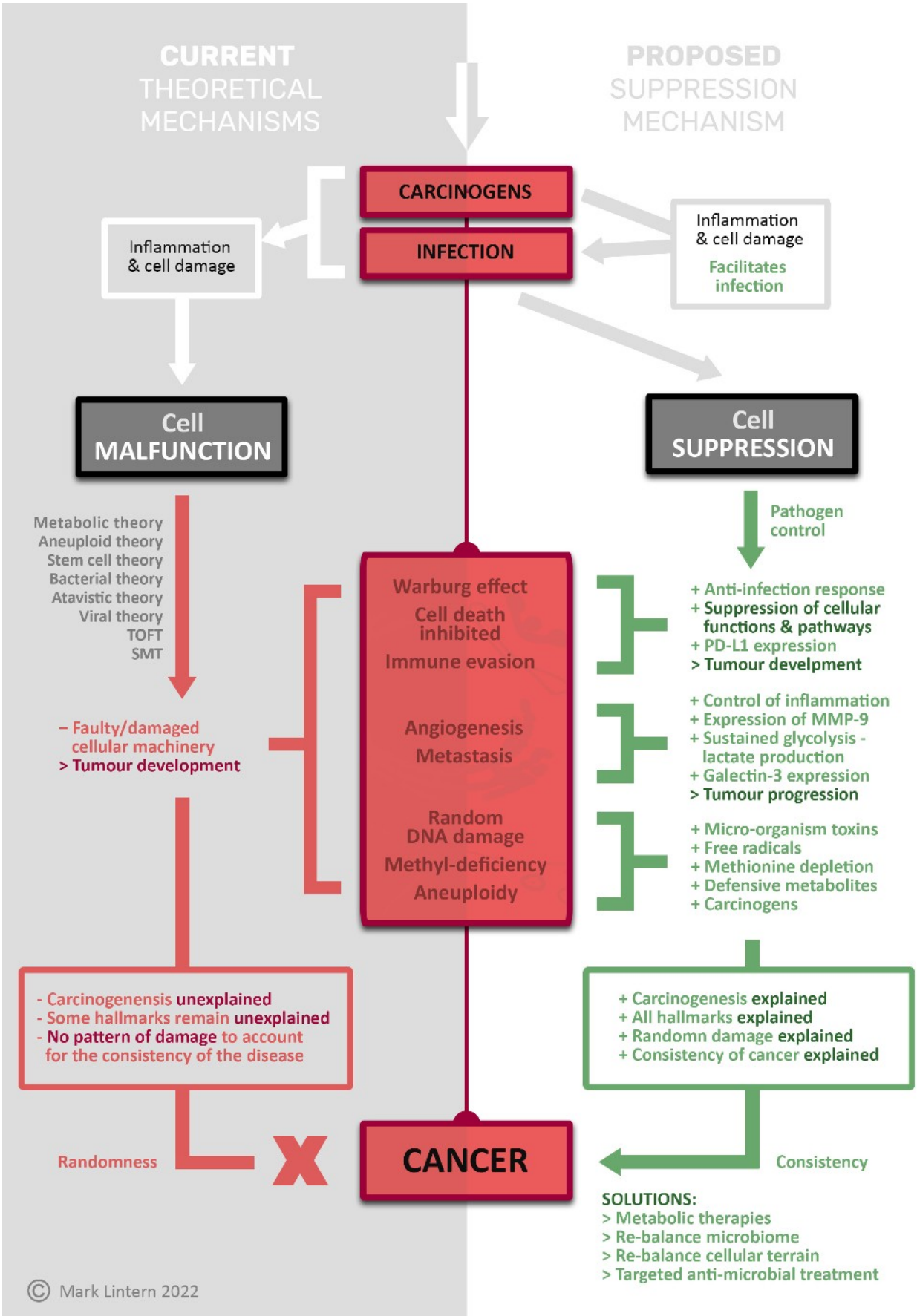
Abundant evidence supports the proposition that cancer is a cell-suppression disease caused by a select group of opportunistic fungal pathogens that take advantage of the conditions arising from chronic inflammation. Emerging data confirms the presence of a dysbiotic tumour-associated microbiome dominated by common intracellular pathogens, present in all cancers. It is clear that the Warburg effect is far more relevant than currently recognised by the medical authorities and by extension most oncologists. At the very least we should be treating cancer as a metabolic disease and taking into consideration cancer stem cells, the cellular terrain, the microbiome and emotional well-being, all of which are rarely considered by supporters of the *Somatic Mutation Theory*. At most, it can't hurt to also factor in the targeting of fungal pathogens, especially as anti-fungal drugs appear to be one of the best performing classes of off-label drugs against cancer, and that many also target metabolic pathways – food for thought.

If the *Cell Suppression Theory* resonates with you, please support my ongoing research and desire to help people with cancer by sharing this synopsis, and by purchasing my book '*THE CANCER RESOLUTION?*', in which this revolutionary theory is published. It's time for change, it's time to kick cancer's butt, let's make it happen together.

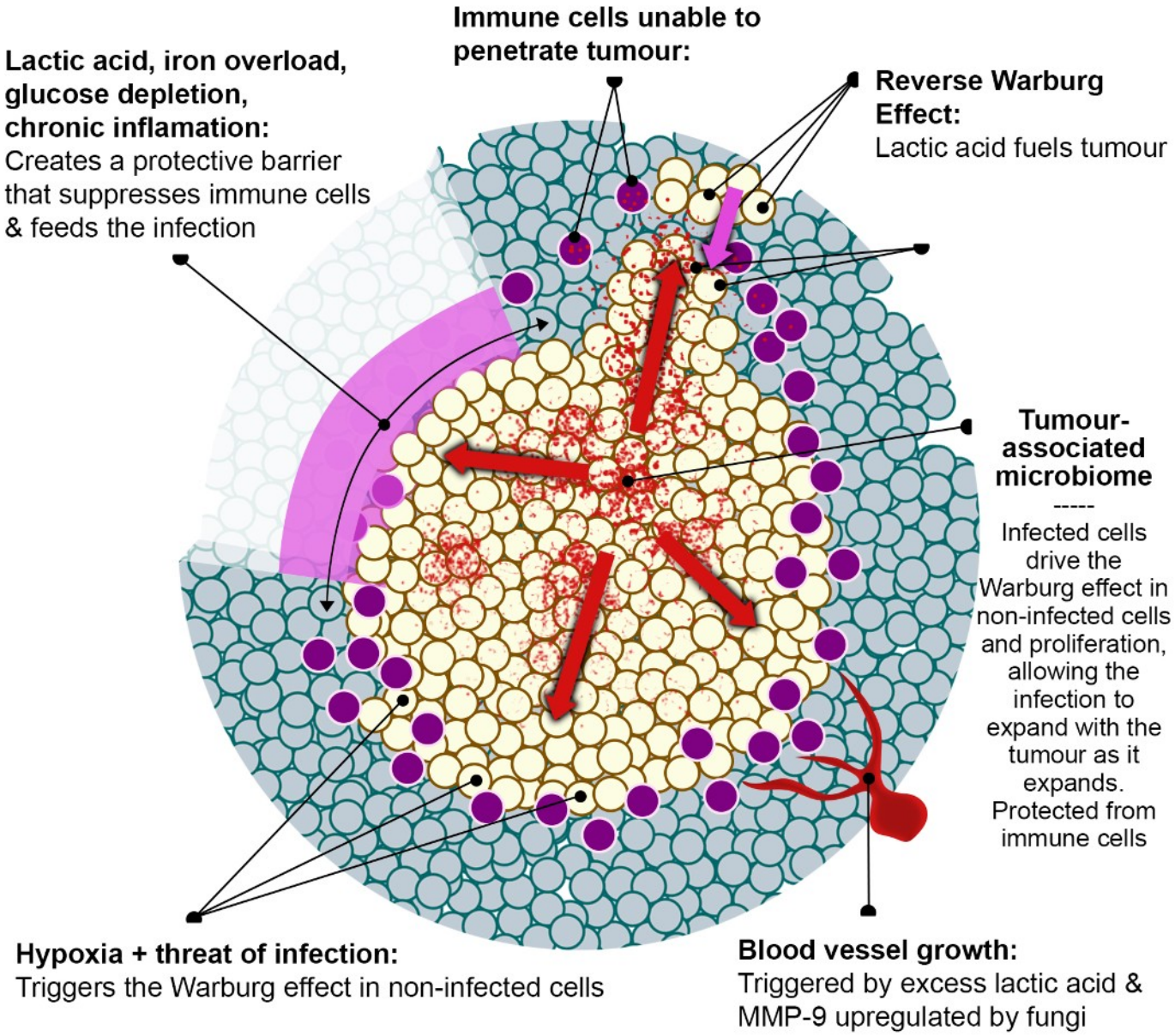
GRAPHICAL ABSTRACT – Hallmarks explained via fungal infection:



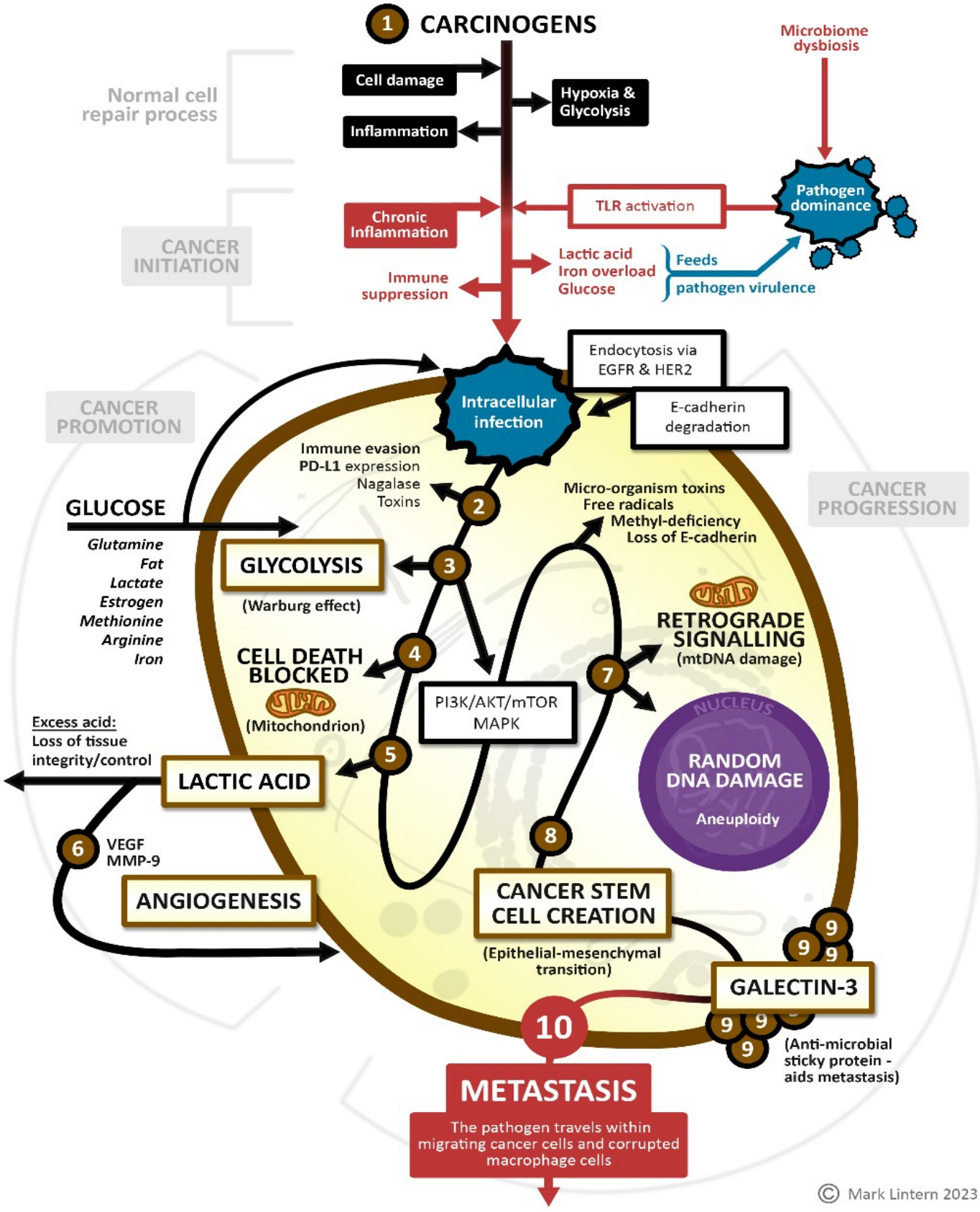
GRAPHICAL ABSTRACT – Cell Malfunction vs Cell Suppression paradigm:



TUMOUR COMPOSITION



GRAPHICAL ABSTRACT – Carcinogenesis explained:



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