CELL SUPPRESSION THEORY

Synopsis

A new theory of **cancer**

All established cancer theories, of which there are at least seven, 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.

What distinguishes this groundbreaking cancer theory, is its capacity to elucidate every fundamental hallmark, signifying that the causative factors have been identified. Through this novel framework, not only does cancer make sense, but an effective treatment approach may at last be defined.

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15th May 2023

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SYNOPSIS:

It has been said that cancer is over one hundred different diseases, but that conclusion has only been reached because we don't yet understand how or why it develops. Any disease will appear infinitely complex before it's mechanisms have been elucidated, and especially if it's symptoms have incorrectly been identified as its cause. Challenging the mainstream narrative, I have amassed a formidable body of evidence that indicates cancer is one disease that is triggered and mediated by a number of opportunistic intracellular pathogens. Taking advantage of chronic inflammation to sustain an infection, leads to the symptoms that we refer to collectively as 'cancer'.

While scientists have made great progress against many diseases, cancer has not fared so well. Despite a monumental effort, the mainstream *Somatic Mutation Theory* (aka the *DNA Theory*) hasn't delivered the results we were hoping for – the consistent pattern of DNA mutations we were expecting to identify for each cancer type, are not present. Instead, seemingly unrelated random mutations are found, even within the same tumour, which means the DNA Theory cannot account for the **consistency** of the disease. While many continue to claim that DNA mutations are responsible, and in the medical literature this concept continues to be unscientifically presented as fact; the fundamental reality is that the origin/underlying cause of cancer is actually **unknown**, hence why there are a minimum of **seven** different cancer theories that co-exist – scientists do not agree on the mechanism driving the disease. If the driving mechanism of cancer had indeed been uncovered, we would have an effective treatment.

So, contrary to the view often presented, the mainstream DNA Theory that guides the majority of medical research and clinical treatment, remains **unproven** by virtue of the fact it is unable to explain the vast majority of the Hanahan and Weinberg Hallmarks – the parameters by which the accuracy of all cancer theories are measured. There are 10 officially accepted hallmarks shared by all solid cancers. A theory capable of explaining all 10 hallmarks is a theory that has likely identified a key aspect of cancer, if not 'the' mechanism responsible. To place this in context, the DNA Theory appears capable of only explaining two of these 10 hallmarks – random mutations cannot account for the vast majority of them. While I praise the efforts of researchers in their attempt to identify a solution despite this incompatibility, it should be noted that their hard work is being hindered by a seemingly unshakable devotion to an incomplete understanding of the disease that is in all likelihood, **incorrect**. Surely, we can't begin to create effective treatments until we have the evidence to show we have identified the underlying cause? To that end we must continue to question what we think we know, and remain open to new perspectives if we are to make sense of the complexity presented before us.

Challenging the current consensus, the *Metabolic Theory* stands as the most accurate mainstream cancer theory currently available when evaluated against the

Hanahan and Weinberg hallmarks. Proponents of the Metabolic Theory posit that an energy pathway within mitochondria, known as 'OXPHOS', becomes faulty. This alleged defect prevents mitochondria from generating energy which forces the cell to rely on it's backup energy pathway of 'glycolysis' – this abnormal energy metabolism associated with cancer cells has been termed the 'Warburg effect' because a German scientist called Otto Warburg discovered it. Through this damage, and a reliance on glycolysis for energy, the Metabolic Theory can explain at least 7 of these 10 hallmarks, indicating that faulty metabolism is a key hallmark that drives the disease. Professor Seyfried argues that the Metabolic Theory explains all 10, however, in my book I address why there is contention over three of them, and why the Metabolic Theory falls short of fully explaining them all.

Regardless, there is a strong case for offering metabolic treatments as standard care for cancer patients, as opposed to the genetic treatments currently offered by mainstream oncologists, which are developed from the concepts put forward by the DNA Theory – a theory that can only explain two of these 10 hallmarks. The immediate concern is that many in the field continue to claim that the DNA Theory is correct even though it remains an unproven theory, and that largely ineffective genetic-based treatments continue to be offered to patients under the questionable assertion that they are the most effective. Confirming this point, Professor Paul Davies and Dr David Agus, along with the *National Cancer Institute* in America, highlight that despite all of the promising new drugs that have been developed, life expectancy for metastatic disease has only been improved by 4 weeks on average. Moreover, when the studies that form the backbone of the DNA Theory were re-assessed, up to 80% of these foundational studies could not be replicated, meaning, they are incorrect; thus indicating that the DNA Theory is incorrect, a reality reflected in the data that shows a lack of efficacy in the mainstream treatments currently in use.

Recognising the shortcomings of placing all our faith in one unproven theory, leaders in the field continue to develop other theories to address the remaining aspects of the disease that the DNA Theory is struggling to account for. For instance, the Metabolic Theory has been complemented in recent years by the *Atavistic Theory* and the *Tissue Organisation Field Theory* (TOFT).

Together these theories have greatly advanced our understanding and provide additional treatment avenues that are potentially more promising than current DNA-based standard-of-care treatments. However, there is a caveat. As I alluded to a moment ago, while the Metabolic Theory acknowledges the Warburg effect (a reliance on glycolysis), there seems to be contention over the mechanism purported to be driving this feature of the disease. Professor Seyfried cites defective Oxidative Phosphorylation (OXPHOS) as the origin of cancer, however, the OXPHOS energy-creating pathway within mitochondria has been shown to be operational to varying degrees in many cancers, suggesting that this pathway isn't actually faulty. Moreover, cells of the body such as endothelial cells, quiescent fibroblasts and stem cells, that rely heavily on glycolysis for energy, do not become cancerous as a matter of normal function, suggesting that a switch to glycolysis isn't the driving mechanism.

Furthermore, cancer is rare in children with Barth Syndrome, despite harbouring the same defective mitochondria that appear in cancer, which indicates

that some additional factors are required for cancer to form. Indeed, studies into oncocytomas highlight this point succinctly. Oncocytomas harbour defective mitochondria with an inability to utilise OXPHOS – the condition that allegedly drives cancer, and yet rather than cancer, these cells form **benign** tumours. This suggests that operational mitochondria are required for cancer to form. Such evidence indicates that while abnormal metabolism is a key feature of cancer, the notion that defective OXPHOS is the origin, is in contention.

This is where identifying the underlying cause of cancer becomes interesting. It is clear that abnormal metabolism, or the Warburg effect, (represented as Hallmark 7 in the hallmark list) is not only a core feature of cancer that appears to drive the majority of hallmarks, but that the reason for this energy switch is still **unknown**. Given it's relevance, identifying the cause of the Warburg effect will likely lead us to the cause of cancer itself. My research indicates that this is the pivotal hallmark to explain, in order to identify the origin of the disease. Once identified, effective treatments can be realised. The reason why cancer is not yet fully understood, despite exorbitant resources being thrown at it, is due to the fact that the vast majority of resources are focused on genetic research, meaning that the significance of the Warburg effect has been overlooked by most scientists.

To this end, I have spent the last eight years investigating this link and collating the evidence for a plausible mechanism that is not only responsible for the Warburg effect, but for all of the other nine hallmarks. The sum of this evidence suggests that the entire process of carcinogenesis (the development and progression of cancer) can be reinterpreted through a different lens entirely. I have documented my findings to show that there is indeed, an additional explanation for the Warburg effect that has not yet been considered. Significantly, I have been able to explain all 10 Hanahan and Weinberg hallmarks of cancer – a first for any cancer theory. Furthermore, I also provide a unique explanation for at least 20 other cancer-related conditions, such as arginine auxotrophy, the reverse Warburg effect and chemotherapy resistance (see the RESULTS section below). This indicates that I have identified an overlooked mechanism that triggers and drives cancer, if not 'the' mechanism responsible for the disease.

Intriguingly, this new perspective does not rewrite how we treat cancer from a metabolic point of view, far from it. In fact, it encompasses all the very same treatments advocated for by the Metabolic Theory, but it does highlight the need to consciously target an **additional factor** that many metabolic treatments are often inadvertently targeting. All that may be needed to improve treatment efficacy is to make minor adjustments to the metabolic approach. Crucially, this additional therapy can be implemented immediately and in conjunction with conventional treatments, providing hope to all cancer patients and healthcare providers.

To elaborate, I would like to shift your perspective of the disease momentarily. Nearly all mainstream theories view cancer through the same lens – the notion that it arises from a malfunction within the cell due to damage. Such a malfunction is thought to develop within the genome, within mitochondria, or within the surrounding tissue leading to a loss of suppressive growth signals. It is this breakdown in cell functionality that allegedly drives the disease. For instance, the DNA Theory claims that mutated DNA genes are responsible, the *Aneuploid Theory* asserts that abnormal chromosome formation is the driver, whereas the

Metabolic Theory claims that faulty mitochondria trigger an energy switch that results in the conditions of cancer. All data, regardless of theory, is interpreted through this cell-malfunction lens, where the cell itself is ultimately to blame – free from its constraints, the cell develops a 'mind of its own'. One could argue that currently, only one overall theory of cancer exists – the *Cell Malfunction Theory* if you will, and that all mainstream theories are sub-theories within this paradigm. The contention between these theories lies in which part of the cell is thought to be faulty and therefore responsible. The problem for all of these theories, has been an inability to identify a pattern of damage that can account for the consistency of the disease, and by consistency I'm referring to the 10 hallmarks shared by all solid cancers.

What if the abnormal behaviour of a cancer cell is not a result of malfunction, but of suppression, where an external factor foreign to the cell influences cell death and growth mechanisms, leaving the cell no longer in full control?

I developed this cell suppression concept through acknowledging the significance of the Warburg effect, and after stumbling upon infection data from immunological studies that were unrelated to cancer. This data showed that a switch to a 'Warburg-like metabolism' occurred in response to pathogen invasion – to my astonishment, I learnt that the Warburg effect is instigated as part of an anti-infection strategy. Under these circumstances our cells **intentionally switch** to glycolysis, even in the presence of oxygen.[01 – 08] I later discovered Dr Robert Naviaux's work that explains in detail, how, and why cells trigger the Warburg effect in response to infection – my many years of research was beginning to align to highlight a potential protagonist. Here, hiding in plain sight is a known cause of the Warburg effect, overlooked in relation to cancer due to the common assertion that the disease results from faulty cell machinery. This led to possibly the most important question of all: what if the infection persists? A question that doesn't appear to have been considered in cancer circles.

In support of this concept, Ravid Straussman's pioneering work has illustrated that tumours – previously thought to be sterile – harbour intracellular pathogens and a tumour-specific microbiome that interfere with cell functionality and drug effectiveness. A pan-cancer analysis of 35 different cancers found intracellular pathogens to be present in all samples. In fact, scientists were able to distinguish a healthy person from someone with cancer, as well as their likely prognosis, based purely on the presence of particular pathogens.

While the notion of cancer resulting from infection is not new, this cell suppression concept is unique and has yet to be explored by scientists. Currently, around 20% of cancers are associated with infection, but not in a suppressive capacity; rather, micro-organisms are thought to damage the cell leading to malfunction – and it is this malfunctioning cell machinery that is ultimately thought to be driving the disease, not the micro-organism per se.

Challenging the mainstream paradigm, I'm proposing that it's the suppressive actions of the pathogen, and it's control over specific cell functions, such as cell death and cell growth mechanisms, that drives the disease, not the random damage inflicted by infection or carcinogens – the pathogen is an active participant. We now know that intracellular micro-organisms exist within all tumours, that pathogens actively suppress tumour-specific cell functions in order

to keep the cell alive so long as it's beneficial for their survival, that the Warburg effect is triggered as part of an anti-microbial mechanism, and that it is sustained until the infection is eradicated. Failure to eliminate the infection, as appears to be the case from Ravid Straussman's work, provides an explanation for cancer's sustained reliance on glycolysis even in the presence of oxygen.

Within this captured, suppressive state, cell damage is magnified and sustained, resulting in excessive damage to mitochondria and the DNA present within the nucleus – the damage thought to be driving the disease. This triggers an epithelial-mesenchymal transition, where a regular cancer cell is transformed into a cancer stem cell, accounting for cancer's unlimited growth and resistance to treatment. Latent survival of pathogens within macrophages and lateral transfer of the pathogen between these immune cells, also helps to explain metastasis, immune evasion, the ability of cancer to cross the blood brain barrier and why macrophages appear to aid disease progression.

When viewed through this suppressive lens, all major aspects of cancer can be explained. For example, in terms of carcinogenesis, scientists are struggling to explain how the random DNA damage caused by so many different toxic carcinogens could lead to the consistency of cancer. This is certainly an impossible task given that randomness cannot generate consistency. To explain how the consistency of cancer can develop from the apparent randomness of carcinogen damage, we have to consider that there must be other consistent conditions generated by all carcinogens – and that these conditions have been overlooked. When we investigate further, this is indeed what we find. All carcinogens generate at least four consistent conditions: a weakened immune response, chronic inflammation, overproduction of lactic acid, and iron overload. This is a crucial point to acknowledge because these conditions further shed light on the underlying cause:

- A weakened immune system offers less resistance to infection.
- Inflammation renders cells more vulnerable to pathogen invasion.
- Lactic acid overproduction and iron overload feeds the infectious process and has the adverse effect of suppressing immune cells at the site of injury.

Essentially, carcinogens generate favourable conditions that facilitate infection of opportunistic pathogens, providing they are present within the local tissue microbiome. This toxic niche not only feeds these pathogens, but provides a protective environment within which they can thrive, because iron overload and lactate accumulation suppresses immune cell activity at the site of chronic inflammation. Consider also the triggering of the Warburg effect, which is a proliferative state, and the suppression of cell death mechanisms by the pathogen to keep the cell alive, and we have the promotion of a mass of unregulated cells with unregulated cell growth that can explain the initiation stage of carcinogenesis.

As the infection is slow-growing and encased within the protective boundary of the tumour, the patient won't be aware of the infection until the tumour grows large enough to be noticed. Assuming that cell malfunction is driving these conditions has meant that the majority of scientists have overlooked this nuanced infection strategy – that sustained infection is stimulating this abnormal cell expansion and metabolic profile.

Naturally, the increased absorption of glucose due to a reliance upon the Warburg effect, feeds the pathogen while depleting glucose within the surrounding tissue – glucose is the pathogen's primary fuel. This further suppresses the immune response at the tumour site because immune cells require glucose to operate. This provides an alternative explanation for why glucose feeds the disease – in sustaining the voracious demand of the pathogen, the monopolising of available glucose simultaneously depletes and weakens the immune response, all while the proliferative state of aerobic glycolysis continues to stimulate cell proliferation.

Acquisition of other nutrients by the pathogen, such as pyrimidines, purines, methionine and arginine, forces the cell to absorb higher quantities of these nutrients to replenish those that are lost. In effect, the cell is operating on autopilot having lost control of cell growth and cell death mechanisms. As with glucose, glutamine receptors are stimulated and increased, because glutamine is converted into many of these essential nutrients to replace them. This makes it appear as if the cell is choosing to adapt it's metabolism to survive, when in reality the cell is simply replenishing the nutrient imbalance caused by the pathogen.

Moreover, when glucose is in short supply, glutamine is utilised by the pathogen itself, and converted to glucose via the gluconeogenesis pathway within the pathogen, to ensure it's access to glucose is sustained. This can explain why glucose restriction as a therapy or even a state of ketosis, may not be effective in eliminating some cancers. The consumption of methionine by the pathogen explains why hypomethylation is a condition of pre-cancerous tissue and accounts for the random DNA damage that occurs in early-stage tumour development. Acquisition of arginine by the pathogen also explains the confusion surrounding arginine auxotrophy and why arginine starvation therapy can be both effective in some aggressive cancer's, but can also render the tumour even more aggressive.

Inhibiting these metabolic fuels has been shown to inhibit cancer cells. Proponents of the Metabolic Theory argue that this is because the fuels required by the cancer cell are restricted. The Cell Suppression Theory proposes that this is not just because the cell requires them to survive, but because the pathogen also requires these same fuels to sustain the infection – restricting them weakens the pathogen. Focusing on the needs of the pathogen, explains why the mechanism of apoptosis (cell death) – which is currently thought to be broken – is once again initiated when anti-microbial drugs or anti-microbial plant compounds (bromelain, sulforaphane, curcumin) are introduced to cancer cells. The pathogen is killed allowing mitochondria to regain control of this process leading the cell to choose to commit cell death – an outcome not thought possible in cancer cells that allegedly cannot, or do not wish to trigger this cell death mechanism. Why would a cancer cell that allegedly wants to survive, choose to kill itself when presented with the nutrients that would ordinarily sustain it? The apoptotic pathway that is assumed to be inoperative, was never faulty or broken, leading us to draw the conclusion that a suppressive mechanism is at play.

Viewing cancer through the lens of cell suppression enables us to re-interpret why certain treatments appear effective, and why the survival rate is so low with current standard of care. For instance: all four drugs used by the *Care Oncology Clinic* (a private oncology clinic in the UK) aimed at inhibiting metabolic pathways

in cancer patients, are also strong anti-microbial drugs. Metformin, Atorvastatin, Doxycycline and Mebendazole are all effective at killing the common pathogens involved, not to mention that the first two inhibit the fuels that these pathogens also require to sustain the infection. Hyperbaric oxygen therapy is also anti-microbial, as is 3BP (3-Bromopyruvate), Tamoxifen, Arimidex, Lovastatin and many more besides.

Regarding chemotherapy, while the free radicals generated by initial chemotherapy treatment can eradicate a large portion of the infection and reduce initial tumour size, chemotherapy often fails because it generates the same inflammatory conditions that go on to feed the infection – namely, immune weakness, chronic inflammation, overproduction of lactic acid and iron overload. Not to mention that the free radical production capacity generated by chemotherapy reduces with subsequent treatments, because its toxicity incapacitates the mitochondria that generate these defensive free radicals. The stimulation of cancer stem cells, and depletion of the protective microbiome also plays a key role in enabling pathogens to gain a foothold the longer chemotherapy is used. This explains why chemotherapy treatment can initially have a dramatic affect at reducing a tumour, but wanes substantially over time, and can become detrimental in the latter stages of treatment by stimulating cancer aggression. It initially reduces the infection, but unless the offending pathogens are eradicated, it will facilitate infection long-term, aided by the ability of these pathogens to upregulate antioxidants, further protecting them from pro-oxidant therapies that have a diminishing impact over time.

This cell suppression concept has enabled the explanation of all major hallmarks, and many other associated features that remain unaccounted for:

Aspects of cancer explained by cell suppression:

- All 10 Hanahan and Weinberg hallmarks
- All stages of carcinogenesis

The following additional features are also explained:

- Glucose, glutamine, lactate, fat, methionine, and arginine used as fuel by cancer cells.
- The Reverse Warburg effect
- Arginine auxotrophy
- Methionine auxotrophy – methionine dependence
- Hypomethylation
- Aneuploidy
- Chemotherapy resistance
- Iron's role in carcinogenesis
- The role of estrogen
- The role of nagalase
- The role of galectin-3
- Why antioxidant supplementation aids tumour development
- The role of CYP1B1 and the reason for its upregulation
- The role of macrophages in tumour progression
- The role of myeloid-derived suppressor cells in tumour progression

- The reason for T-cell suppression
- Why cancer is primarily a disease of old age
- Why cancer incidence is increasing
- Why childhood cancers exist
- Why cancer appears to run in the family

Regarding treatment: evidence supports a metabolic approach, detoxification of the cellular terrain, and re-balancing of the microbiome in conjunction with the addition of a targeted and powerful anti-microbial solution. As Ravid Straussman alludes to, all that may be required to improve current metabolic therapies is to also make a conscious effort to target a specific type of pathogen known to be present within the tumour mass. Data indicates that such a solution would work synergistically to target the dominant infection and render the cellular terrain hostile to the infectious agents. Theoretically, once control over the cell has been relinquished through the death of the pathogen, mitochondria are free to once again instigate apoptosis in severely damaged cells, resulting in tumour regression. Providing the body with the initial help it requires allows for the self-healing process to complete, assuming a supportive diet and lifestyle is pursued.

In my book 'THE CANCER RESOLUTION?', I identify which pathogen type my research indicates is the underlying cause of cancer. It's important to note that other micro-organisms are involved, however, I posit that they enable one type of pathogen to dominate and drive cancerous conditions.

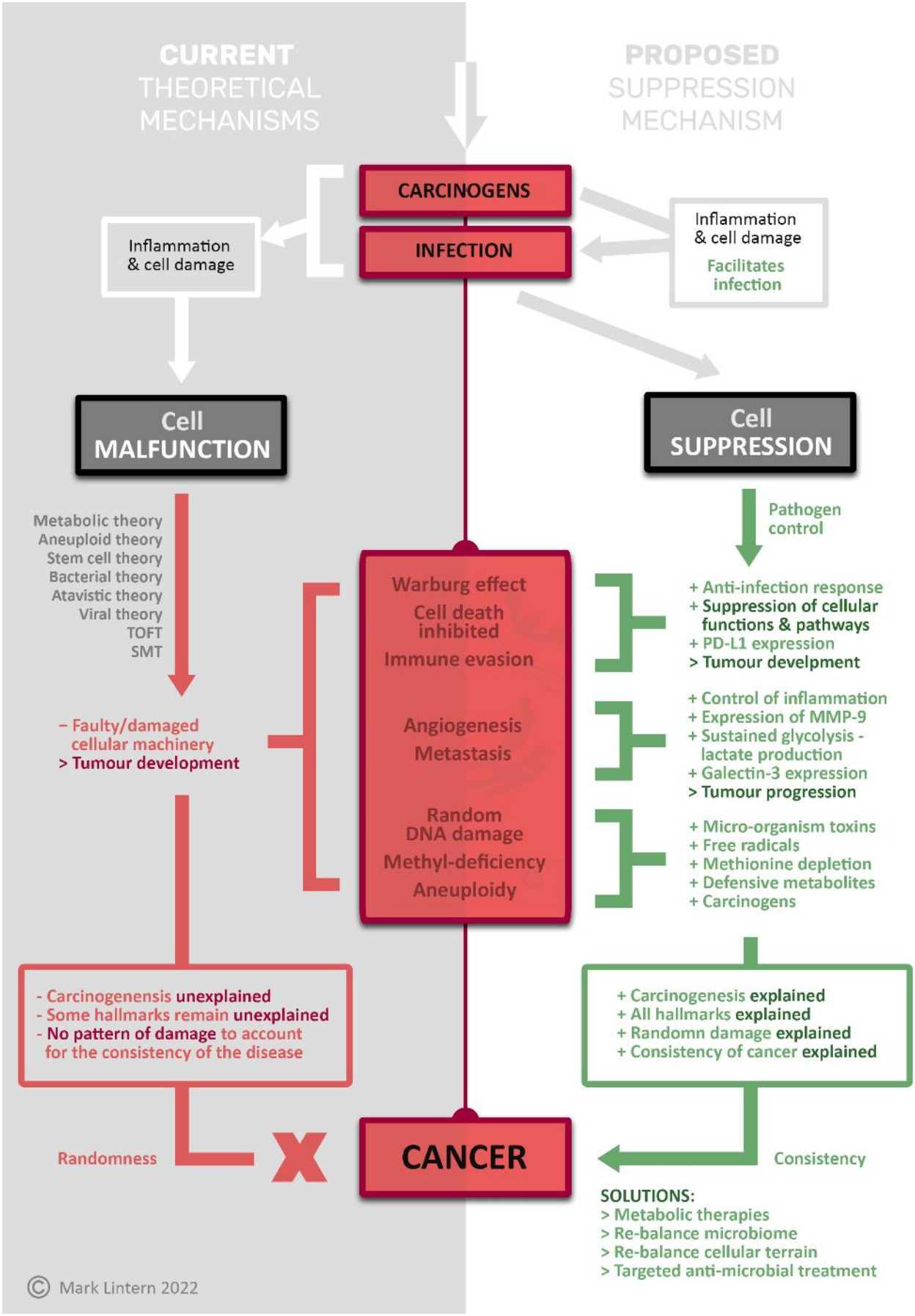
CONCLUSION:

Abundant evidence supports the proposition that cancer is a cell-suppression disease caused by a select group of opportunistic 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 pathogens.

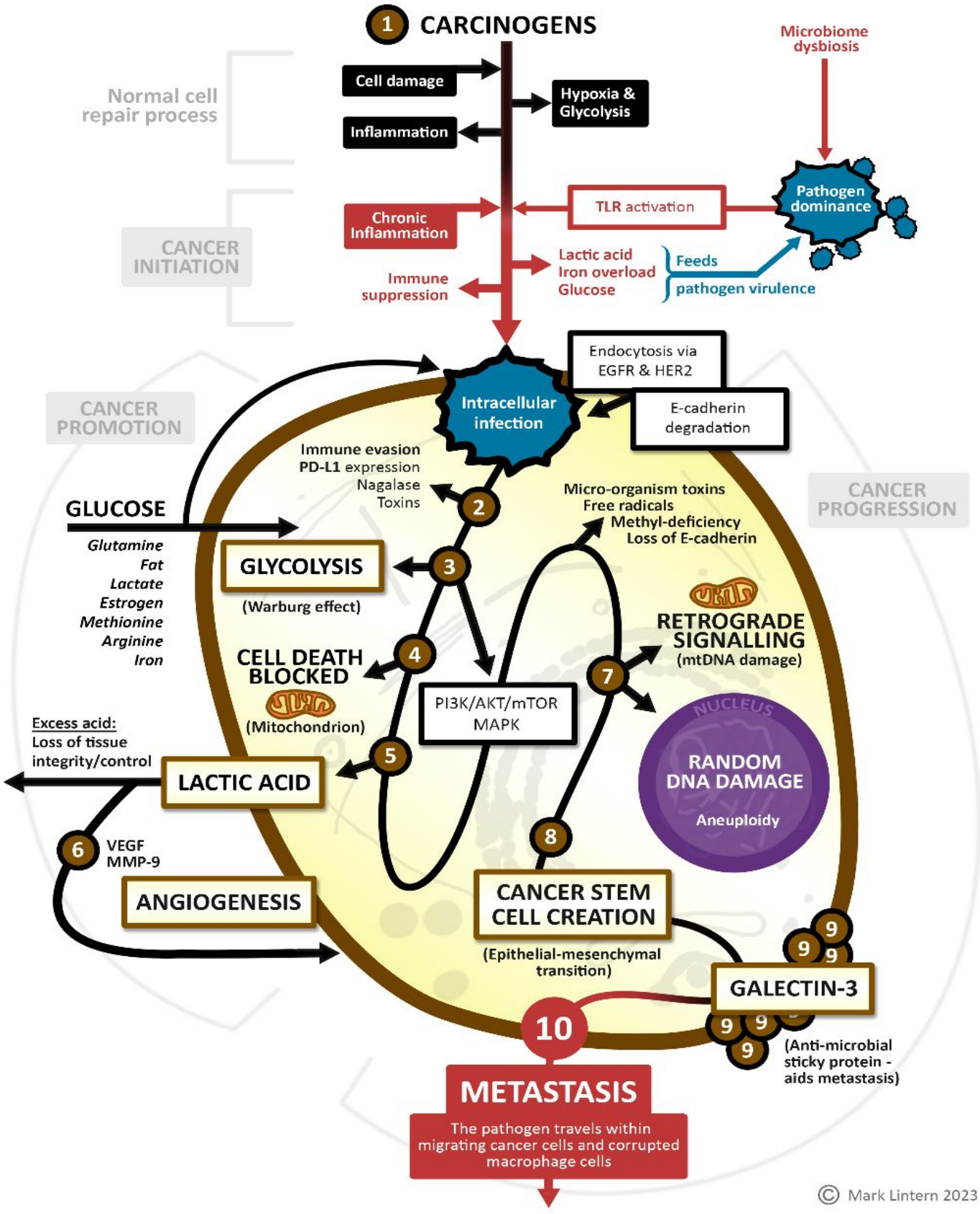
When viewed through the traditional 'cell malfunction' lens, it becomes impossible to identify the cellular mechanism(s) responsible for the odd behaviour expressed by the cancer cell because the cell itself is not at fault. Rather, the odd behaviour exhibited by the tumour – that cannot be attributed to any particular cell damage – is a reflection of the parasitic behaviour of the intracellular pathogen(s) present within the tumour mass. This explains why the Somatic Mutation Theory cannot identify cancer-specific mutations, and why the *Cancer Genome Atlas* data shows that mutations appear random – these mutations are symptoms resulting from the infectious process, ongoing pathogen interaction, and the initial exposure to carcinogens that enabled this opportunist to take root.

The abundant evidence supporting a cell-suppression mechanism for carcinogenesis, in combination with its ability to explain all major hallmarks of the disease, makes it clear that further investigation is warranted to determine the validity of this premise. Discussing its merits openly amongst experts in the field will allow it to receive the attention it deserves, and provides the opportunity for it to improve our understanding of cancer, and hopefully the survival outcomes for patients.

GRAPHICAL ABSTRACT – Cell Malfunction vs Cell Suppression:



GRAPHICAL ABSTRACT – Carcinogenesis explained:



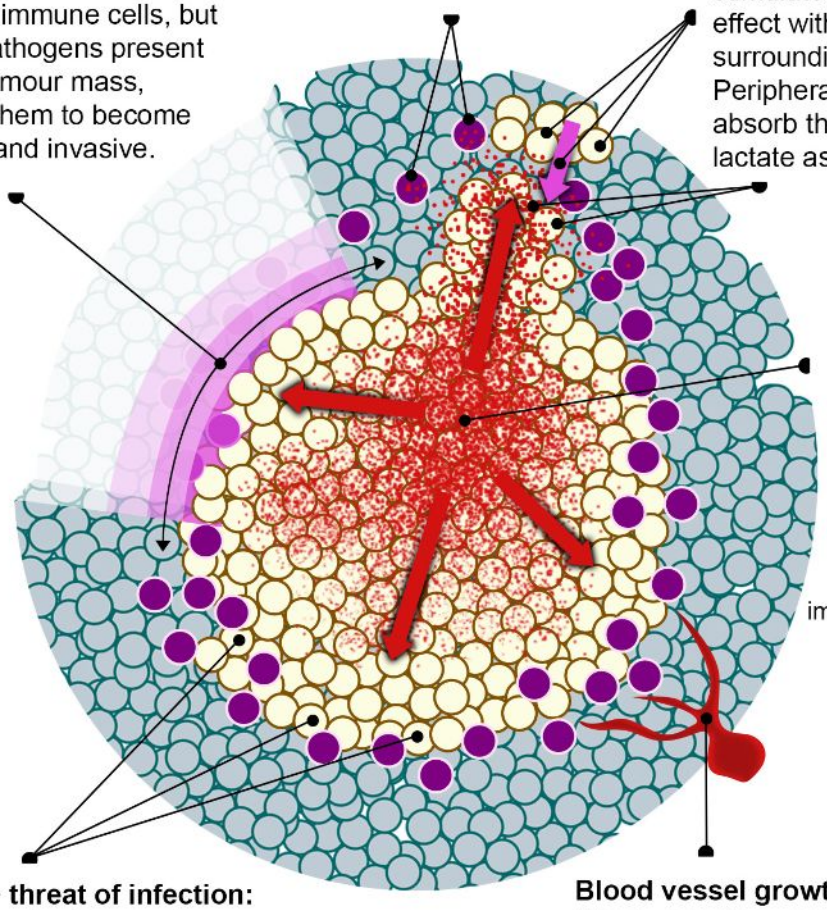
TUMOUR COMPOSITION

Cell suppression model

Lactic acid, iron overload, glucose depletion, chronic inflamation:
Creates a protective barrier around the tumour that suppresses immune cells, but feeds the pathogens present within the tumour mass, stimulating them to become aggressive and invasive.

Pathogen expansion:
Excess lactate fuels the infection which expands into surrounding tissue and dormant immune cells.

Reverse Warburg Effect:
Advancing infection stimulates the Warburg effect within the surrounding tissue. Peripheral tumour cells absorb the excess lactate as fuel.



Tumour-associated microbiome

Cell death inhibited, cell proliferation instigated, ineffective immune response
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Cancer

Hypoxia + threat of infection:
Results in cells close to the infection, but not yet infected, instigating glycolysis, stimulating proliferation. It's only a matter of time before they become infected, and added to the tumour mass.

Blood vessel growth:
Blood vessels expand towards cells over-producing lactic acid. This corrosive environment, plus over-production of MMP-9, results in cells migrating into the bloodstream.

- Healthy tissue
- Immune cell
- Warburg effect / anti-infection response > Tumour cell
- Intracellular pathogens > Cancer cells

SYNOPSIS REFERENCES – infection and the Warburg effect:

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3. Jorge Domínguez-Andrés, et al. '*Rewiring monocyte glucose metabolism via C-type lectin signalling protects against disseminated candidiasis.*' PLOS Pathogens. 2017. doi.org/10.1371/journal.ppat.1006632
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PRESENTATION SUMMARY – 12th February event:

During the online event '**Cancer Through Another Lens**', where my theory was critically analysed and validated by over 200 medical professionals, I talked through three presentations and the evidence that supports it. All three presentations, including the polls that scientists voted on, are available to view from my website – click the link to gain access to Mark's presentations. The content covered in each presentation is sumarised below, accompanied by the relevant references.

PRESENTATION 1

Strengths and weaknesses of prevailing theories of cancer:

Presentation one covers Marks story, establishes the benchmark parameters for assessing any cancer theory, and provides a detailed assessment of the Somatic Mutation and Metabolic Theories of cancer. The pros and cons of both theories are highlighted. A key hallmark is identified that, if explained, can point us towards uncovering the underlying mechanism and driving force behind the disease. The Hallmarks of cancer are re-organised to reflect this new interpretation.

PRESENTATION 2

Closing the gaps in existing theories:

The second presentation establishes a new interpretation of the Warburg effect, proposing a new mechanism of cancer causation via a 'cell suppression' paradigm, which is distinct from the mainstream mechanism of 'cell malfunction' that is currently the established view underpinning all mainstream theories. This is discussed through the work of Dr Robert Naviaux and Ravid Straussman, among others. A full explanation for the consistency of the disease is presented along with a coherent explanation for the initiation phase of carcinogenesis. The multi-factorial view of cancer is defined and challenged. The Hallmarks of cancer are amended to reflect this new paradigm.

PRESENTATION 3

Identifying the driving force:

Building upon the cell suppression paradigm, the third and final presentation lays down a compelling case for a specific driver of cancer, a unique and distinct target for treatment. An explanation for the promotion and progression phases of carcinogenesis is provided, resulting in the complete explanation of all 10 Hanahan and Weinberg Hallmarks. There is a final addition to the re-ordered hallmark list.

PRESENTATION REFERENCES – 12th February event:

PRESENTATION ONE:

SLIDE 03: Exploring the Origin of Cancer:

1. Thomas N. Seyfried. *'Cancer as a Metabolic Disease: On the Origin, Management, and Prevention of Cancer.'* 2012. ISBN: 978-0-470-58492-7

SLIDE 10: Exploring my Journey:

2. Thomas M. Ashton, W. Gillies McKenna, Leoni A. Kunz-Schughart and Geoff S. Higgins. '*Oxidative Phosphorylation as an Emerging Target in Cancer Therapy.*' Clin Cancer Res. June, 2018. (24) (11) 2482-2490. doi:10.1158/1078-0432.CCR-17-3070

SLIDE 19: Treatment success:

3. Professor Paul Davies. '*Cancer from a physicist's perspective: a new theory of cancer.'* New Scientist. National Cancer Institute. June 2013.

SLIDE 20: How effective are mainstream treatments?

4. Cliff Leaf, David Agus, MD, J. Craig Venter, Ph.D. *'How biology and big data converge in the medicine world.'* Fortune Magazine. 2015. https://www.youtube.com/watch?v=fDSQMeRgZHM
5. Anna Wagstaff. '*Jim Watson: DNA revealed the causes, it may never reveal a cure*.' Cancer world.net. September 2013. https://cancerworld.net/cover-story/jim-watson-dna-revealed-the-causes-it-may-never-reveal-a-cure/

SLIDE 21: Treatment success:

6. Dr. Andreas Eenfeldt, MD. '*Using a ketogenic diet to stop brain tumour growth.'* Diet Doctor. July 2016. https://www.dietdoctor.com/pablo-27-beats-cancer-using-ketogenic-diet – PlymouthHerald
7. Paul Hinson. '*Andrew Scarborough: the story of the man who beat cancer using a Paleo Keto diet*.' KetoForHealth.org. 2020.

SLIDE 22: DNA Theory analysis – reproducibility crisis:

8. Professor Paul Davies. '*Cancer from a physicist's perspective: a new theory of cancer.'* New Scientist. National Cancer Institute. June 2013.
9. '*How science goes wrong*.' The Economist. October 2013. https://www.economist.com/leaders/2013/10/21/how-science-goes-wrong

SLIDE 23: DNA Theory analysis – Cancer Hallmarks:

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11. Travis Christofferson. '*Tripping over the truth – The return of the metabolic theory of cancer illuminates a new and hopeful path to a cure.'* 2014. ISBN 9781500600310
12. '*Genomic Data Commons Data Portal.'* National Cancer Institute. February, 2021. https://portal.gdc.cancer.gov
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SLIDE 24: DNA Theory analysis – Summary:

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