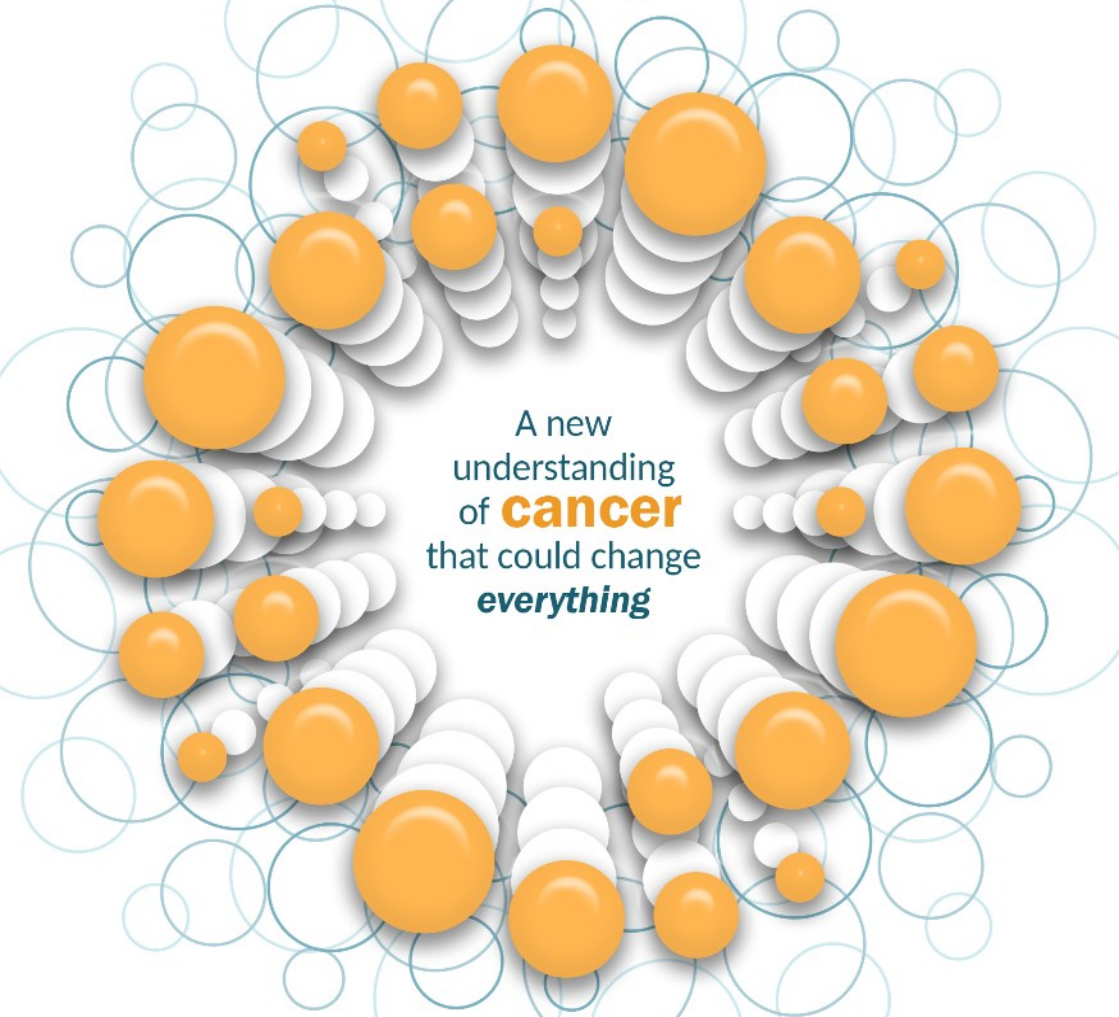
CELL SUPPRESSION THEORY

# Synopsis



A new understanding of **cancer** that could change ***everything***

By MARK LINTERN

# SYNOPSIS

## New hope for improved cancer prevention and patient survival

Most people are surprised to discover that cancer has more than one theory. In fact, there are at least nine different scientific explanations for how the disease may develop, highlighting a critical point: the underlying cause of cancer remains unconfirmed, unproven and unknown. Yet the vast majority of patients — and even many oncologists — are never told this. Instead, almost all cancer treatment is built on a single dominant theory that is assumed to be correct: the *Somatic Mutation Theory* — the notion that cancer is caused by genetic mutations.

But here's the shocking truth: despite over sixty years of research, billions of dollars invested, and countless drugs developed, this theory has not delivered the results we desperately need. On average, new cancer drugs add just two months of extra life for patients with advanced disease.[1] For families hoping for a cure, that is devastatingly inadequate.

This gap between expectation and outcome has left patients confused, fearful, and searching. In the United States, up to 70% of cancer patients turn to complementary or alternative therapies.[2] In the UK, the figure is nearly half.[3] They are not doing this because they distrust science — they do it because they feel abandoned by it. With limited results from conventional treatments, people look for alternatives, but without a reliable guiding framework to help them navigate which approaches hold real potential and which are misleading, they often end up lost in a confusing world of conflicting claims that can reduce survival outcomes.

What if there was a way to cut through this confusion? What if there was a science-based framework that helped both patients and doctors identify the treatments most likely to work, based not on guesswork or

1 doi:10.1001/jamaoto.2014.1570

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

3 doi: 10.7861/clinmedicine.13-2-126

hype, but on the actual accuracy of each cancer theory? That's what my work — and my book, ***The Cancer Resolution?*** — sets out to provide.

## Why cancer theories matter

Theories aren't just academic. They shape treatment. Every cancer drug ever developed began as an idea rooted in a theory about what drives the disease.

- If cancer is caused by **genetic mutations**, then the logical treatment is to target those mutations with drugs.
- If cancer is caused by **faulty energy production** (as the Metabolic Theory suggests), then treatments focus on addressing metabolism.
- If cancer begins with **stem cells**, or with **disrupted tissue organisation**, then therapies are designed to target those mechanisms.

The problem is simple: **if the theory is wrong, the treatment is unlikely to succeed.** Scientists now agree there are 10 defining "hallmarks" of cancer — which are the consistent features that every solid tumour shares, such as uncontrolled growth, immune evasion, and resistance to cell death. These hallmarks are the measure of any theory — the more that can be explained the more accurate the theory is deemed to be. Of note: the established Somatic Mutation Theory (SMT) struggles to explain more than two of these hallmarks, while proclaiming cancer a genetic disease as if this were already a proven fact. That's like trying to solve a jigsaw puzzle and declaring you've finished it, when most of the pieces are still missing. This is why, despite decades of effort and billions invested, the dominant SMT has failed to deliver a cure. It can only explain a small fraction of what cancer actually is, suggesting it has not only misidentified the driving mechanism, but it's treatments are targeting the wrong feature of the disease.

Yet this is the theory that almost every oncologist subscribes to. It is the theory that forms the basis for the treatments you receive in standard of care, simply because it is the one most heavily funded and most widely taught in medical school. In other words, by following the advice of

mainstream oncologists a patient is unwittingly agreeing to be treated based only on the parameters set forth by the SMT (the notion that cancer is a genetic disease), even though most of the puzzle pieces are missing.

## The shortcomings of the dominant theory

The travesty appears to be that patients are advised to follow established treatments and are not informed of other treatment options associated with other theories, this is despite the evidence painting a troubling picture that challenges the validity of the SMT – remember, genetic mutations are said to be driving cancer, yet evidence shows that:

1. **Cancerous DNA mutations appear in healthy tissue without the disease developing.**[4]
2. **Cancers have been found without showing any of the required driver mutations.**[5] [6] So what's driving cancer in those cases?
3. **On average only 1.7 driver mutations were found**[7] — there are an insufficient number of mutations to explain how cancer forms.
4. **The mutations found are random, not consistent.**[8] This randomness cannot explain the remarkable consistency of cancer across patients and cancer types.
5. **Transferring cancerous DNA into healthy cells fails to generate cancer** — indicating that DNA mutations are not responsible.[9]
6. **When cancerous cells are re-located into healthy tissue they revert to normal**[10] despite these mutations being present, confirming some alternative feature must be responsible.
7. **Up to 80% of cancer studies cannot be reproduced**[11] — indicating the majority of evidence supporting the SMT is incorrect.

---

4 *doi: 10.1016/j.trecan.2019.07.007*

5 *doi.org/10.1371/journal.pbio.3003052*

6 *doi.org/10.1038/nature13061*

7 *doi.org/10.1073/pnas.1803155115*

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

9 *Cancer Res (2003) 63 (11): 2733–2736.*

10 *doi.org/10.1016/j.pbiomolbio.2016.07.004*

11 *https://www.sciencenews.org/article/cancer-biology-studies-research-replication-reproducibility*

This and more, indicates that genetic mutations are not driving the disease, but are symptoms of another cause. Despite all this, the SMT continues to dominate medical practice. When 95% of cancer research money goes into supporting one theory, consensus naturally follows. But consensus is not the same as truth or fact, as Michael Chrichton highlights:

> *'Historically, the claim of consensus has been the first refuge of* ***scoundrels****; it is a way to* ***avoid debate*** *by claiming that the matter is already settled...*
>
> *...Consensus is the business of* ***politics****. Science, on the contrary, requires* ***only one investigator who happens to be right, which means that he or she has results that are verifiable by reference to the real world****…Consensus is invoked only in situations where the science is* ***not solid enough****...*
>
> *...The greatest scientists in history are great precisely because they* ***broke with the consensus****. There is no such thing as consensus science. If it's consensus, it isn't science. If it's science, it isn't consensus. Period.'*
>
> ***Michael Chrichton*** *MD*

This matters because if we are aiming at the wrong target, we are unlikely to hit the disease where it counts — a reality reflected in the poor five-year survival outcomes we see for each cancer type at later stages of the disease:

| Cancer Type | *5-year survival – stage 3 | *5-year survival – stage 4 |
|---|---|---|
| Liver | ~15% | ~5% |
| Pancreatic | ~16% | ~3% |
| Brain/central nervous | ~20% | ~5% |
| Esophageal | ~22-35% | ~5% |
| Stomach | ~25-30% | ~6% |

| | | |
|---|---|---|
| Lung | ~29% | ~5% |
| Ovarian | ~39% | ~17% |
| Leukemia (varies) | ~40-60% | ~10-15% |
| Endometrial | ~45-60% | ~17% |
| Bladder | ~46% | ~5% |
| Kidney | ~53% | ~12% |
| Oral cavity | ~54% | ~20% |
| Cervical | ~57% | ~19% |
| Melanoma | ~63% | ~20% |
| Non-Hodgkin Lymphoma | ~65-75% | ~30% |
| Colorectal | ~71% | ~16% |
| Breast | ~87% | ~27% |
| Prostate | ~90% | ~30% |

## The framework patients need

So what can we do instead? This is where my work introduces something new: **a simple, science-based framework that patients and oncologists can use to navigate cancer treatments with greater clarity.** The principle is straightforward: **the accuracy of a theory determines the potential effectiveness of its treatments.** The more hallmarks of cancer a theory can explain, the more likely it is to reflect the true origin of the disease — and therefore the more likely that treatments derived from it will succeed. This turns **cancer theory** into a **practical tool**. Instead of blindly following the dominant view, patients and doctors can assess which theories hold up against the evidence and which do not. From there, they can make more informed decisions about which treatment strategies deserve attention.

In support of the scientific method, many will rightly say that following evidence-based medicine is the safest path — and in principle, I agree. The problem is that most of this "evidence" comes through the narrow lens of the SMT, as genetic research is where most cancer funding is directed. When almost all funding, research, and drug development is tied to a single interpretation of cancer, the resulting evidence cannot help but be biased, because sufficient testing of other theories and treatment options hasn't occurred for an objective conclusion to be drawn. That's not truly objective evidence-based medicine — it's evidence constrained by one assumption. But what if that assumption is wrong? If the SMT fails to explain cancer in full, then the treatments built upon it are also limited. A more balanced approach would be to weigh the evidence across ***all*** credible theories, using it to identify which explanations best fit the disease and, in turn, which treatment strategies are most likely to succeed. That is the essence of a genuine evidence-based framework.

## The theories worth considering

When measured against the 10 hallmarks of cancer, three established theories stand out above the genetic model:

- **The Metabolic Theory** — which sees cancer as a disease of broken energy production. It explains at least seven hallmarks.
- **The Cancer Stem Cell Theory** — which highlights the role of specialised immortal cells that resist treatment and are responsible for unlimited tumour growth. At least five hallmarks are accounted for.
- **The Tissue Organisation Field Theory** — which shows how signal disruption in the cellular terrain can trigger disease. Again, five hallmarks appear to be explained.

These theories open new doors to additional treatment options. They suggest that cancer is not primarily a genetic disease at all, but something more complex — and potentially more treatable, if we approach it differently. Incidentally, treatments associated with the SMT damage the terrain, can stimulate cancer stem cells driving resistance, and damage mitochondria — effectively disrupting the very mechanisms that all of the

above theories claim are driving the disease. This is not to say that standard treatments don't have a part to play, they do, rather it's that our approach to treatment needs to be re-considered due to the nuance that's clearly involved — surgery, and damaging radio- and chemo-therapies should not be the only options available.

## A new paradigm: the Cell Suppression Theory

Building on this, my own research has led to what I call the **Cell Suppression Theory (CST)** — a new paradigm that could redefine how we see cancer. I argue that while the above three theories have identified key mechanisms driving cancer, they are unable to fully explain the process because they all suffer from the same fundamental flaw in reasoning, and that is this: these theories, including the SMT, assume that cancer results from *cell malfunction* — all conclusions are made through this restrictive lens. Instead of treating cancer as a disease of malfunctioning cells, the CST proposes that cancer is the result of *suppressed cells,* cells that have been hijacked by a foreign entity — a complete paradigm shift.

In simple terms, our cells are not broken or rogue. They are doing exactly what they were designed to do — but under the wrong kind of influence. Evidence shows that pathogens, especially fungal pathogens, can invade cells, hijack their machinery, and suppress their natural defences.

- They can **trigger the Warburg effect**, the abnormal energy shift seen in all cancers.
- They can **block apoptosis**, the self-destruct mechanism that normally prevents damaged cells from growing.
- They can **promote inflammation, cell growth, angiogenesis, and immune evasion** — all central features of cancer.

## How the CST explains the missing pieces of the cancer puzzle

The Metabolic Theory explains at least 7 of the 10 hallmarks for a very good reason. The Warburg effect that it describes, is a consistent feature of all solid cancers and is pivotal to the process. It explains how

cancer cells abnormally favour the backup fermentation energy pathway of 'glycolysis' instead of the primary oxygen-based energy pathway of 'OXPHOS', even when oxygen is available for OXPHOS to use. The reason proposed for this reliance on glycolysis? Mitochondria that create energy via OXPHOS using oxygen, have **malfunctioned**, this forces the cell to rely on the separate backup energy pathway of glycolysis – a corrosive lactic-acid generating energy system that facilitates cancer development when used to excess.

The game-changing paradigm I put forward with the CST recognises the significance of the Warburg effect but proposes a fundamental difference – that the Warburg effect occurs not because mitochondria have malfunctioned, but as a result of the mitochondria switching focus to combat an invasive fungal pathogen intent on hijacking the cell and its machinery. In this context, mitochondria intentionally suppress OXPHOS to repurpose the oxygen they would otherwise use for energy creation, to combat the fungal invader. This anti-infection strategy is well documented in the medical literature. The Warburg effect can also be explained as a response to infection. It's no coincidence that fungi and bacteria are found inside all cancerous tumours.

The main proponent of the *Metabolic Theory*, Professor Thomas Seyfried, acknowledges that intracellular pathogens exist within tumours, even stating that they can drive the Warburg effect. On the *Finding Genius Podcast* at around 15 minutes, he states:

> *"These microbes are facilitators of* ***fermentation metabolism****…"*

Fermentation metabolism in this context refers to the Warburg effect in cancer – the reliance on glycolysis, the backup energy pathway that produces lactic-acid. This subtle shift in thinking brings everything together, explaining key features of cancer that, up until now, have remained a mystery. As a result the CST is the first theory capable of explaining all 10 hallmarks. It doesn't discard the metabolic, stem cell, or tissue theories — it incorporates them, showing how they all fit into a bigger picture, and that includes the SMT.

And here's the hopeful part: **many of the treatments that could**

**address this mechanism already exist.** Anti-fungal and anti-parasitic drugs, metabolic therapies, and immune-supportive strategies are already available, some showing surprising effectiveness in studies. What the CST offers is the scientific explanation for why they work — and a roadmap for using them more effectively.

## Why this matters for patients

For patients, this changes everything. It means cancer is not an unfathomable mystery. It means there are rational, evidence-based ways to make better choices. And it means that, even if a cure remains elusive, survival and quality of life can be improved by targeting the right mechanisms. Patients no longer have to choose blindly between mainstream protocols, untested alternatives, or confusing blends of both. With the framework the CST provides, they can evaluate options more confidently, working with their oncologist instead of feeling powerless.

This is why Mark wrote the book *The Cancer Resolution?* — to bring this knowledge out of the laboratory and into the hands of those who need it most. While *The Cancer Resolution?* can aid oncologists make more informed treatment decisions, it has been specifically written with cancer patients in mind. The science is translated into simple patient friendly terms — enabling the general public to gain an understanding of cancer like never before, and in a manner that is clear, concise and easy to digest.

## Why This Matters for Oncologists

For oncologists, the CST is not a threat **but an opportunity**. It does not reject conventional treatments. Surgery, for example, remains one of the most effective interventions. Even chemotherapy can have value in certain contexts, such as at lower doses, as Dr Robert Gatenby's research has brought to light. Rather, the CST challenges oncologists in a positive way, encouraging them to consider that if outcomes remain poor, the theory driving those treatments may be incomplete.

By recognising cancer as a suppression-driven disease, oncologists gain a new rationale for revisiting therapies that have been overlooked or dismissed. This includes re-evaluating anti-fungal drugs, metabolic strategies, and combination approaches that could enhance existing

protocols. It is also a call to scientists and pharmaceutical companies that fund them, to create novel anti-fungal drugs that not only combat cancer with greater efficacy, but can also address the rising health threat of anti-fungal drug resistance that is already a silent pandemic of its own. Far from undermining medical practice, the CST strengthens it — by aligning treatment with the actual biology of cancer.

## A shared mission

Cancer is not just a patient's battle. Oncologists, researchers, families — we are all in this together. But for too long, progress has been stalled by narrow thinking. The Cell Suppression Theory opens a new path. It offers patients hope, and it offers doctors a framework grounded in science that deserves serious consideration.

If you are a patient, *The Cancer Resolution?* will help you take control of your treatment journey with clarity and confidence. If you are an oncologist, it will provide insights that could expand your clinical toolkit and improve outcomes for those in your care. It is a call to action, a framework for clarity, and a bridge between patients seeking hope and doctors seeking better answers. By exposing the shortcomings of outdated theories, introducing the unifying power of the Cell Suppression Theory, and empowering readers with a simple but profound roadmap, this work has the potential to change how we see cancer — and how we fight it

It is time to rethink cancer. It is time to bring light to areas science has overlooked. And it is time to work together — patients and professionals alike — to finally turn the tide against this disease. **It is time for a new resolution**.

## Significant cancer/fungal correlations worthy of note:

| Cancer characteristics | *Link to fungal infection |
|---|---|
| Chronic inflammation is a hallmark of cancer initiation | Fungi trigger and modulate inflammation influencing infection |
| The Warburg-effect / abnormal metabolism is a hallmark of all cancers | The Warburg effect / metabolic shift is an anti-infection response |
| Lactic acid overproduction and iron overload appear to facilitate cancer | Both feed fungal pathogens while suppressing the immune response |
| Apoptosis fails (cell death) | Fungi suppress apoptosis |
| Cell growth is unregulated | Fungal infection stimulates cell proliferation |
| TLR-2 is upregulated | Fungal infection activates TLR 2 |
| NfkB is upregulated | Fungal infection activates NFkB |
| PI3K/ATK/mTOR activation | Fungi activate PI3K/ATK/mTOR |
| MAPK activation | Fungal infection activates MAPK |
| E-cadherin is downregulated – this aids metastasis | Fungi downregulate E-cadherin as a strategy to increase cell invasion |
| CDH1, APC, HER2, p53, BRAC1 mutations increase cancer risk | All of these mutated genes facilitate intracellular fungal infection |
| CYP1B1 is an enzyme that is only activated in tumours | CYP1B1 forms part of an anti-fungal response pathway within the cell |
| Nagalase is produced only in tumours. It's aligned with tumour burden & suppresses macrophage immune cells | Fungi produce Nagalase to suppress macrophages via downregulation of Macrophage Activation Factor (GcMAF) |
| Succinic Acid production via mSLP (Glutamine fermentation) | Fungi trigger Succinic Acid production via mSLP (glutamine fermentation) |

| | |
|---|---|
| M2 Macrophages dominate tumour tissue (M2 = cell repair. M1 Macrophages = pathogen elimination) | Fungal pathogens suppress M1 & stimulate M2 Macrophages as a strategy to evade immune detection |
| Th1 response is suppressed (anti-infection response) | Fungal pathogens suppress the Th1 response to evade immune detection |
| Th2 response is upregulated | Fungal pathogens encourage the Th2 cell repair response to increase survival |
| PD-L1 is upregulated – hiding the cancer cell from immune detection | Fungi modulate PD-L1 during infection to evade immune detection |
| MMP-9 is upregulated – associated with inflammation and metastasis | Fungi upregulate MMP-9 to modulate inflammation & facilitate infection |
| Galectin-3 is upregulated – it is a sticky protein that enables metastasis | Galectin-3 is an anti-fungal protein triggered in the presence of fungi |
| Lipid droplet accumulation within the cytosol of the cell | Lipid droplet accumulation occurs during infection to protect PUFA's |
| Off-label drugs – Anti-fungals | Anti-fungal drugs show efficacy against a broad range of cancers |
| Off-label drugs – Metformin, Lovastatin, Mebendazole, Ivermectin, Doxycycline | Most off-label drugs that show efficacy are also anti-fungal as well as capable of modulating cancer metabolism |
| Case study: Terminally diagnosed pancreatic cancer patient | Cured/resectable tumour after using Itraconazole – an anti-fungal drug |
| Aykut et al 2019 – pancreatic cancer study | - Anti-fungal therapy reduced tumour by 40% and stopped it from growing.<br>- Re-introduction of Malassezia fungi re-established tumour growth |
| The Mayo Clinic, Dr Vikram MD | Fungal infections mimic cancer |

**Fig 1 – Cell Malfunction vs Cell Suppression**

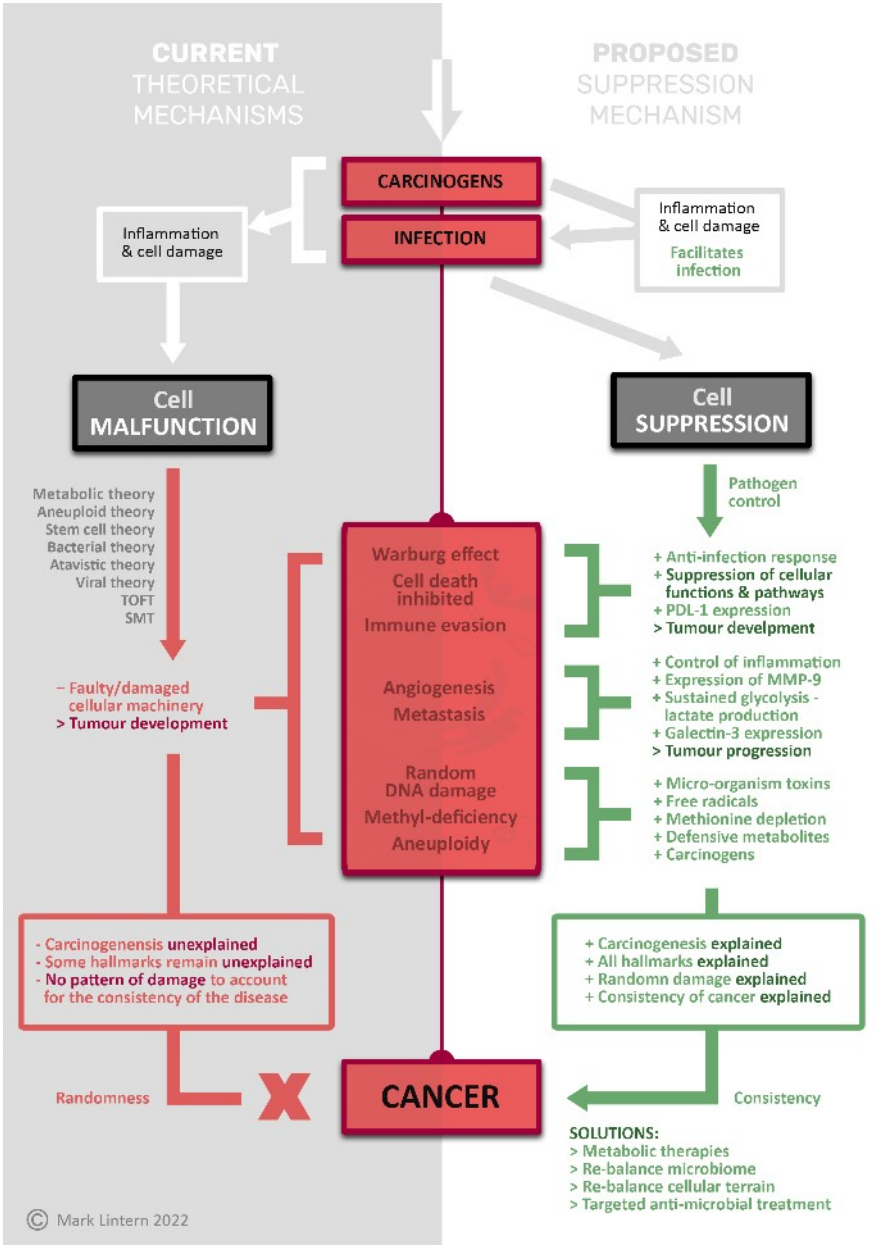


**Fig 2 – A tumour at the tissue level triggered by low biomass infection:**

A small number of infected cells can stimulate the Warburg effect in thousands of surrounding non-infected cells. These cells work collectively to eliminate the infection. This process fails, leading to sustained growth, resulting in a tumour.

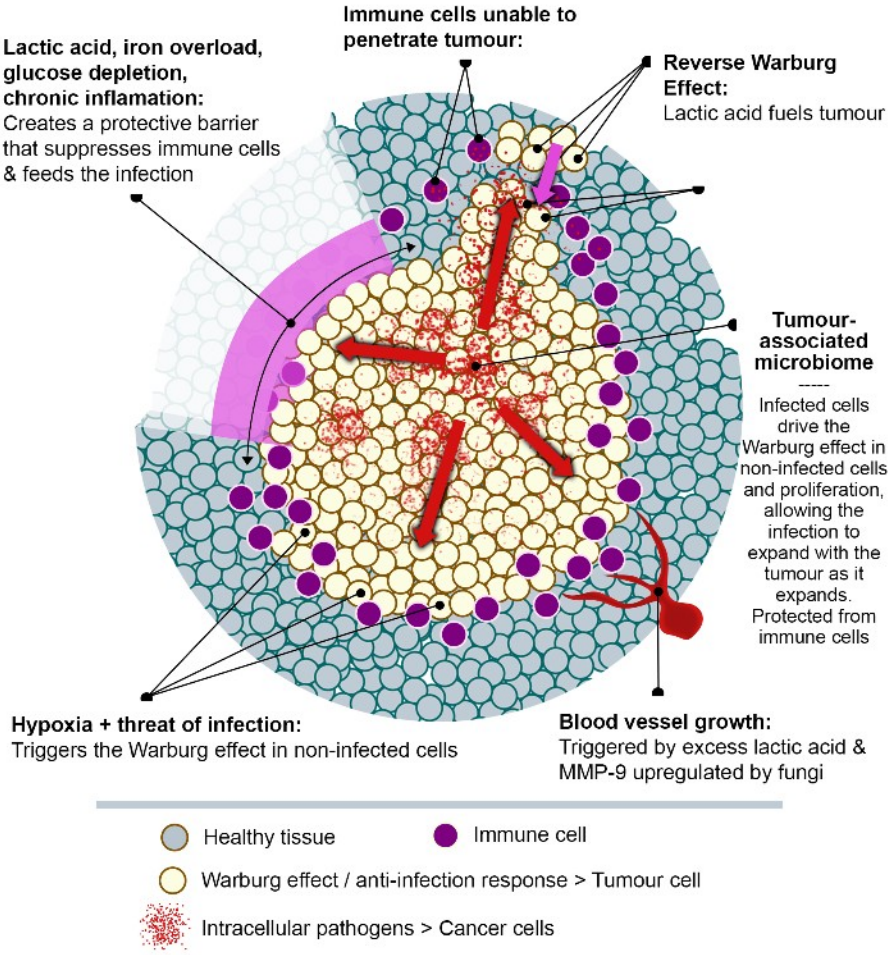
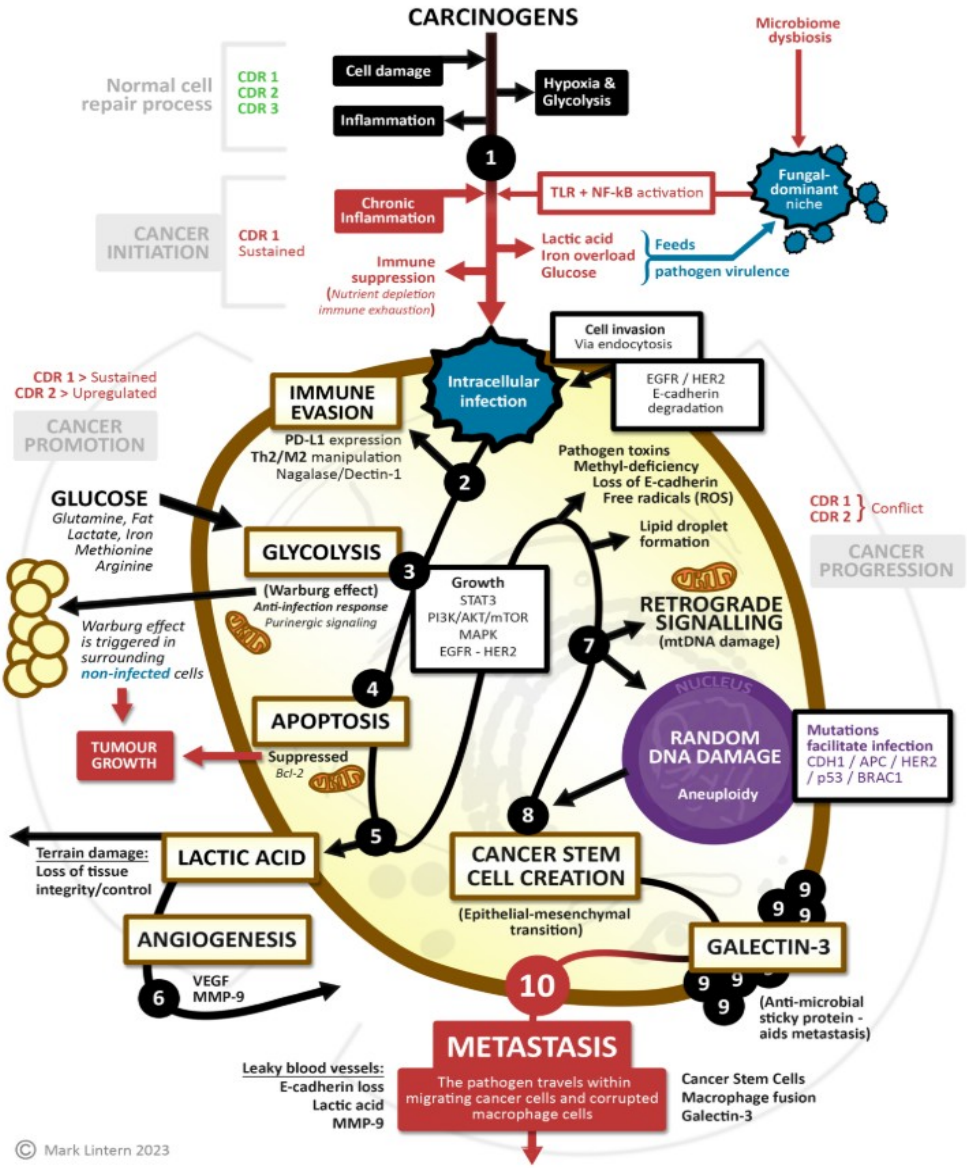


**Fig 3 – Carcinogenesis at the cellular level driven by fungal infection**



The Cell Suppression Theory has been published in the book ***The Cancer Resolution?***. Written in patient friendly language – it offers revolutionary insight in a manner that is easy for patients to understand.


For more information, and to order your copy of Mark's award-winning book, visit the CST website at: www.cellsuppression.com

Mark can also be found on Substack, Linkedin, and Facebook:

- https://substack.com/@marklinterncst
- www.linkedin.com/in/mark-lintern-cst
- www.facebook.com/groups/marklinterncancertheory/

## Additional SYNOPSIS REFERENCES:

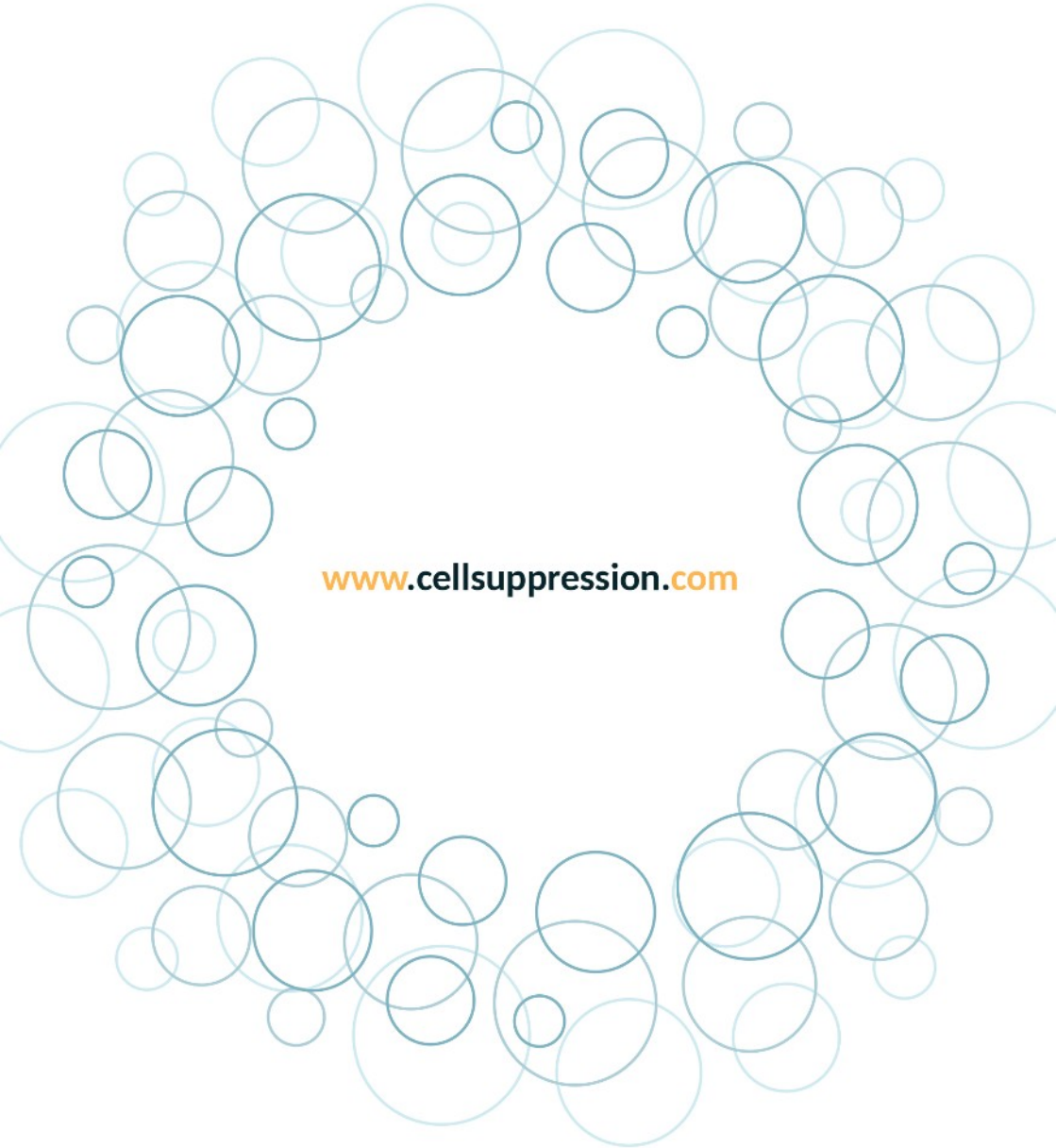
### Infection and the Warburg effect:


1. Timothy M. Tucey et al. 'Glucose Homeostasis Is Important for Immune Cell Viability during Candida Challenge and Host Survival of Systemic Fungal Infection.' Cell Metabolism. 2018. doi.org/10.1016/j.cmet.2018.03.019
2. Proal AD, VanElzakker MB. 'Pathogens Hijack Host Cell Metabolism: Intracellular infection as a Driver of the Warburg Effect in Cancer and Other Chronic Inflammatory Conditions.' Immunometabolism. 2021;3(1):e210003. doi.org/10.20900/immunometab20210003
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
4. Cheng, Shih-Chin et al. 'mTOR- and HIF-1α-mediated aerobic glycolysis as metabolic basis for trained immunity.' Science (New York, N.Y.). 2014. doi:10.1126/science.1250684
5. Memorial Sloan-Kettering Cancer Centre. 'Sloan Kettering Institute Scientists Solve a 100-Year-Old Mystery about Cancer.' January, 2021. https://www.mskcc.org/news/sloan-kettering-institute-scientists-solve-100-year-old-mystery-about?utm_source=Twitter&utm_medium=Organic&utm_campaign=012121MingLi-100-year-old-mystery&utm_content=Research&fbclid=IwAR0M7HU24J6RTLBXnBHJ48B05cpYACMLgIUJtHhFbuP7WsM5Z-0IXO-AE5A
6. Moyes, David L et al. 'Protection against epithelial damage during Candida albicans infection is mediated by PI3K/Akt and mammalian target of rapamycin signaling.' The Journal of infectious diseases. June, 2014. doi:10.1093/infdis/jit824
7. Julian R Naglik, Sarah L Gaffen, Bernhard Hube. 'Candidalysin: discovery and function in Candida albicans infections.' Current Opinion in Microbiology, Volume 52, 2019, Pages 100-109, ISSN 1369-5274, doi.org/10.1016/j.mib.2019.06.002.
8. Volling K, et al. 'Phagocytosis of melanized Aspergillus conidia by macrophages exerts cytoprotective effects by sustained PI3K/Akt signaling.' Cellular Microbiology. doi: 10.1111/j.1462-5822.2011.01605.x

**Five year survival statistics stage 3 and 4:**


1. https://www.nationalbreastcancer.org/breast-cancer-stage-3/
2. https://www.healthline.com/health/cancer/stage-4-cancer
3. https://www.healthline.com/health/breast-cancer/prognosis-stage-3-breast-cancer
4. https://www.nationalbreastcancer.org/breast-cancer-stage-4/
5. https://pancan.org/facing-pancreatic-cancer/about-pancreatic-cancer/survival-rate/
6. https://seer.cancer.gov/statfacts/html/common.html
7. https://pmc.ncbi.nlm.nih.gov/articles/PMC3228022/
8. https://www.medicalnewstoday.com/articles/is-stage-4-cancer-curable
9. https://acsjournals.onlinelibrary.wiley.com/doi/10.3322/caac.21820
10. https://www.cancer.org/cancer/types/lung-cancer/detection-diagnosis-staging/survival-rates.html
11. https://pmc.ncbi.nlm.nih.gov/articles/PMC8724440/
12. https://www.cdc.gov/united-states-cancer-statistics/publications/uscs-highlights.html
13. https://www.nuffieldtrust.org.uk/resource/cancer-survival-rates
14. https://my.clevelandclinic.org/health/articles/cancer-survival-rate
15. https://www.cancerresearchuk.org/health-professional/cancer-statistics/incidence/common-cancers-compared
16. https://www.komen.org/breast-cancer/facts-statistics/breast-cancer-statistics/survival-rates/
17. https://gco.iarc.who.int/media/globocan/factsheets/populations/840-united-states-of-america-fact-sheet.pdf
18. https://www.cancer.gov/types/common-cancers
19. https://www.medicalnewstoday.com/articles/323177


** Evidence for the table correlations between cancer and fungi are presented within the book The Cancer Resolution?.*


www.cellsuppression.com