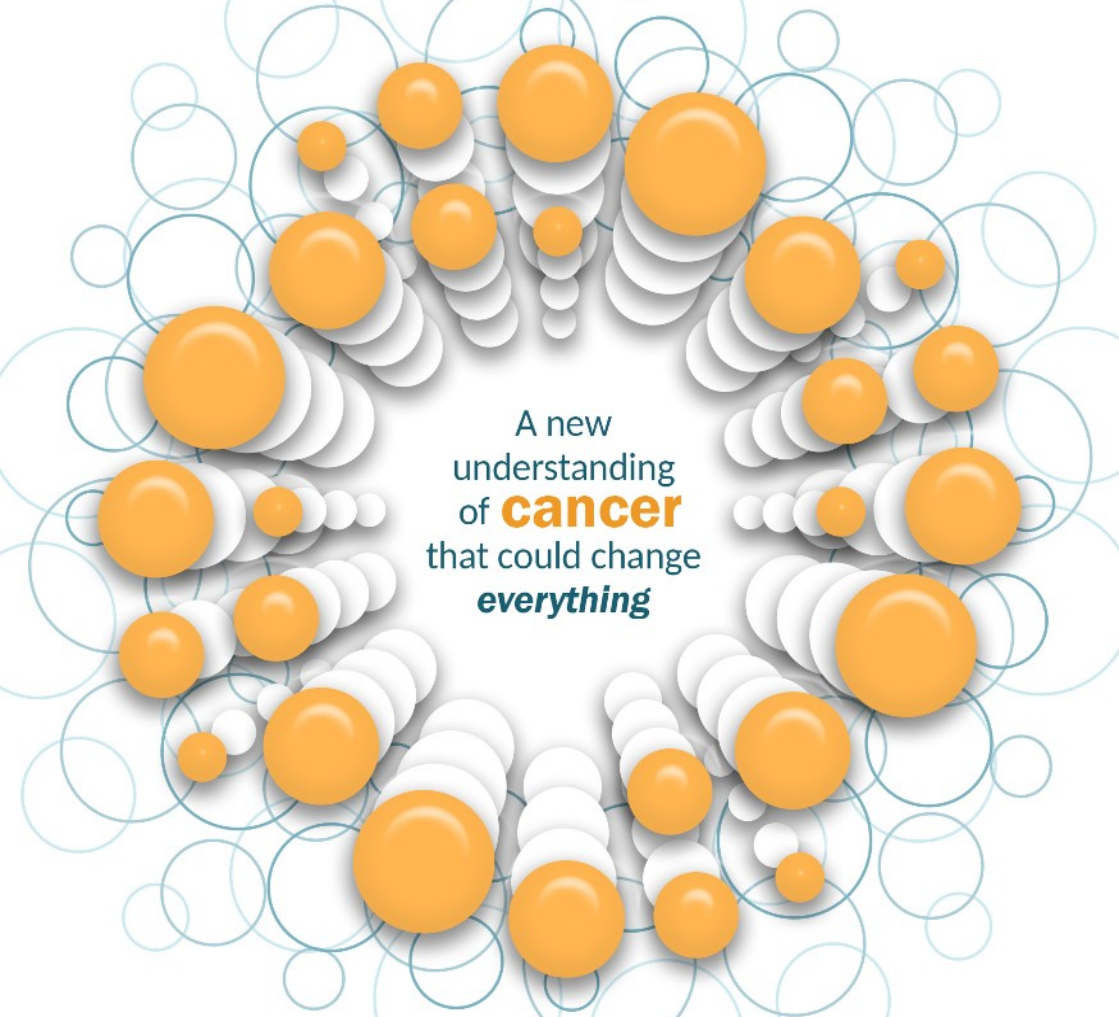
CELL SUPPRESSION THEORY

Synopsis



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

By MARK LINTERN

SYNOPSIS

A new framework for rethinking cancer and the questions worth asking

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. Why does this matter? Many assume that cancer treatments are chosen from across the full spectrum of cancer research – that the best possible options are identified and offered to patients based on the latest understanding of the disease. That's how clinical practice works, isn't it? Not exactly. It is true that standard cancer treatments are based on the latest evidence. But that evidence comes largely from one narrow interpretation of the disease – it does not encompass all of the science on what cancer is thought to be and that's a serious problem for anyone navigating a cancer diagnosis.

Not only is the origin of cancer still up for debate, but the nine or so competing cancer theories that propose different mechanisms, remain **unproven** – I cannot stress that point strongly enough. Scientists do not agree on the cause or the driving mechanism behind cancer, yet all of the standard cancer treatments are based on one theory under the presumption this theory is correct before it has been proven so. The Somatic Mutation Theory; the notion that DNA mutations drive the disease is the established theory through which cancer treatments are developed and justified in a clinical setting. But you don't need to be a rocket scientist to understand the potential implications – what if that unproven theory is wrong?

Here's the shocking truth: despite over sixty years of research, billions of dollars invested, and countless drugs developed, the treatments developed from the dominant theory have 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.

And here's the key bone of contention. Treatments are developed from cancer theory, meaning that different theories offer different treatment options based on the scientific evidence that supports each theory. For instance, the Metabolic Theory suggests that cancer arises from defects within the energy system of the cell, not mutated DNA. It provides scientific justification for metabolic treatments – and there is certainly a large evidence-base supporting this, particularly with brain cancer. The contention, is that patients are rarely made aware of these additional theories and their associated treatment approaches, all because the medical system favours one theory above all others.

The Cancer Stem Cell Theory nicely illustrates this oversight. Chemotherapies target the DNA of fast dividing cells because DNA mutations are said to drive the disease and fast dividing cancer cells absorb more of the treatment – that sounds like a logical approach. And it is, until you factor in cancer stem cells. Chemotherapy can fail because cancer stem cells are slow-growing and have superior toxin removal systems. Professor Robert Weinberg admitted back in 2011 that we've failed to understand the problem because we are so focused on pushing one theory and the treatments associated with it – as a result we are missing something highly significant. Evidence created by proponents of the Cancer Stem Cell Theory indicate that around 2% of a tumour is made up of stem cells – immortal cells that regenerate damaged tissue – they are the reason why the liver is the only organ that can completely regenerate itself, it's full of stem cells. While chemotherapy can eliminate most of the regular cells of a tumour, shrinking it dramatically, the cancer stem cells remain. They duplicate, and then re-grow the tumour, often at a more aggressive pace. The shortcoming of placing all of our treatment eggs in one basket is abundantly clear – yet we continue to offer the same chemotherapies as we have for the last 40 years, to focus on the same target without broadening our outlook, expecting a different result.

This gap between expectation and outcome has left patients confused, fearful, and searching. In the United States, up to 70% of cancer

1 doi:10.1001/jamaoto.2014.1570

patients turn to complementary or alternative therapies.[2] In the UK, the figure is between 30-40%.[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 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**. But here's the point that tends to get overlooked – cancer science has not yet advanced beyond the theory stage. The origin of cancer has not been determined. Yet one theory has been elevated to the status of fact before it has been proven – creating the appearance that cancer's origin has been identified and that cancer science has progressed

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

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

past that point, when it hasn't. An entire clinical framework has been built on that premise. The evidence above and below suggests that premise may be wrong. Which means the most useful place to make treatment decisions is not beyond the theory stage – it is at that stage. When we accept that, something shifts: rather than deferring to one unproven theory as though the question were settled, we can use cancer theory as a practical tool, weighing each one against the evidence and letting that comparison guide which treatment strategies deserve the most attention.

How then do you assess the relevance of any given theory so that you may use that information to better inform treatment decisions? The hallmarks framework provides exactly this metric. Scientists agree there are 10 defining "hallmarks" of cancer – 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. And when we assess each theory, an interesting pattern emerges.

Of some concern: the established Somatic Mutation Theory (SMT) on which nearly all mainstream cancer treatments are currently based, struggles to explain more than two of these hallmarks, while proclaiming cancer a genetic disease as if it 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 may be why, despite decades of effort and billions invested, the dominant SMT has failed to deliver a cure. Either cancer is genetic but too complex for us to fathom with current technology, or mutated DNA isn't the driving mechanism. If it's the latter, its treatments are targeting the wrong feature of the disease entirely, and will largely appear ineffective – as the five-year survival statistics presented in *Table 1* below seem to confirm.

When viewing Table 1 consider that five-year survival statistics do not represent a cure, just that patients are still alive five years after diagnosis. This metric does not take into account the current health status of the patient, who could be living with other chronic conditions, while their prognosis may still be terminal past those five years.

Table 1: Treatment effectiveness – late-stage 5 year survival statistics

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%

**Statistics as of 2025*

Additional shortcomings of note

The reality is that patients are advised to follow established treatments without being informed of other options associated with other theories – this, despite the evidence painting a troubling picture:

1. **The cancerous DNA mutations thought to drive the disease appear in healthy tissue without cancer developing.**[4]
2. **Cancers can arise without showing any of the required driver mutations.**[5] [6] [7]
3. **In a study of over 7,000 samples, only 1.7 driver mutations were found on average**[7] – there are an insufficient number of mutations to explain how cancer forms. The SMT requires between 3 to 8.
4. **The mutations found are random, not consistent.**[8] This randomness cannot explain the remarkable consistency of cancer (10 shared hallmarks) 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.

This evidence, taken together, points in one direction: genetic mutations are not the primary driver of cancer – they are more likely a symptom of another cause, an innocent bystander implicated by association. It is the kind of mistake a police investigation makes when it locks onto a suspect early, builds its case around that assumption, and

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*

stops looking for alternative explanations – even as the contradictory evidence mounts. The question is why that mistake persists. When 95% of cancer research money flows into supporting one theory, consensus naturally follows – and consensus, once established, is extraordinarily difficult to dislodge. But consensus is not the same as truth, as Michael Crichton observed:

> *'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 Crichton*** *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 presented in *Table 1* above.

A framework for navigating cancer

So what can we do instead? This is where my work introduces something beneficial: **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. In February 2023, I presented my research to a panel of 10 independent experts – including oncologists, researchers, and clinicians – at an event called *Cancer Through Another Lens*. They were asked to score the theory based on their confidence in 25 questions dotted throughout the 3 presentations and then a final vote of confidence at the end of the 6 hour webinar. The expert panel returned an average score of 7.4 out of 10. When the wider audience of more than 200 medical professionals was included, that figure rose to 7.9. This wasn't a peer review – but it was public, expert scrutiny. And it suggests the framework deserves a closer look.

The paradigm shift: 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 share the same unexamined starting point: the assumption that cancer results from *cell malfunction*. It is a conclusion dressed as a premise – and it matters, because once you assume the cell is broken, you stop asking what might be acting on it from the outside. That closed door is where the CST begins. Rather than treating cancer as a disease of malfunctioning cells, the CST proposes that cancer is the result of *suppression*, where an external influence – the pathogen – hijacks key functions/pathways to survive, and as a result the cell remains stuck in a process of infection control, over-expressing certain pathways that are required for pathogen clearance. The problem: the pathogen isn't eliminated. This chronic state generates all of the symptoms of cancer, due entirely to a failure to deal with this small, near invisible infection – thus

offering a complete paradigm shift in thinking.

In simple terms, our cells are not broken, rogue or evil. They are doing exactly what they were designed to do – but under the wrong kind of influence and for much longer than is deemed beneficial. And indeed, evidence confirms that fungal pathogens in particular, can invade cells, hijack their machinery, and suppress their natural defences leading to a proliferative state. Under this paradigm it makes sense to support the body's natural healing abilities to aid in pathogen clearance.

The core feature that highlights pathogens are at the heart of cancer

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 the backup energy system? Mitochondria that create energy via OXPHOS using oxygen, have **malfunctioned**, this forces the shift to glycolysis – a corrosive lactic-acid generating energy system that facilitates cancer development when used to excess.

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 intentional re-purposing of the OXPHOS pathway requires that glycolysis (the backup energy pathway) is upregulated to support this defensive response. This anti-infection strategy is well documented in the medical literature. In other words, 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.

Even the main proponent of the *Metabolic Theory*, Professor Thomas

Seyfried, admits that intracellular pathogens exist within tumours, and that they can, and do, drive the Warburg effect in cancer. 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.

The missing piece of the cancer puzzle

Many different pathways are upregulated in cancer, which are said to help cancer cells survive and adapt, these include:

- **NF-κB** – a central inflammatory switch that keeps immune and survival genes active.
- **MAPK** – signals that drive proliferation and survival under stress.
- **IL-6** – inflammatory and immune messaging, telling cells to grow, survive, and ignore stop signals.
- **STAT3** – a "master survival switch" that promotes proliferation, immune evasion, and resistance to cell death.
- **mTOR** – a nutrient and growth sensor, driving protein production and high metabolic demand.
- **HIF-1α** – shifts cells toward sugar-based energy (glycolysis), a key feature of the Warburg effect.
- **Glycolysis (Warburg effect)** – the main energy pathway in cancer.

Loosely speaking, this is the general order in which these pathways are activated. Together, they trigger glycolysis and support survival, inflammation, and uncontrolled growth. From this perspective, cancer cells appear to be "working against us," so inhibiting these pathways seems logical.

But what if we've misinterpreted this? What if cells aren't working against us, but for us, and it's their context that has been compromised? Evidence shows that these exact pathways are upregulated during fungal infection in the same order – the only difference is **duration**.

In acute infection, activation is brief. In cancer, it never switches off.

The missing piece of the puzzle, overlooked because cancer is assumed to be a result of cell malfunction, is that the infection remains in the tissue. That's the defining concept that sets the CST apart, and is the key mechanistic difference that potentially offers a more plausible explanation for the Warburg effect in cancer. And as the CST predicts, studies by Ravid Straussman and colleagues have shown that all tumours tested contain intracellular fungal and bacterial pathogens that are surviving within these cells – tumours that the consensus previously claimed were sterile (free of micro-organisms). The revolutionary insight I'm offering here is that the inability to clear this ongoing infection keeps these anti-infection pathways activated far longer than intended, driving the unrestrained proliferation we recognise as cancer. In other words, chronic activation is not a rogue cell behaviour, but a **sustained, adaptive response to a persistent intracellular pathogen** – the real driver of tumour growth. This response becomes pathological because the danger signal never ends.

This subtle but profound shift in thinking, of not blaming the cell, but of considering the external influence of a pathogen, 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 and many additional features besides. 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 framework for understanding why they may deserve closer attention, because as it turns out, most off-label drugs that appear to have efficacy against cancer, also have secondary anti-fungal properties – see *Table 2* below for some intriguing examples.

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 there may be mechanisms worth exploring with your oncologist that mainstream treatment protocols haven't yet addressed. Patients no longer have to choose blindly between mainstream approaches, untested alternatives, or confusing blends of both. With the framework the CST provides, patients have better questions to bring to their oncologist – and a clearer way of evaluating the answers.

This is why I 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 help 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.

Table 2: Significant correlations between anti-cancer off-label drugs

Drugs with anti-cancer properties	Primary + Additional properties
Itraconazole, Miconazole, Fluconazole	Anti-fungal + Metabolic inhibition
Metformin (diabetes drug – glucose)	Metabolic + Anti-fungal
2-DG (glucose)	Metabolic + Anti-fungal
Atorvastatin, Lovastatin (fat)	Metabolic + Anti-fungal
Gleevec (imatinib – glucose)	Metabolic + Anti-fungal
3BP (3-bromopyruvate – glucose)	Metabolic + Anti-fungal
DON (6-diazo-5-oxo-L-norleucine – glutamine)	Metabolic + Anti-fungal
Artemisinin (Malaria)	Anti-viral + Anti-fungal
Mebendazole, Fenbendazole, Atovaquone, Ivermectin	Anti-parasitic + Anti-fungal + Metabolic
Doxycycline	Anti-bacterial + Anti-fungal + Metabolic
Tamoxifen	Estrogen inhibition + Anti-fungal
Arimidex (a triazole derivitive)	Estrogen inhibition + Anti-fungal
Propranolol (inhibits adrenaline)	Beta-blocker + Anti-fungal
Disulfiram (treats alcoholism)	Enzyme inhibition + Antifungal

The pattern is difficult to ignore. These drugs arrive from entirely different pharmacological directions (metabolic, anti-parasitic, anti-bacterial, hormonal), yet a secondary anti-fungal property runs through all of them. The dual metabolic and anti-fungal profile begins to look less like coincidence and more like a signal, an observation aligned with the number of cancer's hallmarks each theory can explain: 7/10 and 10/10 respectively. This supports using cancer theory as a practical tool for treatment decisions, raising a question that deserves serious attention: are patients benefitting from off-label drugs because they're inadvertently targeting fungal pathogens as the CST proposes?

We find other significant correlations that look more like a signal than a coincidental pattern. Table 3 presents the uncanny correlations between what we see happening in cancer, compared to how fungi interact with our cells, tissue and immune system to sustain themselves intracellularly. It's almost as if fungal infection and cancer are one and the same thing.

Table 3: Significant correlations of note – fungi and cancer

Cancer characteristics	*Link to fungal infection
Chronic inflammation triggers and drives cancer	Fungi trigger and control inflammation to aid infection
P53, CDH1, APC, HER2, BRAC1 mutations increase cancer risk	All of these mutated genes facilitate intracellular fungal infection
E-cadherin is downregulated	Fungi downregulate E-cadherin to aid with cell invasion
Toll-Like-Receptor 2 (TLR-2) is activated	Fungal infection activates TLR 2
Nf-kB pathway is activated	Fungal infection activates Nf-kB
MAPK pathway is activated	Fungal infection activates MAPK
IL-6 is upregulated	Fungal infection activates IL-6
STAT3 pathway is activated	Fungal infection activates STAT3
PI3K/ATK/mTOR is activated	Fungal infection activates PI3K/ATK/mTOR
HIF-1a is activated	Fungal infection activates HIF-1a
The Warburg-effect (glycolysis) is a pivotal metabolic shift seen in all cancers	Fungi trigger the Warburg effect during infection – it's an anti-infection response
Lactic acid overproduction and iron overload fuel cancer	Lactic acid and iron feed fungal pathogens while suppressing immune cells
Programmed cell death fails (apoptosis)	Fungi suppress apoptosis to survive intracellularly, keeping the infected cell alive
Tumours do not stop growing	Fungi activate cell growth receptors

Lipid droplets accumulate within the cytosol of the cell	Lipid droplet accumulation occurs during fungal infection to protect PUFA's
CYP1B1 is an enzyme that is only activated in tumours	CYP1B1 forms part of an anti-fungal response pathway within the cell
Succinic Acid is produced via Glutamine fermentation (mSLP)	Fungal infection triggers Succinic Acid production
Nagalase is produced only in tumours. It suppresses macrophage immune cells	Fungi produce Nagalase to suppress macrophages to evade elimination
M2 Macrophages dominate tumour tissue (M2 = cell repair. M1 Macrophages = pathogen elimination)	Fungal pathogens suppress M1 & stimulate M2 Macrophages to evade immune detection
Th1 immune response is suppressed	Fungal pathogens suppress the Th1 response to evade immune elimination
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 within infected cells
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
Anti-fungal drugs show efficacy against many cancer types	Anti-fungal drugs inhibit and kill fungi
Off-label drugs – Metformin, Lovastatin, Mebendazole, Ivermectin, Doxycycline show efficacy against cancer	These drugs have secondary anti-fungal properties
Case study: Terminally diagnosed pancreatic cancer patient	Cured/resectable tumour after using Itraconazole – an anti-fungal drug
Aykut et al 2019 – pancreatic cancer study	Malassezia fungi confirmed to be driving tumour growth

Aparicio-Fernandez L, et al – melanoma cancer study	Candida fungi increase the aggression and metastatic potential of melanoma
Ravid Straussman pan cancer analysis	Fungi found in all cancers studied
The Mayo Clinic, Dr Vikram MD	Fungal infections mimic cancer – almost impossible to differentiate the two

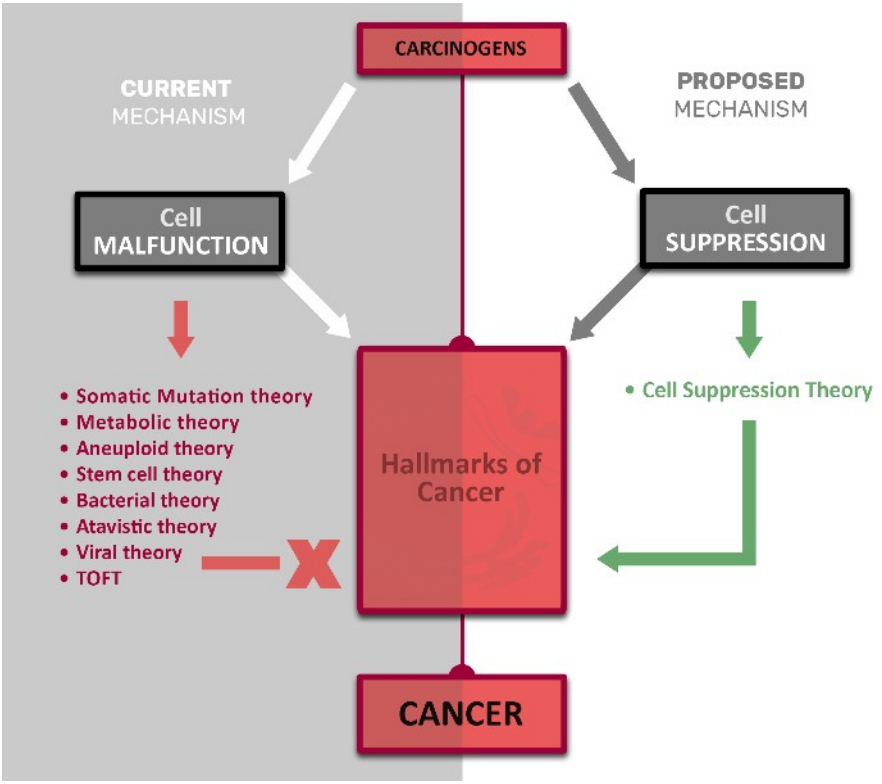
A shared mission

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?* offers a framework for understanding your options – and the questions worth raising with your care team. 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**.

Fig 1 – Cell Malfunction vs Cell Suppression paradigm shift



Is the premise that *cell malfunction* is the driving mechanism behind cancer, the fundamental error in thinking that is preventing us from understanding cancer's true origin? Do the symptoms of cancer arise as a result of *suppression* orchestrated by a novel intracellular infection and the body's failed collective response to eliminate it?

Fig 2 – Tumour development explained via a 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.

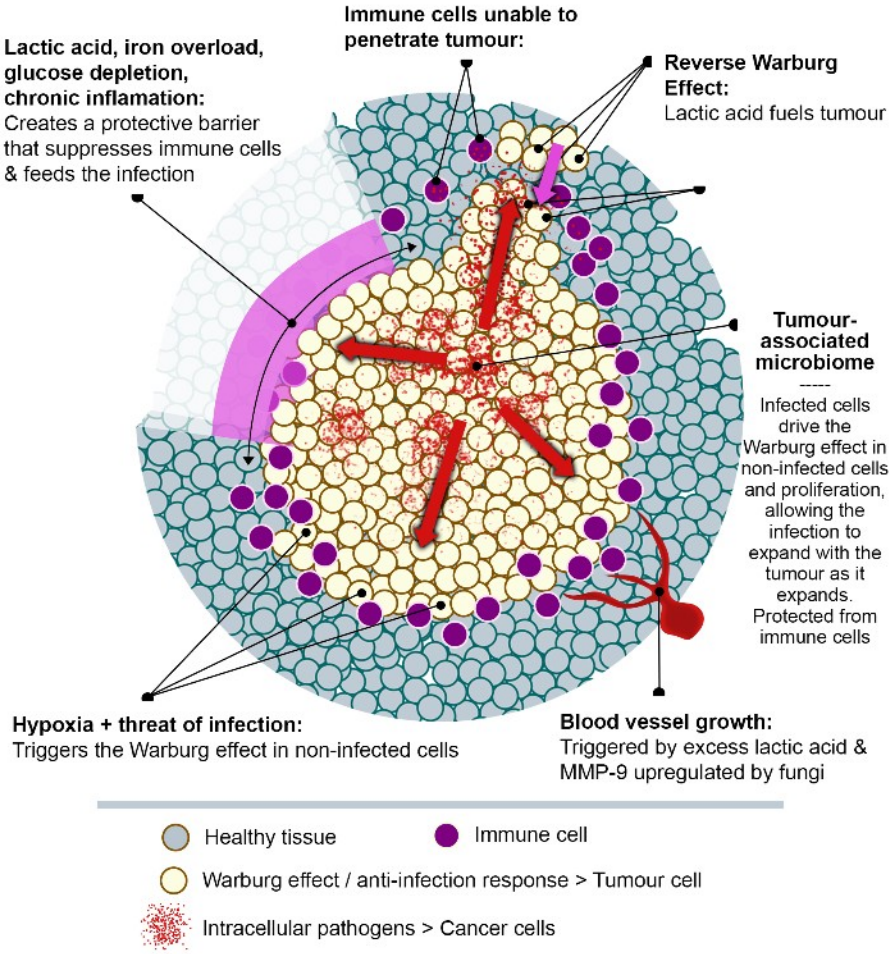
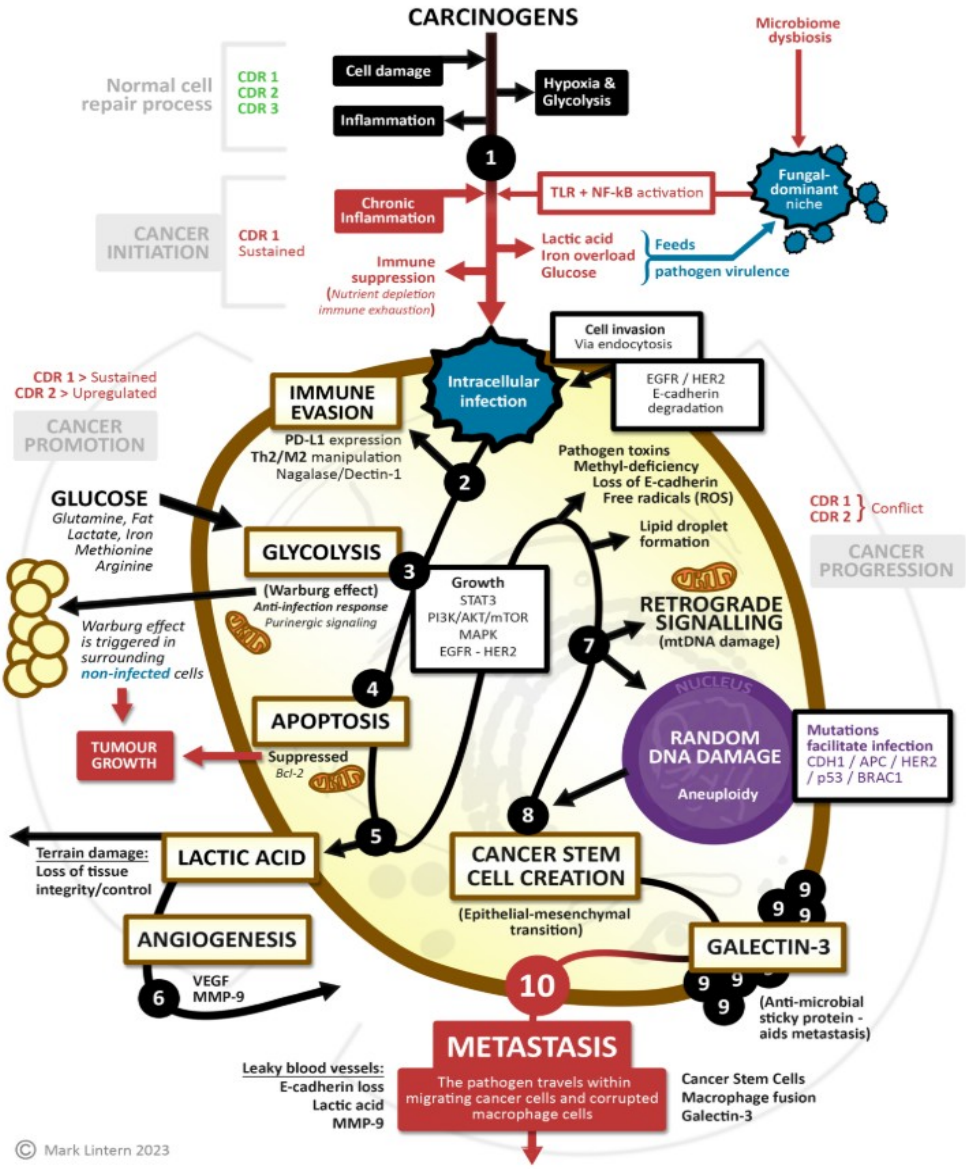


Fig 3 – Carcinogenesis at the cellular level driven by fungal infection

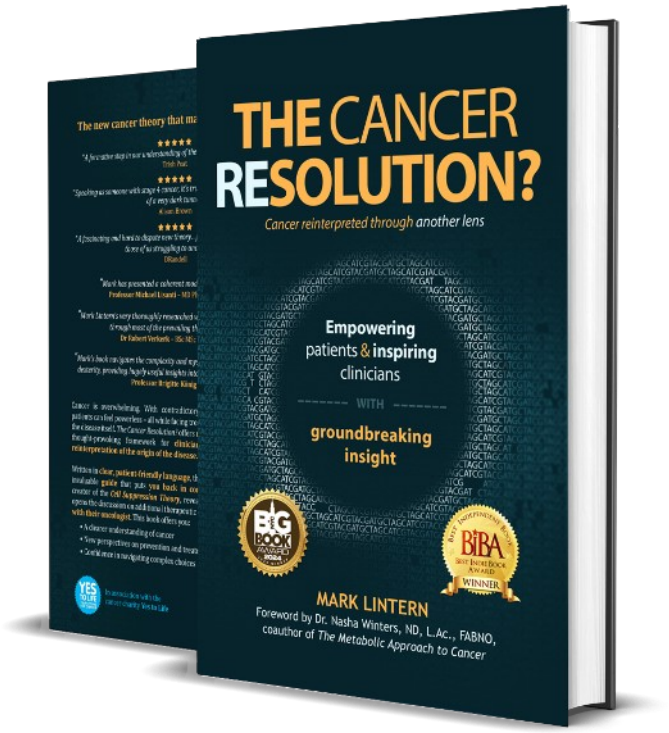


The Cell Suppression Theory has been published in the book ***The Cancer Resolution?***. Written in patient friendly language – it translates a decade of independent research into terms that are clear, concise, and genuinely useful.

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/



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Infection and the Warburg effect:

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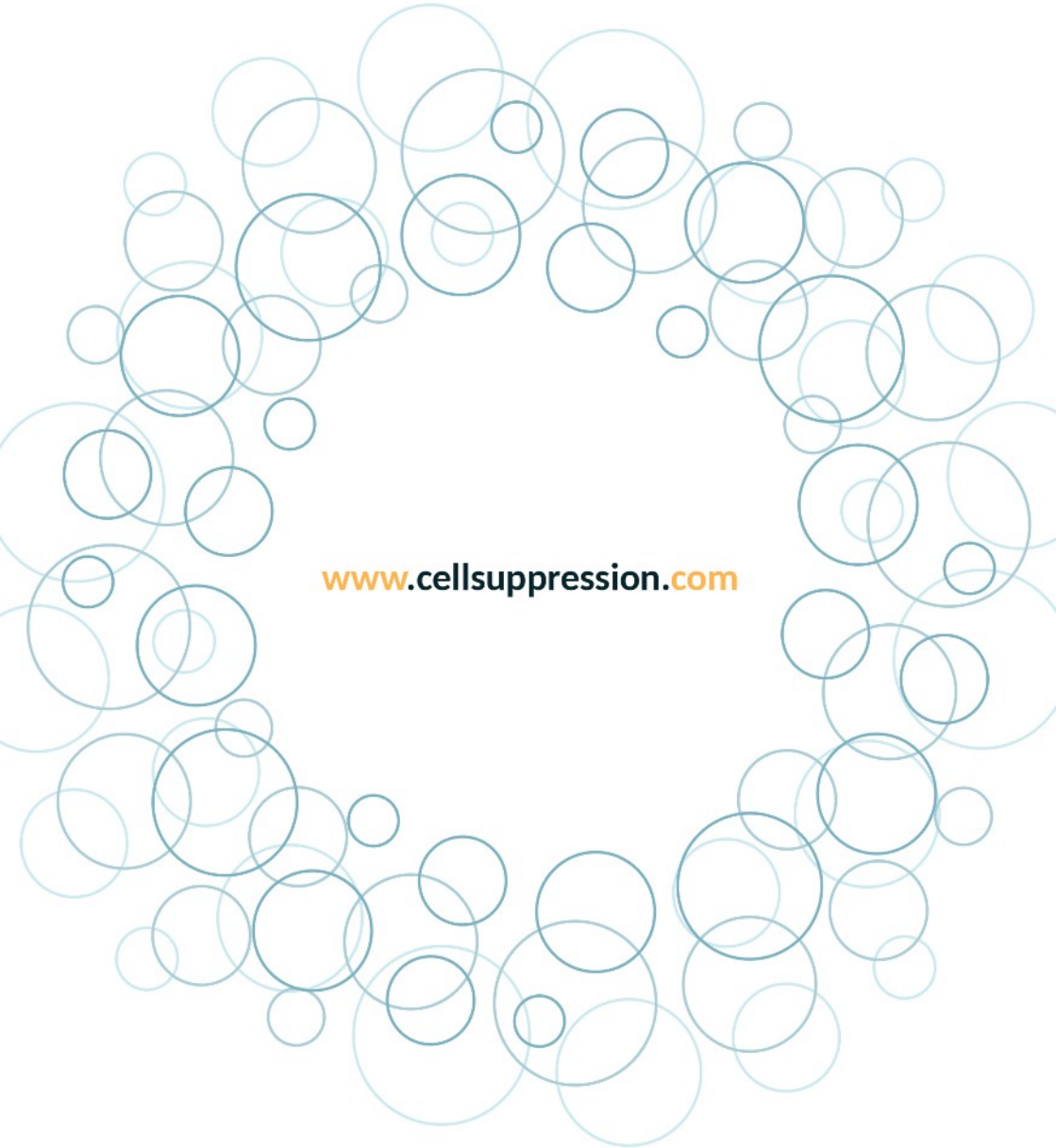
*Further supporting evidence available on request: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