GTI Perspectives on Cancer: A Systemic and Evolutionary Framework

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Article

GTI Perspectives on Cancer: A Systemic and Evolutionary Framework

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Abstract: Based on the General Theory of Innovation (GTI), this paper proposes a new cancer origin hypothesis and reframes cancer as a system-level adaptation to an existential threat. Cancer is modeled as a network of structural transformations, each attempting to preserve cellular viability under increasing systemic stress. When mitochondrial function collapses, the cell activates emergency fallback strategies such as fermentation and de-differentiation, optimizing energy management for survival. The transformational process leading to cancer emergence, traditionally viewed as pathological, is interpreted as an act of systemic preservation. The paper introduces a predictive framework for early detection and intervention based on this dynamic systems perspective. It also presents a comprehensive summary of novel insights and concepts, opening new directions for research, diagnostics, and systems-based health modeling. Using the same framework, GTI also offers new definitions of health and disease, and innovative strategies for disease modeling, treatment, and diagnostics.

Keywords: Cancer diagnosis; cancer origin; cancer treatment; disease modeling; definition of health; definition of disease; General Theory of Innovation

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Disclaimer on Language and Systemic Framing

This paper uses terms such as "choice," "response," or "survival strategy" when describing cellular behavior. These expressions are not intended to suggest cognition, intention, or anthropomorphism. Rather, they reflect a systems-theoretic interpretation rooted in the General Theory of Innovation (GTI), where biological behavior is explained through structural logic—not mental states.

Cells are not conscious, and GTI does not attribute awareness or agency to them. Instead, such language characterizes how systems adapt organizationally to preserve existence under threat. These formulations are used for explanatory clarity within a framework that treats systems as goal-oriented through their design and function, not through deliberate intent.

1. Introduction

This paper presents the results of the application of the General Theory of Innovation (GTI) to the domain of cancer. We used GTI's systems logic to reframe cancer emergence, behavior, and progression. The introduction provides background context on the current dominant theories and their limits.

The prevailing scientific understanding of cancer has been shaped for decades by the genetic mutation model. According to this theory, cancer begins with genetic damage that triggers uncontrolled cellular growth (Hanahan & Weinberg, 2011). While this paradigm has yielded important insights, it is increasingly inadequate as a total explanation.

Not all individuals with "cancer genes" develop cancer. Conversely, cancer frequently occurs in individuals without genetic mutations (Vogelstein et al., 2013). Moreover, transplant studies have shown that placing a cancerous nucleus into a healthy cell does not lead to tumor formation—while transplanting cancerous mitochondria into a healthy cell does result in cancer emergence (Seyfried, 2015). These inconsistencies suggest that genetics is likely not the root cause.

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The metabolic theory of cancer, originally proposed by Otto Warburg and revived by Dr. Thomas Seyfried and others (Warburg, 1956; Seyfried, 2012), offers a more coherent model. It asserts that cancer is a disease of energy metabolism, driven by mitochondrial dysfunction. The transplantation experiments mentioned above offer compelling evidence for the critical role mitochondria play in cancer emergence.

Regarding the mechanism of emergence, Dr. Seyfried argues that mitochondrial damage also impairs cellular regulation, particularly the control of growth and division, thereby contributing to the uncontrolled proliferation characteristic of cancer (Seyfried et al., 2014).

Additionally, this model offers promising therapeutic strategies: caloric restriction, ketogenic diets, hyperbaric oxygen therapy, and control of glucose and glutamine intake—all aimed at starving cancerous cells by disrupting their metabolic pathways (Seyfried et al., 2014).

Yet one critical question remains. If damaged mitochondria are the true cause of cancer, why do many cells with such damage never become cancerous (Seyfried, 2015)? This suggests that mitochondrial damage is neither the immediate nor the only cause.

Under the first scenario, damaged cells must undergo additional transformations before reaching an unidentified "X-event" that results in crossing the threshold and loss of regulatory control. Not all cells progress to this point. If the second scenario holds, then mitochondrial failure must be accompanied by an unknown "X-factor" that tips the system into collapse. In both cases, the mitochondrial damage is a necessary but insufficient condition, and the full mechanism remains undefined.

Like the genetic model, the metabolic theory lacks predictive structure. It describes the internal mechanism well but does not offer early warning that fundamental structural changes are approaching, or that the system is becoming prone to collapse.

2. Theoretical Framework and Method

This is where GTI enters the picture. It reframes cancer not as a defect but as a system-level adaptation—an emergent response to existential threat. From this view, genetic mutations and metabolic shifts are not root causes, but intermediate phases in a process aimed at preserving the system's existence.

To facilitate discussion, the next section introduces GTI, the Five Flows model, and other concepts relevant to this context.

2.1. GTI Foundations Used to Create the Cancer Model

The General Theory of Innovation was originally developed in the USSR by Greg Yezersky in the late 1980s, introduced in the US around 1998, and formally published in English in 2006 (Yezersky, 2006). It has since evolved into a fully axiomatic theory concerned with the evolution of artificial systems – those that are man-made or involve human participation. GTI explains why systems emerge, transform, and ultimately die in response to environmental changes. Transformations that allow systems to survive disruptions are classified as innovations.

Though originally focused on artificial systems, GTI has proven effective in analyzing complex natural systems, due to deep structural parallels.

A system, in GTI, is a structured set of interconnected elements whose relationships yield an emergent property – a capacity enabling it to perform a function that addresses a problem in its environment.

In return for this service, the environment sustains the system with vital resources. These relationships are in Equilibrium if no change is required - a preferred state of problem-free existence.

Equilibrium disruption threatens relationships. The system must either adapt (solve the problem) or die, for the environment will withdraw resources.

 doi:10.20944/preprints202504.1618.v1

2.2. The Five Flows Model

To operationalize equilibrium, GTI introduces the Five Flows framework—a model describing how systems acquire, manage, and release resources to survive.

GTI classifies resources as Positive (those that support survival) and Negative (those that threaten it). Systems rarely encounter them in isolation, so separation becomes a core task.

The Five Flows are:

- **Inflow of Positive Resources**: Beneficial inputs (e.g., oxygen, nutrients, information).
- **Inflow of Negative Resources**: Harmful inputs (e.g., toxins, pathogens, misinformation).
- **Outflow of Negative Resources**: Expulsion of waste or threats (e.g., CO_2, cellular debris).
- **Outflow of Positive Resources**: Unintentional loss or required sacrifice (e.g., heat loss, nutrient leakage).
- **Internal Resource Management**: Redistribution and regulation of internal flows (e.g., blood circulation, cellular signaling).

When these flows are balanced and uninterrupted, the system state is defined as health. Disease arises when one or more flows become blocked, excessive, or persistently imbalanced, resulting in functional degradation.

This model applies to all systems—cells, organs, ecosystems, cities, corporations. In disease states, it allows us to analyze biological structures not just anatomically, but **functionally**, in context.

Most critically, GTI identifies the structural origin of disease as the point at which balance across the Five Flows is lost. It offers a complementary lens through which health, disease, and systemic behavior can be reinterpreted via structural logic.

3. Results and Discussion

This chapter applies the General Theory of Innovation (GTI) to analyze cancer through a systems lens. We reinterpret the metabolic theory of cancer in GTI terms, present cancer progression as a system-level transformation, and decompose it into discrete phases. This enables the identification of new intervention and testing points. We also propose two diagnostic strategies—progressive and probabilistic testing—aligned with GTI's logic of system evolution. Finally, we present a working hypothesis on the systemic conditions under which cancer may emerge.

3.1. Reframing the Mitochondrial Theory of Cancer

In GTI terms, a cell is a system; it fits the definition. It exists in relationship with its environment, performing functions, thereby maintaining equilibrium. In return, the environment—whether tissue, bloodstream, or whole body—provides resources (oxygen, nutrients, and regulatory signals—Positive Inflow) and facilitates the removal of waste (Negative Outflow).

When all five flows—Positive Inflow, Negative Inflow, Positive Outflow, Negative Outflow, and Internal Resource Management—are balanced and well-managed, the system remains healthy. It performs its functions, sustains itself, and supports the larger system.

However, chronic exposure to low-level stressors—such as toxins, radiation, hypoxia, or metabolic overload (Negative Inflow)—disrupts the fragile equilibrium. Mitochondria, key to Internal Resource Management, begin to falter. Energy production declines, impairing the system's ability to absorb inputs, expel waste, and maintain internal order.

Dr. Seyfried identifies mitochondrial dysfunction as the root cause of cancer. When oxidative phosphorylation fails, the cell can no longer produce energy efficiently or regulate growth. As compensation, the cell activates fermentation—a fallback pathway that enables survival, not restoration. This shift, while not cancer itself, is a key step in its metabolic reprogramming (Seyfried, 2012).

GTI adopts this model without reinterpretation. It treats mitochondrial damage as a structural failure and fermentation as a survival response. But unlike conventional views, GTI sees this transformation not as isolated stages, but as a continual transformation.

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This reframing of cancer as a structured process enables new opportunities to model its progression, identify earlier detection points, and design targeted interventions—before the system fully transitions.

3.2. GTI Problem Chain: From Equilibrium to Cancer

In the previous section, we reframed cancer as a continuous process—a sequence of system-level changes unfolding over time. GTI decomposes this progression into a sequence of distinct system events—each representing a specific problem to be solved. This breakdown not only aligns with GTI's problem-solving logic but also reveals new testing and intervention points that are often obscured in traditional models.

What follows is an example of the process decompoosition.

0. Initial State: System in Equilibrium

The cell begins in a state of health. All five flows are active, stable, and balanced. Vital resources are received and processed; waste is expelled efficiently; internal systems manage allocation and repair. Mitochondria perform oxidative phosphorylation without error.

1. Environmental Stressor Introduced

Chronic stressors—such as toxins, radiation, processed foods, hypoxia, or persistent inflammation—begin to destabilize the cell. These do not destroy the system outright but gradually erode its balance.

2. Mitochondrial Stress

Inflammation, oxidative stress, etc. impair mitochondrial performance. Internal resource management begins to break down. Energy demand begins to outpace supply.

3. Distress Registered and Signaled

The cell detects internal failure through disrupted ATP levels, oxidative imbalance, and other metabolic cues. In response, it broadcasts a systemic distress signal — a molecular "SOS" — indicating critical dysfunction and the need for intervention. Cellular distress signaling is well known in biology (Tang et al., 2012), including in the development of cancer (Krysko et al., 2012).

4. Mitochondrial Damage and Collapse of the Function

Mitochondria are damaged. Oxidative phosphorylation fails. Although oxygen remains available, the cell can no longer use it to generate energy.

5. System Approaches Death

Energy collapse puts the cell at risk of death. It must find a new way to survive or cease to exist.

6. Fermentation Activated as Fallback

The cell switches to fermentation. This less efficient metabolic pathway provides temporary survival, albeit with accumulating byproducts and reduced regulatory control.

7. Loss of Regulation Begins

Control systems degrade. Feedback loops fail. Internal signaling and repair are compromised. The cell is surviving but has exited its original functional architecture.

8. Cancerous System Emerges

The cell has transformed into a new system with its own flows, goals, and survival logic – no longer contributing to the original system but extracting resources to sustain itself.

Implications

Each event in this chain becomes a basis for problem formulation, often yielding more than one problem per phase. For example, at Stage 2: How might we slow down mitochondrial degradation under chronic stress? Or: How can we prevent degradation even when stressors persist? This structural approach transforms a monolithic disease into a map of smaller, solvable challenges—each offering a new entry point for intervention, prevention, or systemic management.

The sequence of events in the above chain is highly meaningful.

From the GTI perspective, no developed system can passively degrade into failure. It must first perceive its own instability and initiate a self-preserving response. The signaling event, therefore,

 doi:10.20944/preprints202504.1618.v1

must precede the collapse. In fact, it represents the earliest possible evidence that the system is under threat.

This insight provides a strategic opportunity for diagnostics: to detect a system under attack before any structural damage occurs. From a GTI perspective, each event represents a distinct signal: a detectable deviation from equilibrium that may precede any overt symptoms.

3.3. GTI-Based Decomposition Enables New Testing Strategies

In the previous section, we showed how decomposing cancer into system-level events expands the number of intervention points. Each phase also represents a testable moment—something that might be detected, tracked, or reversed. This section presents three diagnostic strategies enabled by this decomposition.

3.3.1. Chain-Based Testing: Expanding the Diagnostic Landscape

GTI decomposes cancer into a continuum of system-level transformation events, each representing an opportunity to test, intervene, or monitor—before, during, or even late into the disease. This expanded diagnostic landscape maps testing points across:

- Early stress indicators (e.g., inflammation, mitochondrial strain)
- Mid-stage failures (e.g., metabolic shift, signaling dysregulation)
- Late-stage breakdowns (e.g., loss of internal regulation)

This broader reach allows clinicians to act sooner and more precisely. Every "leftward" test, even a small one, offers potential gains in time, options, and survivability.

3.3.2. Progressive Testing: Mapping Trajectories Over Time

Viewing cancer as a stepwise progression allows for trajectory-based testing. Rather than relying on isolated markers, clinicians can deploy tests:

- Simultaneously – to locate the patient in the failure chain
- Sequentially – to observe progression, stasis, or recovery over time

Even modestly sensitive diagnostics, layered across phases, can reveal directionality—opening new paths for personalized monitoring, risk modeling, and preemptive action.

3.3.3. Probabilistic Testing: Leveraging Cumulative Confidence

GTI also enables a low-cost, high-yield strategy: probability stacking. Instead of a single expensive, high-specificity test, clinicians can combine multiple inexpensive, imperfect diagnostics, each targeting a different phase. Statistically, five tests with 50% reliability can yield over 95% cumulative accuracy.

This method supports dynamic, individualized health profiles—regularly updated to reflect changes across flows and functions. It democratizes early detection and brings precision medicine closer to public health scale.

Conclusion: Transformation of Cancer Testing

GTI transforms cancer testing into systemic exploration. Each structural phase becomes a point of insight, detection, or prevention. Testing becomes dynamic, layered, and responsive mirroring the disease process itself.

3.4. Reconsidering the Origin of Cancer: A System-Level Hypothesis

As discussed above, the metabolic theory of cancer offers a compelling but incomplete explanation for cancer's origin: not all cells with damaged mitochondria become cancerous. We now propose a GTI-based hypothesis to explain why only some cells cross this threshold. Though speculative by design, it is logically grounded and consistent with the framework developed in this paper.

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3.4.1. What We Know

According to Dr. Thomas Seyfried in Cancer as a Metabolic Disease (Seyfried, 2012), the process of cancer emergence begins with a metabolic disruption. The sequence below outlines core observations:

- Chronic low-grade stress—such as inflammation, toxins, or radiation—impacts the cell over time.
- Mitochondrial damage accumulates gradually. If the insult is too severe, the cell dies. If moderate and persistent, the cell survives in a compromised state.
- Oxidative phosphorylation declines. Mitochondria can no longer reliably produce energy.
- Fermentation is activated as an alternative energy-generating pathway.
- Unregulated cell division begins, and the cell departs from cooperative tissue function.
- Cancerous cells are observed to be de-differentiated—having lost their specialized identity.
- Cancerous behavior follows—marked by autonomous operation and sustained proliferation.

Dr. Seyfried emphasizes that mitochondria, beyond their role in energy production, also regulate cell growth and division (Seyfried, 2012; Nunnari & Suomalainen, 2012). Mitochondrial dysfunction is a consistent feature of cancer cells, suggesting a strong link between impaired mitochondrial function and tumorigenesis.

He also states that this dysfunction triggers a metabolic shift from oxidative phosphorylation to fermentation. This metabolic transition is well documented in cancer cells, yet its role in triggering malignancy remains unclear.

De-differentiation is another hallmark of cancer. Cells lose their specialized roles and begin to resemble primitive or stem-like forms (Mani et al., 2008). Like fermentation, this is widely observed, but its causal relationship to cancer remains unclear.

This progression—from mitochondrial stress to energy shift, to de-differentiation, and finally to deregulation—is extensively documented. But the internal logic connecting these stages remains unresolved. **Why do only some cells follow this path?** That is the central question we now address through GTI.

3.4.2. The Light at the End of the Tunnel

The progression from mitochondrial stress to fermentation, de-differentiation, and deregulation is observable—but its internal logic remains elusive. Establishing causal relationships among these transitions is difficult, and it's unclear why this sequence results in malignancy.

To address this, we propose a reversal of perspective. Rather than tracing the process forward from stress, we begin at the endpoint—unregulated division—and ask a simpler question: Why would a system make that choice?

This framing is not intended to imply cognition, intention, or consciousness on the part of the cell. Rather, the 'choices' described reflect structural responses consistent with system survival.

GTI brings this paradox into sharp focus. A distressed system is expected to conserve energy, reduce exposure, and seek equilibrium. Yet cancer cells launch an aggressive strategy that violates every principle of structural preservation:

- High energy demand: division is metabolically expensive. A system facing scarcity would normally downregulate, not escalate (Vander Heiden et al., 2009).
- Instability and error: rapid division increases mutation and disorder (Loeb, 2001; Vander Heiden et al., 2009).
- Immune exposure: growth breaks conformity, triggering detection (Dunn, Old, & Schreiber, 2004).
- Collapse of order: expansion increases entropy, not control (West, You, & Zhang, 2019).
- Loss of support: the cell exits cooperative flow: nutrients, signaling, repair (Hanahan & Weinberg, 2011).

 doi:10.20944/preprints202504.1618.v1

- Low survival odds: every step increases risk, not resilience (Greaves & Maley, 2012; Maley et al., 2017).

Why would a system under threat choose such a self-destructive path?

Because the status quo guarantees death. This behavior isn't strategic—it's the last resort. Not a bid for dominance, but an escape from extinction.

In GTI terms: the system doesn't evolve because it wants to; it evolves because it must. This reframes cancer not as mystery, but as desperation—a structural solution to existential threat.

If true, then the next step becomes clear: identify the threat the cell can no longer tolerate.

3.4.3. The Enemy Within

What is the pressure that makes a system abandon order for chaos? The answer lies not in external attack, but internal consequence.

When oxidative phosphorylation fails and fermentation becomes chronic, the cell gains energy—but pays a steep price.

Fermentation isn't pathological. In healthy cells under acute stress—like exercise—it is brief, reversible, and harmless.

But under persistent mitochondrial failure, fermentation becomes a long-term survival mechanism that produces lactic acid.

Acid is not neutral. It accumulates, disrupts pH, corrodes structures, impairs signaling, and threatens cellular homeostasis (Estrella et al., 2013).

Normally, waste clears and balance is restored. But when recovery fails, **acid builds**, flow breaks down, and internal regulation falters. The system becomes toxic to itself (Hui et al., 2022).

This is the turning point: the cell is no longer threatened externally—it is poisoned from within.

Staying means death.

So, the cell moves—not to grow, but to escape. It divides in search of survivable configuration. Each daughter inherits the same threat and makes the same choice.

This is not malignancy—it is **recursive survival**. But the logic is fatal.

Cancer is not a plan. It is a chain reaction: a system trying to survive an intolerable internal condition.

3.4.4. De-Differentiation: Another Solution to the Same Problem

Let us now return to a key observation: cellular de-differentiation**.** What triggers this regression?

Fermentation is active. Acid is accumulating. The cell is under mounting stress. This state of structural degradation and declining capacity is not unique, it mirrors a universal pattern among systems under existential threat.

What does a business do in crisis? It lays off consultants, halts travel, freezes salaries. These aren't optimizations, they are survival decisions.

Biological organisms follow the same logic. When humans fall ill, they reduce activity. Appetite drops. Movement slows. Focus narrows. The body cuts expenditures to preserve core flows (Hart, 1968).

The cell obeys this principle with striking fidelity. In its differentiated state, it performs a specialized function—an identity that demands energy and regulatory overhead. Useful in health, this identity becomes a luxury in crisis. So, the cell lets it go (Talchai et al., 2012; Iwase et al., 2011).

This is not degeneration—it is prioritization.

Differentiation is costly. It requires signaling, transcription, and constant confirmation of role and context. When survival is at stake, these functions are paused (López-Otín et al., 2013). The cell retracts, simplifies, becomes more like a stem cell: flexible, undirected, unspecialized.

GTI interprets this not as pathological failure but as a systemic strategy: the suspension of non-essential functions to preserve core viability. In a system of declining resources, complexity is a liability. Simplicity becomes a strength (Pakos-Zebrucka et al., 2016; Csete & Doyle, 2002).

 doi:10.20944/preprints202504.1618.v1

And so, like every distressed system, the cell returns to a more primitive configuration—not to advance, but to survive. This transformation precedes malignancy (Berger et al., 2014); it is part of the survival response, not its malignant outcome.

3.4.5. Implications of the Cancer Emergence Hypothesis

Framing cancer as a system-level adaptation to existential threat reshapes our approach to diagnosis, intervention, and theoretical understanding.

Cancer as a Systemic Solution

Cancer is not a random aberration—it is a last-ditch solution to system collapse. GTI reframes cancer not as a genetic mistake or metabolic glitch, but as a system's final adaptation to internal failure. Unregulated growth is not irrational aggression but a desperate attempt to survive when equilibrium can no longer be maintained. It is not a plan; it is the only remaining option.

Cancer Is a System and Must Be Treated as Such

Cancer fits the definition of a system: it has structure, function, internal flows, and environmental relationships (Hanahan & Weinberg, 2011; Bissell & Hines, 2011). This opens new avenues for diagnostics and treatment. Instead of merely targeting outcomes (like tumor mass), we can intervene at what sustains the system: resource acquisition (e.g., glucose uptake—Seyfried et al., 2014), fermentation stability, signaling coordination, and immune evasion. Understanding its systemhood offers multiple entry points for disrupting its survival logic.

Multiple Solutions to the Same Problem

Fermentation and de-differentiation both arise from mitochondrial failure. Fermentation maintains energy production; de-differentiation reduces energy demand. These are not root causes of cancer but survival responses—systemic strategies for preserving viability under collapse. The existence of both mechanisms strongly suggests that other energy-preserving solutions must also exist. Mapping other such mechanisms can expand our diagnostic and therapeutic reach.

Prediction: Other Than the De-Differentiation Survival Strategies Must Exist

De-differentiation is a drastic maneuver—like a company cutting core operations to survive. If this analogy holds, it implies earlier, less destructive adaptations must exist. Before shedding identity, a system may reduce signaling, metabolic demands, or resource acquisition (Pakos-Zebrucka et al., 2016; Szalay et al., 2007). Identifying these early responses offers valuable diagnostic windows—before irreversible shifts occur.

The Cancer Process Is a Network, Not a Line

Fermentation, de-differentiation, and other stress responses form not a linear sequence but a network of adaptive shifts. GTI predicts this: real transformation rarely follows a single path. Some changes occur in parallel, others are recursive, conditional, or supportive. Mapping this network enables earlier detection and more precise intervention.

De-Differentiation Is Not Exclusive to Cancer

Any system under threat will reduce non-essential functions to preserve survival. Cellular de-differentiation exemplifies this logic. If it's truly a structural response, it should appear in other stressed but non-cancerous cells—which it does: under ischemia, toxicity, and mechanical injury (Bonventre, 2014; Dietrich et al., 2022; Rangel et al., 2024). This supports GTI's view that de-differentiation is a survival tactic, not necessarily a malignant signature.

 doi:10.20944/preprints202504.1618.v1

Conflict Resolution as a Prerequisite for Optimal Results

Cancer exemplifies a GTI core concept: **Conflict**. Fermentation resolves energy scarcity but introduces acid toxicity. This is a classic trade-off: the solution to one problem becomes the cause of another. It's not limited to biology—pharmaceutical side effects follow the same logic. GTI insists: never introduce a solution without structurally resolving the conflict it creates.

This demands a shift in medical best practices—from patchwork fixes to conflict modeling. Only by addressing failure consequences up front can we avoid turning today's treatment into tomorrow's problem.

4. Conclusion

This paper introduced a series of concepts and hypotheses derived from the application of the General Theory of Innovation (GTI) and its systems-theoretic evolutionary logic to the domain of cancer. While some of these concepts are novel, others are already supported by empirical research.

We see this convergence as a validation of GTI's explanatory and predictive power. More importantly, the central contribution of this work lies not in individual insights (though they matter), but in their conceptual re-assembly into a unified, system-based framework. This framework offers a fresh perspective—not only on the origins and treatment of cancer, but on the systemic nature of Health and Disease. It also reveals a powerful, largely untapped synergy between specialized biomedical research and universal theories of systems and innovation.

The following table offers a compact summary of the proposed ideas, each paired with its practical utility and recommended next steps.

##	**Proposed Concept / Insight**	**Utility / Benefits**	**Recommendation**
1	Five Flows - Health	Defines health; enables system-level insight and monitoring	Debate and adopt in education, diagnostics, intervention, prediction
2	Five Flows - Disease	Explains disease as flow disruption; defines disease origination point, supports detection and system repair	Debate and adopt in education, diagnostics, intervention, prediction
3	Cancer as Adaptation / Solution	Reframes cancer as response to threat; opens new intervention logic	Validate as core concept; apply to early detection and therapy
4	Cancer as a System	Enables new intervention logic aimed at system-forming parameters	Validate and adopt at diagnostic and therapeutic levels
5	Cancer Emergence Hypothesis	Explains transition from normalcy to malignancy	Validate hypothesis; test across cancer types
6	Cancer Progression as a Process	Enables discovery and use of multi-node diagnostics and systemic disruption strategies	Validate and adopt at diagnostic, therapeutic, and educational levels
7	Cancer as a System (Network) of Problems	Expands the intervention and diagnostics opportunities	Validate and adopt at diagnostic, therapeutic, and educational levels

 doi:10.20944/preprints202504.1618.v1

8	Any Disease as a Process/System	Extends GTI disease modeling beyond cancer; supports general application	Adopt for general disease system analysis
9	Any disease as a System (Network) of Problems	Makes complex disease more solvable via structural mapping	Apply to other diseases; develop problem maps for every disease
10	Problem Network Testing Strategy	Enables more discrete, stage-specific testing, including at earlier stages, by using newly discovered failure events	Design detection models around network of events
11	Progressive Testing Strategy	Tracks movement along disease trajectory over time (both directions)	Build time-aware detection models that track evolving system signals
12	Probabilistic Testing Strategy	Combines low-fidelity or low-accuracy signals to enable high-confidence, low-cost testing.	Build and adopt low-cost screening protocols from stacked signals
13	Cell Distress Signal as Earliest Diagnostic Point in Cancer	Enables ultra-early detection before structural damage	Develop & validate diagnostic strategy and tools targeting this phase
14	Cell Distress Signal as Universal Early Marker of Disease	Enables early diagnostics across diseases with cellular stress; allows preemptive interventions & diagnostics	Develop & validate diagnostic strategies targeting this phase
15	De-Differentiation as Energy Preservation	Explains identity loss as energy-saving; offers diagnostic marker	Incorporate into early diagnostic models
16	Additional Survival Energy-Saving Mechanisms Must Exist	Predicts existence of additional early signals for intervention and diagnostics before identity loss	Explore and validate additional adaptive mechanisms
17	De-Differention of Cells Beyond Cancer	Confirms hypothesis; expands diagnostic space beyond cancer	Study other diseases for similar behaviors
18	Conflict in Systems	Predicts emergence of negative consequences when any change is introduced into an established system	Adopt for structural analysis of existing and future interventions
19	Conflict Resolution as Requirement for Innovation Introduction	Improves safety and impact of new products and procedures	Integrate conflict resolution as a required step in product and procedure development

 doi:10.20944/preprints202504.1618.v1

20	Introduction of Systems and Innovation Theories into Education	Enables early systemic thinking; improves future innovation literacy and application skills	Integrate into STEM and medical education

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