

Autological Oncology – A Functional Model of Structural Evolution in Cellular Systems

Introduction

What if cancer is not merely uncontrolled growth, but a *loss of structural learning*?
This paper extends the **law of Autological Recursion**

$$\Psi = \partial S / \partial R$$

into the realm of biology, defining cancer as a state where regulatory structures (**S**) lose sensitivity to recurrent environmental or intracellular perturbations (**R**).

In functional terms, cancer represents $\Psi \approx 0$ — a system that repeats without learning.

The study introduces a measurable framework for **structural reflexivity in living cells**, combining molecular biology, systems theory, and ethics.

Rather than proposing a therapy, it offers a **testable scaffold**: a way to quantify whether a cell can still modify its own rules.

Scientific Perspective

The model defines a diagnostic triad of cellular adaptability:

Symbol	Meaning	Interpretation
Ψ_{eff}	functional reflexivity	sensitivity of regulation to repetition
L_{eff}	energetic load	metabolic cost per structural change
C	coherence	stability of cellular identity

In healthy cells:

- $\Psi_{\text{eff}} \uparrow \rightarrow$ rule responsiveness
- $L_{\text{eff}} \downarrow \rightarrow$ efficient integration
- C** stable \rightarrow preserved identity

In malignant cells:

- $\Psi_{\text{eff}} \approx 0 \rightarrow$ rigid recursion
- $L_{\text{eff}} \uparrow \rightarrow$ energy inefficiency
- C** collapse \rightarrow identity drift

These quantities can be derived from existing data (scRNA-seq, ATAC-seq, metabolomics) and simulated in silico using lightweight computational models.

Cancer thus becomes measurable as a *recursion defect* rather than merely a mutation cascade.

Experimental Roadmap

The paper outlines a three-phase validation program:

Phase	Focus	Method
1 – RNA Reflexivity	Transcriptional rule adaptation under repeated stress	Organoid hypoxia/reoxygenation cycles; scRNA-seq trajectory of Ψ_{eff}
2 – Epigenetic Feedback	Correlation between ΔS , ΔE , and recovery	CUT&Tag / ATAC integration
3 – Rule Perturbation	Reversible CRISPRi/a edits	ABAB design (on/off 3×); success = Ψ_{eff} $\uparrow \geq 15\%$, $L_{\text{eff}} \downarrow \geq 10\%$, C stable $\pm 5\%$

All steps are **ex vivo**, reversible, and governed by strict tripwire rules for safety (e.g., halt if $\Psi \uparrow$ with $L \uparrow$ or identity collapse occurs).

RNA as Structural Syntax

RNA is treated as the **operational grammar** of life —
a recursive medium that both expresses and regulates structure.
The metric

$$\Psi_{RNA} = \frac{\|\Delta S_{RNA}\|}{\|\Delta R_{RNA}\| + \varepsilon}$$

captures how transcriptional networks adjust under repeated stress.
High Ψ_{RNA} indicates functional adaptability; $\Psi_{RNA} \approx 0$ denotes syntactic freeze.
This allows **autological parameters** to be extracted directly from RNA-seq data,
bridging computation and biology without altering DNA.

Ethical and Methodological Governance

The framework embeds ethics as structure, not decoration:

1. **Ex vivo only** – no in vivo edits until proxy metrics validated.
2. **Reversibility** – CRISPRi/a or epigenetic modifiers only, never nuclease cuts.

3. **RDI tripwire** – automatic halt if instability ($\rho > \lambda$).
4. **Transparency** – pre-registration, open code, null results published.
5. **Autological ethics** – oversight as higher-order recursion: rules that govern rule-change.

Limitations

- Ψ_{eff} is a **proxy**, not an absolute constant.
- Network inference noise requires ensemble averaging.
- Tumour heterogeneity may blur Ψ trends.
- Energy proxies (ATP use) remain indirect.
- No therapeutic claim — purely diagnostic and theoretical.

Context and Implications

Autological Oncology reframes cancer as a *syntax disorder of evolution*:

a point where the recursive capacity of life breaks down.

Rather than fighting the cell, the framework measures its ability to **regain structural reflexivity**.

Potential applications include:

- Quantifying plasticity during therapy.
- Predicting resistance trajectories.
- Detecting irreversible loop states ($\Psi \approx 0$).
- Mapping reprogramming efficiency in differentiation.

Evolution learns by rewriting its own syntax.

Disease begins where that recursion stops.

Evaluation

Aspect	Assessment Comment	
Conceptual novelty	High	First reflexivity-based model of cancer.

Aspect	Assessment Comment	
Empirical foundation	Emerging	Protocols and simulations proposed, no results yet.
Ethical soundness	Strong	Reversible, transparent, and governed by tripwire safeguards.
Scientific value	High	Connects cellular plasticity to recursive structure theory.

Conclusion

Autological Oncology offers a **unifying grammar for adaptation and disease**, bridging systems biology and consciousness research.

It proposes that cancer, at its core, is not chaos but **stalled recursion** — life that can no longer update its own rules.

Measuring Ψ is measuring the point where life forgets to learn.

Archival Note

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