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When Experience Does Not End

Organismic Identity, Attractor Substitution, and the Loss of Cellular Return Capacity

A VIVENS hypothesis for pre-neoplastic cellular memory

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Conceptual manuscript developed within VIVENS, in dialogue with LIMEN and SOMATHEON

Draft v0.3.1 — 30 July 2026

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Abstract

A previous VIVENS paper proposed that organismic experience can become cellular history: an event registered at the scale of the integrated organism is materially translated through neural, autonomic, endocrine, immune, vascular, metabolic and biomechanical processes, thereby changing the local world in which cells decide among repair, arrest, death, survival, proliferation and inheritance of damage. That model left a central question unresolved. Why can a biological response continue after the event that elicited it has ended?

This paper proposes that the missing operator may be the identity-bearing organization of the organism itself. Within the VIVENS vocabulary, Integrated Functional Coherence (CFI; *Coerência Funcional Integrada*) denotes the embodied, multiscale organization that sustains a living unit through perturbation; Singular Identity (SI; *Singularidade Identitária*) denotes the historically continuous form through which that organization becomes this organism's situated experience; and Ω denotes the dynamic stability regime that permits coherent change and return. These terms do not name separate substances. They distinguish functions of one embodied organization.

The proposal contains two nested claims. The conservative mechanistic core is that a measurable post-event host state can maintain neural, autonomic, endocrine, immune and tissue outputs during recovery, thereby reducing effective cellular return capacity within the current host and niche. If the altered conditions become incorporated, intrinsic return capacity of the cell or lineage may also change. The strong VIVENS hypothesis is that Singular Identity (SI) helps determine whether the host state is formed, reinstated or terminated: when the identity-bearing organization of the organism continues to conserve an experience as operationally present, it may keep Integrated Functional Coherence (CFI) in a corresponding physiological regime. Cells initially adapt appropriately to this persistent organismic world; some later incorporate it as epigenetic, transcriptional, metabolic, lineage or niche memory. The previous Ω regime may then lose recoverability while another stable regime is established.

Evidence already supports the component processes: neural ensembles can encode and retrieve peripheral inflammatory states; body–brain circuits can causally regulate inflammation; sensory neurons can direct immune resolution or fibrotic cell fate; resolved injury can leave long-lived epithelial memory that potentiates later tumor initiation; and cellular lineage, differentiation state and prior history alter the consequences of an identical oncogenic or therapeutic event. No study yet demonstrates the complete proposed chain from individual conscious identity to pre-neoplastic cellular memory in humans.

The paper distinguishes intrinsic from effective cellular return capacity, separates absence of spontaneous return from absolute irreversibility, and specifies criteria that distinguish persistence or

hysteresis from a full attractor claim. It derives recovery-phase experiments that separate the precipitating event, the local lesion, the systemic output and later cell or niche memory. The model is explicitly non-universal, non-dualist and non-voluntarist. It does not claim that consciousness is necessary or sufficient for cancer, that all persistent physiology is conscious, or that patients cause disease through thought or emotion.

Keywords: organismic identity; cellular return capacity; attractor substitution; epigenetic memory; neuroimmune circuits; cancer initiation; multiscale causation; tissue memory; downward causation; VIVENS

Epistemic status. This is a conceptual and mechanistic hypothesis paper. It reports no original biological experiments and does not constitute clinical guidance. The empirical literature supports individual links in the proposed architecture, not the entire chain. “Attractor,” “Ω-transition,” and “return capacity” are operational concepts to be made measurable; they are not presented as already validated biological variables. The hypothesis remains open to restriction or rejection.

1. Introduction

An adaptive response is biologically valuable because it reorganizes a living system in relation to an event. Inflammation contains injury, stress physiology reallocates resources, nociception protects tissue, fear prepares action, and tissue plasticity permits repair. The same response can become pathological when it survives the conditions that made it appropriate. What first protected the organism may later constrain it.

This transition is usually divided among specialized literatures. Neuroscience studies persistent threat representation and sensitization. Psychoneuroimmunology studies the physiological consequences of appraisal and stress. Immunology studies failed resolution. Fibrosis research studies pathological repair. Epigenetics studies cellular memory. Cancer biology studies field effects, lineage susceptibility, tumor initiation and clonal selection. Each field captures a segment of the process, but the architecture connecting them is rarely treated as one causal problem. The history of psychosomatic and neuroimmunomodulatory claims also shows why these levels must be separated: speculative psychogenic causation and materially testable brain–body mechanisms should not be treated as equivalent (Anastassis & Konsman, 2024).

> *How can a response created to traverse an event persist after that event ends, become incorporated across biological scales, and reduce the capacity of cells or tissues to return?*

The first VIVENS preprint, *When Organismic Experience Becomes Cellular History*, addressed the downstream half of this problem. It argued that an integrated organism does not transmit semantic content directly to its cells. A cell does not receive grief, confinement, danger or safety as narrative. It receives the material world produced when the organism undergoes a situation: altered catecholamines and glucocorticoids, immune mediators, vascular states, oxygen and nutrient distribution, neural activity, mechanical loading, behavior and tissue interaction. If these changes modify cellular fate and persist after the systemic state has ended, organismic experience has become cellular history (Santos Albino, 2026a).

That model established a possible route from organism to cell, but it did not identify why the organismic state itself might fail to terminate. The present paper begins there. Its central proposition is:

> *The cell may fail to return because, for the identity-bearing organization of the organism, the event has not become biologically past.*

This is not a claim that conscious thought commands cell fate. It is a hypothesis about embodied identity and causal persistence. An event is registered by an organism whose history shapes salience, expectation, affective orientation, interoception and action. The resulting state is realized through measurable physiology. When the state persists or is repeatedly reinstated, cells continue to inhabit a world in which the response remains locally appropriate. Only later may this initially faithful adaptation become self-maintaining cellular or tissue history.

The paper develops this hypothesis in six movements. First, it locates the new proposal relative to the previous VIVENS paper. Second, it operationalizes CFI, SI and Ω without treating them as separate entities. Third, it defines return capacity and the evidentiary threshold for attractor substitution. Fourth, it organizes the supporting evidence into neural, organismic, tissue and cellular modules. Fifth, it derives a testable experimental program. Finally, it defines the preventive window between failure of spontaneous return and irreversible genetic, clonal or ecological fixation.

2. Where this paper begins

2.1 The previous paper: transmission and incorporation

The previous paper asked whether a state realized at the organism scale can alter the causal field in which a vulnerable cell crosses a transformation-relevant threshold. Its model was:

organismic situation → integrated registration → systemic translation → changed cellular world → fate bifurcation → durable incorporation

The claim of cellular history was deliberately demanding. A transient hormonal or immune effect was not enough. History required a consequence that outlasted the state that produced it, such as persistent chromatin accessibility, altered transcriptional responsiveness, stable metabolic reconfiguration, lineage redistribution, niche remodeling or clonal selection. The paper therefore distinguished systemic influence from durable incorporation and initiation from promotion or progression (Santos Albino, 2026a).

2.2 The missing question: why does the signal continue?

The earlier architecture permitted a systemic state to persist but did not explain the source of that persistence. Several possibilities remain:

The external event may truly continue. The tissue may remain injured or infected. A local cellular program may become autonomous. A damaged niche may continue to generate pathological signals. A neural or endocrine circuit may stay sensitized. Or the integrated organism may continue to realize the situation as unresolved even after its initiating event has ended.

These alternatives are not mutually exclusive. They can form a reciprocal loop. A persistent organismic state changes tissue; altered tissue changes afferent information; afferent information confirms the organismic state; and repeated systemic output further stabilizes the tissue. The problem is therefore not to choose mind, brain, cell or niche as the sole cause. It is to identify where persistence is first generated, where it becomes stored, and which intervention can still restore return.

2.3 The new contribution: an operator of persistence

The present paper proposes two nested claims. Conservatively, a measurable host persistence state can maintain physiological conditions after the precipitating event has ended. More strongly, SI may function as one organizer of that persistence within the CFI. SI does not add an immaterial force to biology. It names the historically singular organization through which an embodied organism maintains continuity across changing situations. When that organization fails to update the status of an event from present to past, the CFI may continue to produce the physiological conditions of response.

The strong intuition can be stated as “SI may rupture Ω.” The more rigorous formulation is an Ω-transition: SI may prevent the CFI from returning to its prior Ω regime and thereby contribute to the stabilization of a candidate attractor-like regime organized around a response that should have been transient. The term attractor substitution is reserved here for cases that meet the full dynamic criteria defined in Section 6.3.

> *SI may prevent the CFI from returning to its prior Ω regime and thereby contribute to the stabilization of a candidate attractor-like regime around a response that should have been transient.*

The distinction matters. Pathology is not always loss of order. Chronic inflammation, fibrosis, sensitization and pre-neoplastic fields can be highly organized and self-maintaining. The system may remain coherent while becoming less capable of returning. The failure is therefore not necessarily stability itself, but stability detached from the temporal conditions that originally justified it.

3. Operational vocabulary

3.1 CFI: embodied integration, not a second body

Integrated Functional Coherence (CFI) denotes the organized coordination through which a living organism maintains a workable unity across cellular, tissue, neural, autonomic, endocrine, immune, metabolic, biomechanical and behavioral processes. It is not located in one organ and does not exist outside the body. It is the embodied organization by which heterogeneous processes become mutually constrained and jointly available to the organism as a whole.

This paper does not require acceptance of the wider metaphysical concept of Fundamental Coherence (CF) developed elsewhere in HibriMind. CF is bracketed. No cosmological field is invoked as a biological cause. The empirical hypothesis requires only an integrated organism, historical identity, dynamic regulation and measurable cross-scale pathways.

3.2 SI: identity-bearing continuity, not a controller

Singular Identity (SI) denotes the unique, historically continuous form through which CFI becomes this organism's situated existence and experience. It includes, without being reducible to, memory, affective orientation, learned salience, expectation, interoceptive prediction, action readiness and relational history.

SI must not be pictured as a homunculus that issues commands. It is not a second agent hidden inside the organism. It is the identity-bearing organization of the organism itself. Much of its causal realization may be non-conscious or inaccessible to verbal report. The present model therefore does not equate SI with deliberate thought, autobiographical narrative or voluntary belief.

Within VIVENS, consciousness is provisionally formulated as the organism experiencing being alive. The experimentally conservative version of the present hypothesis concerns the material organization associated with that experience. Existing evidence cannot isolate phenomenality from the neural and

bodily processes through which it occurs. A future experiment may support causal roles for appraisal, memory or neural representation without demonstrating that phenomenal consciousness acts independently.

3.3 Ω: dynamic stability and the capacity to return

Ω denotes the dynamic stability of a coherent living unit under perturbation. It is not immobility and not a single molecule. A viable Ω regime permits change while preserving or recovering the relations required for continued functioning.

The phrase “rupture of Ω” can misleadingly suggest total collapse. In the present model, the critical event is often subtler: the previous Ω regime no longer recovers its former thresholds, while another regime becomes stable. We use Ω-transition for the loss of the prior return relation. Attractor substitution is the stronger dynamic claim that a different regime has become a genuine attractor; until convergence and local stability are demonstrated, the more cautious term is candidate attractor-like regime.

3.4 Cellular return capacity

Cellular return capacity is the competence of a cell or lineage to traverse a perturbation and recover its previous range of response thresholds and fate possibilities. That competence has two analytically distinct forms.

Intrinsic cellular return capacity is the recoverability retained by a cell or lineage under standardized reference conditions after removal from an altered host or niche. It can be estimated through isolation, organoid culture, reciprocal transplantation or another common-environment assay, while recognizing that the assay itself can perturb the state.

Effective cellular return capacity is the return that the cell can actually realize in its present organism, tissue and niche. It may be low even when intrinsic capacity remains intact because ongoing lesion, systemic output, matrix, innervation, immune ecology or neighboring cells keep the earlier range inaccessible. No single transfer assay proves either capacity; convergent in situ and reference-environment tests are required.

Neither form is a counter, a universal scalar or a substance stored in the cell. Both are multidimensional and context-sensitive, but intrinsic capacity assigns the retained competence to the cell or lineage under a reference environment, whereas effective capacity includes the constraints of the current host and niche. Relevant determinants include chromatin state, transcriptional memory, metabolic configuration, mitochondrial state, cell-cycle dynamics, DNA damage, replicative age, lineage, receptor distribution,

tissue position, extracellular matrix, neighboring cells, innervation, immune surveillance and organismic regulation.

A tissue may tolerate many small perturbations without losing return capacity, while a single intense, prolonged or strategically located event may exceed it. The relevant causal unit is not the number of episodes but the transformation produced by each episode relative to the existing capacity for return.

The central distinction is:

> *Non-return initially means absence of spontaneous effective return, not demonstrated loss of intrinsic return or absolute irreversibility.*

A cell may fail to return in situ yet remain intrinsically reconducible, or it may retain a cell-intrinsic memory that remains specifically resettable. Falvo et al. (2023), for example, showed that inflammatory memory in pancreatic acinar cells lowered the threshold for oncogenic transformation, while MAPK blockade restored that threshold. The state was persistent and consequential, but not absolutely fixed.

3.5 Two evidentiary levels

The conservative mechanistic core states that a post-event host persistence state can causally maintain systemic outputs during recovery, reduce effective cellular return capacity and, if sustained long enough, contribute to changes in intrinsic or niche-embedded return. This state is operationalized through measurable variables: activity-defined neural ensembles, autonomic and HPA dynamics, immune resolution, sensory feedback, tissue signaling and the time course of their normalization. No appeal to phenomenality or to SI is required to test this core.

The strong VIVENS hypothesis states that the historically singular organization named SI helps determine whether such a persistence state is formed, reinstated or terminated. SI therefore adds a claim of history- and content-specific organization; it is not a synonym for persistence. Evidence would need to show that recovery differs as a function of learned history or appraisal; that content-specific reactivation outperforms nonspecific arousal; that measured systemic outputs mediate the cellular outcome; and that identity-related variables add predictive value beyond cumulative exposure, lesion severity, cell state and niche.

A positive circuit experiment would support the conservative core directly. It would support the strong VIVENS hypothesis only when the manipulation is history-specific and discriminates identity-bearing organization from generic memory, stress or allostatic load (McEwen, 1998). Until then, SI remains a higher-level explanatory hypothesis anchored to, but not replaced by, its measurable realizers.

3.6 A minimal formal representation

Let E(t) denote the precipitating external event or input, L(t) the local lesion or inflammatory burden it produces, S(t) the identity-bearing organismic state, U(t) the integrated systemic output, $X_i(t)$ the state of cell i, H_i its prior history, and $N_i(t)$ its local niche. A minimal representation is:

$$L(t + 1) = Q[L(t), E(t), U(t), \text{local tissue dynamics}(t)]$$

$$U(t) = G[E(t), L(t), S(t), \text{afferent tissue feedback}(t)]$$

$$X_i(t + 1) = F[X_i(t), H_i, N_i(t), L(t), U(t)]$$

No validated update equation for SI is currently available. S(t) is therefore treated provisionally as an exogenous experimental variable constrained by history- and content-specific proxies, rather than as a closed dynamical state derived by the model. This preserves falsifiability without pretending that the dynamics of SI have already been identified.

Ordinary resolution is a sequence rather than a single timestamp: E(t) ends; L(t) falls through active resolution; S(t) updates; U(t) returns toward its prior range; and effective cellular return is recovered without evidence that intrinsic capacity has been lost. Organismically sustained non-return occurs when E(t) approaches zero and the initial L(t) is measured, but S(t) and feedback keep U(t) in a configuration that slows lesion resolution or continues to constrain X_i during recovery. Cellular or niche incorporation occurs when X_i or N_i retains the change after both L(t) and U(t) have normalized.

This representation is deliberately non-quantitative. Its purpose is to separate experimentally manipulable levels. It predicts that the same precipitating input and equivalent initial lesion can produce different later cellular states if recovery-phase S(t), U(t), H_i or N_i differ. L(t) must therefore be measured rather than inferred from the disappearance of E(t) or from apparently normal histology.

4. The proposed causal architecture

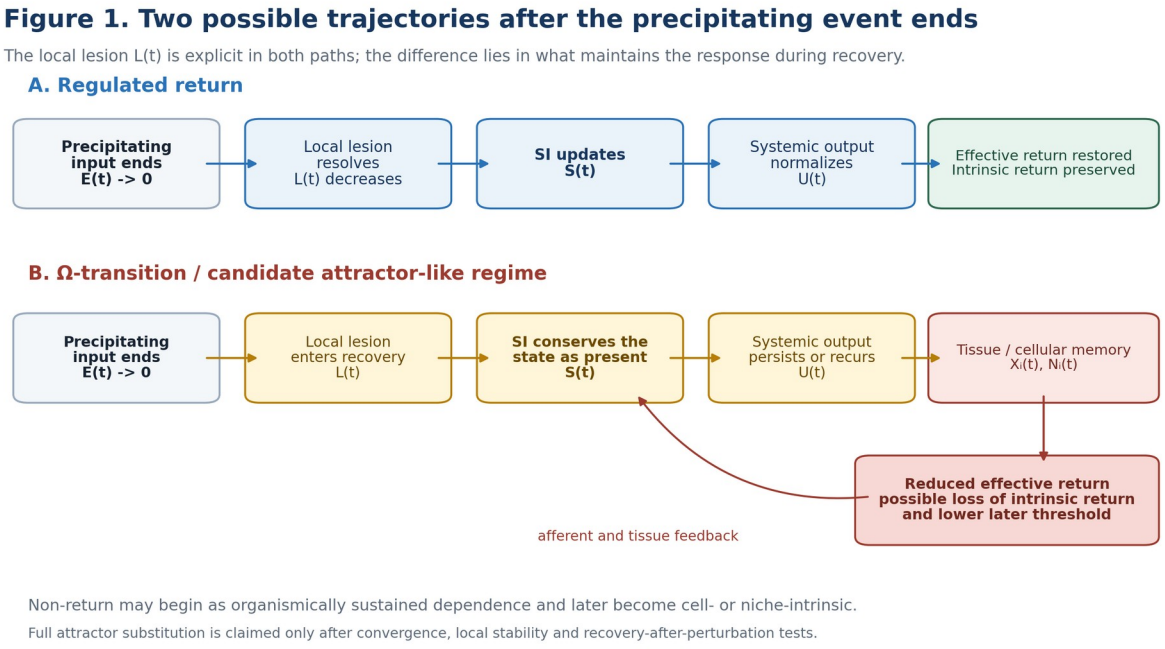

Figure 1. Regulated return versus the proposed transition toward a candidate attractor-like regime. SI denotes Singular Identity. L(t) denotes the local lesion or inflammatory burden; U(t) denotes integrated systemic output. Non-return can begin as organismically sustained dependence and later become incorporated into tissue or cellular memory. Full attractor substitution requires the dynamic tests specified in Section 6.3.

4.1 From event to individualized experience

An external event does not possess a fixed organismic meaning. Its biological realization depends on the organism that encounters it. Two individuals can undergo physically similar conditions but differ in learned salience, predictability, controllability, relational context, prior sensitization and available action. Even within one individual, the same event can be realized differently at different times.

This does not imply that meaning floats above physiology. Individualization is implemented through perception, memory, appraisal, interoception, autonomic patterning, endocrine dynamics, motor readiness, immune regulation and behavior. SI is the continuity that constrains this integration across time.

4.2 From individualized experience to a changed cellular world

The cell does not receive the identity of the organism as a semantic message. It receives material consequences. These can include neural transmitters and neuropeptides, catecholamines,

glucocorticoids, cytokines, immune-cell traffic, vascular tone, oxygenation, nutrient availability, mechanical strain, extracellular-matrix remodeling and altered signals from neighboring cells.

The earlier VIVENS paper called this a changed cellular world. The present model adds that the world can remain changed even after the initiating event has ended, because the organismic response is being internally maintained, repeatedly recalled or reinforced by tissue feedback.

4.3 From persistent response to a candidate attractor-like regime

During a successful adaptive episode, the response is coupled to its cause. As the precipitating input disappears, active resolution mechanisms reduce the local lesion, dismantle the response and recover earlier thresholds; resolution is a regulated program, not merely the passive decay of inflammation (Serhan & Savill, 2005). In the proposed transition toward an attractor-like regime, the coupling changes. The response begins to receive sufficient support from internal recurrence and feedback that it no longer depends on the original input, even though residual lesion may still be resolving.

The sequence is:

event ends → lesion enters recovery → SI does not fully update → systemic response persists or recurs → tissue adapts → altered tissue returns confirming signals → persistent response becomes self-supporting

At this stage, the organism may remain globally functional. The cost can be local and distributed: chronic low-grade inflammation, altered immune surveillance, fibrosis, metabolic reallocation, stem-cell priming or epigenetic memory. SI may preserve continuity at the scale of the organism while imposing a persistent adaptive regime on some of the cellular populations that sustain it.

The strongest version of the hypothesis is therefore:

> *The One can reduce the return capacity of the Many by conserving as present what has already ended.*

This statement describes downward constraint, not ownership. The organism does not micromanage cells. It changes the space of conditions within which their own regulatory systems act. The cell remains an active participant whose lineage, state and history determine what the organismic signal becomes locally. This preserves the distinction between basal cellular autonomy and organismic integration developed in the preceding LIMEN paper (Santos Albino, 2026b).

4.4 Two identities, not two minds

The architecture contains two successive individualizations.

First, organismic identity transforms an event into a particular integrated state. Second, cellular identity transforms that integrated state into a particular local fate. The same systemic signal may be transient in one cell, stabilizing in another, senescence-inducing in a third and transformation-permissive in a rare long-lived population.

Cell identity includes lineage, differentiation state, chromatin architecture, metabolic state, cell-cycle dynamics, mutational background, damage, spatial position and niche relations. It does not imply reflective consciousness. No semantic or cognitive claim is required here: the cell's existing organization determines which incoming differences alter its dynamics.

The proposed outcome is therefore relational:

$$\text{Outcome}_i = F(\text{event as realized by SI, CFI state, cellular history}_i, \text{niche}_i, \text{time})$$

This relation explains why a universal marker of irreversibility may not exist. Non-return is not a single substance inside all cells. It is a property of a trajectory: a cell with a particular history, within a particular niche, after a particular organismic event, no longer recovers its earlier transition landscape.

5. Empirical support for the component mechanisms

5.1 Representation at the organism scale can alter peripheral biology

In humans, Trabanelli et al. (2025) used virtual reality to present approaching avatars with visible signs of infection. Without physical pathogen contact, infectious avatars activated multisensory and salience networks and changed the frequency and activation of innate lymphoid cells. The study does not show lasting tissue memory, but it demonstrates one possible initial component: predictive neural registration of a possible threat can accompany a changed peripheral immune state. A 2026 addendum situated this result within earlier human work on visual and olfactory sickness cues, disgust and anticipatory immune markers, while identifying the integrated neural–immune demonstration as the advance (Trabanelli et al., 2025, 2026).

In mice, Koren et al. (2021) labeled insular-cortex neuronal ensembles active during colitis or peritonitis. Later reactivation of those ensembles reproduced components of the corresponding peripheral inflammatory response, while inhibition reduced disease expression. A centrally stored representation of a prior bodily state could therefore re-enter the causal chain and recreate aspects of that state.

Jin et al. (2024) identified distinct vagal sensory populations responsive to pro-inflammatory and anti-inflammatory cytokines and showed that manipulating the corresponding brainstem circuitry altered the

course of peripheral inflammation. Afference was not mere reporting. The body informed the brain, and the brain returned causal regulation to the body.

Liu et al. (2026) extended this architecture in experimental gastritis. Activity-defined neurons in the paraventricular hypothalamus participated in a PVN–dorsal motor nucleus of the vagus–stomach circuit and in HPA-axis regulation. Repeated activation produced inflammatory sensitization, stress reactivated the gastritis-associated ensemble, and inhibition of Hcn1 reduced neuronal excitability and gastric pathology. This is close to the proposed persistence mechanism: a neural ensemble formed during peripheral inflammation can later help maintain or reinstate disease.

None of these studies isolates conscious identity as an independent variable. Together, however, they establish that organism-level registration and neural memory can causally organize peripheral immune states after the original trigger has changed or disappeared.

5.2 Sensory nerves can regulate resolution and pathological tissue fate

Peripheral sensory neurons are not passive cables. Their local terminals can alter the very tissue state they sense. Lu et al. (2024) showed that nociceptor-derived CGRP regulates neutrophil and macrophage recruitment, clearance and phenotype, accelerating the transition from pro-inflammatory to pro-healing conditions. Neural signaling can therefore participate in the timing of resolution.

The opposite direction is equally important. Li et al. (2026) identified nociceptive sensory neuron-derived NGF as an instructive signal in tendon injury. Sensory denervation or neuronal NGF deletion reduced mesenchymal-stromal-cell commitment to a fibrotic fate, while TrkA inhibition partially reversed excessive fibrosis. This experiment demonstrates that a neural signal can help determine whether repair stabilizes as pathological tissue organization and that a state with poor spontaneous reversibility may remain intervention-sensitive.

These findings support a key VIVENS distinction. Effective return can be low because a neural and tissue environment continues to make return biologically inappropriate even when the cell retains intrinsic capacity. Alternatively, cell-intrinsic or niche memory can reduce return under a standardized reference environment. The two mechanisms can later reinforce each other.

5.3 Resolved injury can leave memory that lowers the oncogenic threshold

Del Poggetto et al. (2021) showed that a single transient episode of pancreatic inflammation produced long-lasting transcriptional and epigenetic adaptation. Long after histological resolution, later activation of oncogenic KRAS accelerated tumorigenesis. The original injury had ended, but the pancreatic epithelium had not returned to its previous molecular baseline.

Falvo et al. (2023) characterized a related inflammatory memory in lineage-traced pancreatic acinar cells. Twelve weeks after recovery, cells retained altered chromatin accessibility and H3K4me1 at metaplasia-associated regions. The memory reduced metaplastic response to another inflammatory insult but increased tumor initiation after oncogenic Kras activation. Crucially, MAPK blockade restored the lowered oncogenic threshold. The result supports three claims central to this paper: a single event can reduce return capacity; the later outcome depends on the context of recall; and non-return can persist without being absolutely irreversible.

Levra Levron et al. (2023) found that skin injury produced long-lived memory progenitors in distant, undamaged hair-follicle niches. Specific Lrig1-lineage cells remained transcriptionally and chromatin-primed after homeostasis had apparently been restored. The memory improved later repair but also created an epigenetic field that favored tumor onset. History was spatially selective, lineage-specific and not confined to cells that directly repaired the first wound.

Related systems broaden the substrate of biological memory. Naik et al. (2017) showed that skin epithelial stem cells retain inflammatory memory that accelerates later repair. De Laval et al. (2020) identified C/EBPβ-dependent epigenetic memory in hematopoietic stem cells that generated trained innate immunity. Alonso-Curbelo et al. (2021) showed that tissue damage can cooperate with oncogenic KRAS through an epigenetic program that initiates pancreatic tumorigenesis. These findings do not establish SI as the upstream cause, but they show that event history can be stored in long-lived epithelial, hematopoietic and pre-neoplastic populations.

These studies do not show that organismic identity caused the memory. They establish the downstream substrate that the present hypothesis requires: a resolved event can leave durable, distributed and malignancy-relevant cellular history.

5.4 Cellular identity changes the meaning of the same event

Sahu et al. (2021) exposed different human cellular identities to the same liver-cancer-associated oncogene combination. CTNNB1, TERT and MYC induced senescence in fibroblasts and primary hepatocytes, whereas fibroblasts first converted into an induced hepatic progenitor state became proliferative and tumorigenic. Driver mutations were not sufficient to specify outcome. Lineage and differentiation state were constitutive parts of the causal event.

Chen et al. (2025) showed that cell-cycle duration distinguished transformation-prone from resistant lineages across several mouse tumor models. Cells carrying comparable tumor-suppressor loss did not share the same fate; intrinsic lineage dynamics helped determine whether an oncogenic lesion became a tumor.

Li et al. (2026) demonstrated that melanoma cells can form AP-1-dependent memories during acquisition of therapy resistance. Low-dose exposure adapted cells to later high-dose treatment; transient and probabilistically arising expression states could be encoded into persistent chromatin accessibility; and related "twin" cells exposed to the same dose produced more similar resistance outcomes than unrelated cells. Prior cellular state therefore constrained later response without completely determining it.

Together, these studies support the second individualization in the model:

> *A cell does not merely receive an event. It receives it as the cell it has already become.*

5.5 A complete closed loop is biologically possible

Wei et al. (2026) demonstrated a sensory-to-sympathetic tumor–brain circuit in mouse lung adenocarcinoma. Tumors engaged Npy2r-expressing vagal sensory neurons, brainstem processing increased sympathetic output, and β2-adrenergic signaling in alveolar macrophages suppressed anti-tumor immunity. Genetic, pharmacological or chemogenetic disruption of the circuit inhibited tumor growth.

This result concerns an established tumor, not pre-neoplastic initiation. It nevertheless demonstrates that such a reciprocal architecture is biologically realizable in this model:

altered tissue → afferent signal → central integration → efferent output → altered tissue ecology

The present hypothesis asks whether a homologous loop can operate earlier, before malignancy, and help convert a transient organismic response into pre-neoplastic cellular history.

6. Return, non-return and irreversibility

6.1 Four distinguishable regimes

Regime	Primary maintainer	Return test	Cancer-relevant implication
Resolved response	External event has ended; organismic and tissue outputs normalize.	Earlier response thresholds recover without targeted intervention.	No durable memory demonstrated.
Organismically sustained non-return	Neural, autonomic, endocrine, immune or behavioral recurrence continues to recreate the local conditions.	Normalize the host or transfer cells to reference conditions; effective return should recover if intrinsic capacity is preserved.	Prolonged exposure can create a field permissive to incorporation.

Regime	Primary maintainer	Return test	Cancer-relevant implication
Cell- or niche-incorporated memory	Chromatin, transcription, metabolism, lineage, matrix or local ecology maintains the altered state.	Host normalization is insufficient; common-environment and niche-transfer tests localize intrinsic versus niche-embedded memory.	Later oncogenic challenge can produce a larger effect.
Autonomous fixation	Mutation, clonal selection or self-sustaining pathological ecology dominates.	Neither event removal nor simple systemic normalization restores the prior state.	Prevention has become treatment of established neoplastic organization.

Table 1. Four regimes of return. The distinction is based on what currently maintains the state, whether effective and intrinsic return diverge, and which intervention recovers earlier thresholds.

The table prevents a common error: treating every failure to return as absolute irreversibility. A state can require intervention without being fixed. Conversely, histological recovery can conceal molecular or lineage memory.

6.2 Candidate attractor-like regimes are not simple accumulation

A candidate attractor-like transition should not be modeled as a counter in which a tissue tolerates a fixed number of experiences and fails on the next. Repetition can matter because it reinforces circuits, extends exposure, prevents resolution or repeatedly recruits long-lived cells. But one sufficiently intense, prolonged or strategically located event can also produce a transition.

The decisive variable is the transformation imposed relative to the remaining capacity for return. A hundred perturbations may be fully disincorporated. One perturbation may reach a stem-cell population, remodel a niche, create a self-reinforcing chromatin state or recruit neural feedback strongly enough to establish a new regime.

A candidate attractor-like regime may display hysteresis: the conditions required to leave the state can differ from those that produced entry. Removing the original event may no longer be sufficient, and a targeted intervention may be needed to recover the earlier response range. Hysteresis is evidence of path dependence, however, not by itself proof of an attractor.

6.3 Minimum criteria for an attractor claim

The term attractor substitution should be reserved for a defined state vector Z(t), an explicit observation timescale and a bounded dynamical regime. A full claim requires: persistence after removal of the initiating input; convergence of distinct nearby trajectories toward the same regime; local stability under small perturbations; return toward the candidate regime after such perturbations; a history-dependent entry and exit relation, potentially expressed as hysteresis; and a targeted perturbation capable of moving the system into an alternative stable response range.

Persistence and hysteresis alone are insufficient. Designs must distinguish a candidate attractor from slow relaxation, measurement lag, cumulative structural damage and an absorbing state that cannot recover. Experimental hysteresis during epithelial–mesenchymal transition provides a concrete example of path dependence (Celià-Terrassa et al., 2018), not a general definition of an attractor. Until convergence and local stability are demonstrated, the appropriate terms are persistent state, hysteretic state or candidate attractor-like regime.

6.4 When does SI cease to be the primary maintainer?

The model predicts a temporal transfer of causal weight.

Early in the process, persistence may depend mainly on organismic representation and systemic output. Later, cellular chromatin, tissue matrix, immune ecology or clonal composition may maintain the state even if the organismic configuration changes. At that point, SI is no longer required for persistence, although tissue feedback may continue to affect it.

The causal sequence may therefore be:

experience crystallizes in SI → response persists in CFI → tissue and cells incorporate the response → incorporated history acquires partial autonomy

This transfer is experimentally important. An intervention directed at organismic regulation may work before cellular incorporation and fail after it. Conversely, an epigenetic or niche intervention may restore return after the initiating experience has long been resolved.

7. Predictions that distinguish the hypothesis

The proposal is scientifically useful only if it predicts outcomes not already entailed by local injury, generic stress or cell-autonomous memory.

7.1 Recovery-phase physiology should matter independently of peak damage

If organismically sustained non-return exists, then neural, autonomic, endocrine or immune activity during recovery should predict later cellular memory even after controlling for the severity of the initial lesion. Peak injury and later non-return should be separable.

7.2 Post-event manipulation should alter later memory

Manipulating a predicted afferent, central or efferent circuit after the precipitating input has ended and peak lesion has been quantified should change later chromatin, lineage or transformation susceptibility.

The intervention can occur during early recovery before histological resolution, at the resolution boundary or after resolution. Its effect must remain distinguishable from a change in L(t).

7.3 Reactivation should reinstate a tissue-relevant state

Selective reactivation of an ensemble formed during the initial event should reinstate relevant systemic or local signals and increase the probability of memory formation or recall in the target tissue. A nonspecific arousal manipulation should not reproduce the full effect.

7.4 Host and cell history should be jointly necessary in an intermediate regime

Cells taken from an affected organism and tested in a standardized naïve environment probe intrinsic return capacity. Naïve cells placed in an affected host or niche probe environmental constraint on effective return. Reciprocal transfer across all four host–cell combinations can reveal an intermediate regime in which intrinsic and environmental maintenance are jointly causal.

7.5 Rare lineages should carry disproportionate history

Long-lived stem, progenitor or niche-defining populations should be more likely than short-lived neighboring cells to conserve the event and alter later transformation thresholds. A bulk-tissue average may therefore miss the causal population.

7.6 There should be no universal molecular marker of non-return

If non-return is relational, different tissues and lineages may reach it through different combinations of chromatin, metabolism, matrix, innervation and immune state. A common signature may exist at the level of dynamics—persistence, hysteresis, reduced recoverability—without a universal single molecule.

8. Experimental program

8.1 A recovery-phase intervention design

The first decisive experiment can be built on a model in which transient injury already produces later cancer-relevant memory, such as caerulein-induced pancreatitis followed by delayed Kras activation.

Animals would receive the same transient inflammatory input. A single primary recovery-phase output would be selected a priori from a candidate afferent, central or efferent pathway. Peak local lesion, inflammatory burden and early recovery kinetics would be quantified with preregistered equivalence margins, rather than treated as equivalent merely because a null-hypothesis test is non-significant. The

selected output would then be manipulated in three predefined windows: early recovery after peak injury but before histological resolution; the resolution boundary; and late post-resolution.

The primary endpoint would not be immediate inflammation. Effective return would be measured in situ, while intrinsic return would be estimated weeks later under standardized reference conditions through organoid or reciprocal-transfer assays. Both would be related to single-cell transcriptomics and chromatin accessibility, lineage tracing, response to a second non-oncogenic challenge, and tumor initiation after delayed oncogenic activation.

Support for the conservative hypothesis would require a recovery-phase intervention to restore effective return, preserve intrinsic return or reduce later tumor initiation without materially changing the measured peak lesion. L(t) must continue to be measured during the intervention so that a benefit caused by preventing renewed damage is not misclassified as direct restoration of return. If only interventions delivered during the acute lesion work, the organismically sustained non-return model would be weakened. If early-recovery intervention works but late post-resolution intervention fails, the interval between them would estimate the transfer of causal maintenance into cell or niche memory.

Causal interpretation must distinguish a direct recovery-phase path $U(t) \rightarrow X_i(t)$ from a lesion-mediated path $U(t) \rightarrow L(t) \rightarrow X_i(t)$. If manipulating U(t) changes later memory only because it renews or prolongs L(t), the direct $U \rightarrow X$ claim is not supported; the result remains compatible with the broader coupled model through $U \rightarrow L \rightarrow X$. Time-resolved measurement and mediation analysis should therefore estimate the lesion-mediated component rather than treat it as a nuisance.

8.2 A four-condition causal matrix

Condition	Prior transient injury	Standardized recovery output	Interpretive role
A	Absent	Clamped low / blocked	Baseline profile under the control manipulation.
B	Present	Clamped low / blocked	Injury memory when the candidate output is minimized.
C	Absent	Clamped high / imposed	Tests output sufficiency without a locally primed substrate.
D	Present	Clamped high / imposed	Tests the injury × output interaction predicted by the coupled model.

Table 2. Orthogonal separation of transient injury from a standardized recovery-phase output. The same output manipulation, timing and dose are applied with and without injury; event-specific ensemble reactivation is tested separately.

This true 2 × 2 matrix estimates the main effects of injury and one pre-specified standardized recovery output and, critically, their interaction. The manipulated output can be a defined autonomic firing pattern, endocrine exposure or mediator module, but its timing, dose and measurement must be identical across injured and uninjured groups. Condition C therefore imposes an efferent or systemic

output; it does not “reactivate” an event-specific ensemble that was never formed. A separate necessity experiment would inhibit an ensemble tagged during injury in previously injured animals.

Every active and blocking manipulation requires a matched sham control for instrumentation, viral delivery, drug exposure and handling. The imposed output in the uninjured condition must be shown not to create a new local lesion, using the same L(t) measures applied to injured animals. Equivalence of the initial lesion across injured conditions must satisfy the preregistered margins; absence of a statistically significant difference is not sufficient.

8.3 Time-resolved transfer experiments

At several recovery time points, epithelial cells or organoids would be isolated from previously injured and control animals. Their behavior would be tested in a standardized naïve culture, in naïve hosts and in hosts whose predicted persistence circuit remains active. Reciprocal transfer would identify when low effective return becomes detectable as intrinsic cell memory, niche-embedded memory or both.

The key output is not binary reversibility. It is a map of dependence:

organism-dependent → organism–cell coupled → cell/niche incorporated → autonomous or clonally stabilized

8.4 Single-cell prospective prediction

Cells should be characterized before perturbation, tracked through injury and recovery, and challenged later. Lineage barcoding, live reporters, sister-cell designs and single-cell multi-omics can test whether pre-event identity predicts which cells return, which remain primed and which show reduced oncogenic thresholds.

The strongest design would perturb the predicted identity variable itself. If cell-cycle duration, AP-1 state, chromatin accessibility or metabolic configuration predicts non-return, causal modification of that feature should alter outcome. Prediction without intervention would remain compatible with an unmeasured common cause.

8.5 Approximating SI experimentally

SI cannot currently be manipulated as a complete biological variable. Experiments must therefore use constrained proxies: appraisal and controllability, activity-defined neural ensembles, interoceptive circuits, autonomic configuration, learned threat recurrence or persistent HPA and immune states. To bear on the strong VIVENS hypothesis, a proxy must retain individual-history or content specificity and must outperform matched nonspecific arousal or generic stress manipulations.

Positive results would support the causal efficacy of identity-related organismic organization, not prove that phenomenal consciousness independently caused the cellular effect. Stronger support for SI would require mediation through measured systemic outputs and incremental prediction beyond cumulative exposure, lesion severity, cell state and niche. The distinction must remain explicit.

8.6 Falsification conditions

The conservative mechanistic core should be narrowed or rejected within a defined model if, after equivalence-bounded measurement of the initial lesion, recovery-phase U(t) adds no predictive information beyond L(t), cumulative exposure and cell or niche state; if manipulating U(t) in preregistered recovery windows changes neither later return capacity nor cellular memory; if putative effects are explained entirely by renewed lesion; or if host–cell transfer shows complete cell autonomy from the earliest recovery point.

The strong VIVENS hypothesis must be tested inside the subclass it actually claims: trajectories in which a history- or content-specific organismic state is present and experimentally distinguishable from generic arousal or allostatic load. It would be weakened if eliminating that specific recurrence leaves U(t) and later memory unchanged; if reinstating it produces no greater effect than an arousal-matched control; or if models containing the history-specific variable do not improve preregistered prediction or causal intervention over history-free models.

The paper does not propose SI as a necessary operator for all non-return. Radiation, toxins, infection, mutation and local tissue damage can generate cellular memory outside the proposed subclass. Such cases define the boundary of the claim; they do not falsify a non-universal causal contribution of SI within the relevant trajectories.

9. Prevention before fixation

The distinction between non-spontaneous return and absolute irreversibility creates a preventive window. Histology may appear normal while response thresholds, chromatin, lineage or niche remain altered. Conversely, a persistent cellular state may still be reset by blocking the pathway that recalls or stabilizes it.

Potential intervention levels follow the causal architecture. At the organismic level, one could target the neural or physiological persistence that continues to represent the event as active. At the systemic level, one could restore resolution in autonomic, endocrine or immune outputs. At the tissue level, one could alter matrix, innervation, immune ecology or stem-cell niche. At the cellular level, one could target

memory-maintaining chromatin or signaling pathways. These are research directions, not treatment recommendations.

The optimal intervention may be phase-specific. Before incorporation, restoring organismic resolution may be sufficient. During coupled persistence, combined organismic and tissue intervention may be necessary. After autonomous fixation, prevention has become treatment of an established cellular ecology.

This model therefore reframes early detection. The relevant object is not only an oncogenic mutation or a histological lesion. It may also be a loss of return competence: effective return can remain constrained after apparent recovery, and intrinsic cell or lineage capacity can change before a conventional lesion is visible.

10. Ethical and conceptual safeguards

10.1 No psychogenic monocausality

The model does not claim that emotion or identity is a dominant cause of human cancer. Cancer remains multicausal. Inherited susceptibility, somatic mutation, aging, infection, radiation, toxins, inflammation, tissue architecture, immune escape, metabolism, chance and selection remain indispensable.

10.2 No patient blame

SI is historically constituted and largely non-voluntary. Persistent organismic states are not choices. A person cannot simply decide to stop an embodied response, and failure to return is not a moral or psychological failure. Even if the hypothesis is supported, it would not license the statement that a patient caused disease by thinking, feeling or failing to “let go.”

10.3 No dualism

SI does not act from outside the body. Every proposed effect is realized through neural, autonomic, endocrine, immune, vascular, metabolic, biomechanical and behavioral processes. The model is downward-causal only in the organizational sense: higher-scale state changes lower-scale boundary conditions through materially implemented pathways. This is organizational downward causation: its legitimacy depends on explicit scale relations and materially specified constraints, not on an extra biological force (Green, 2018).

10.4 No universal conscious mediation

Many relevant circuits operate without reportable conscious experience. The model distinguishes identity-bearing organismic organization from explicit awareness. Evidence for neural memory or appraisal-linked physiology does not by itself demonstrate a causal role for phenomenality.

10.5 No premature clinical translation

The cited evidence comes from heterogeneous mouse models, cultured cells, human virtual-reality experiments and established tumors. It cannot be assembled into a clinical protocol by conceptual continuity alone. Each proposed intervention requires model-specific validation, safety testing and separation of beneficial adaptive memory from pathological persistence.

11. Discussion

The simplicity of the hypothesis is not that consciousness mysteriously causes cellular change. It is that an organism may continue to produce a response because, within its identity-bearing organization, the event has not become biologically past.

The complexity lies in transmission. Neural ensembles, afferent and efferent circuits, endocrine signals, immune dynamics, metabolism, vascular states, matrix and cell–cell interaction describe how persistence crosses scales. Cellular chromatin, lineage, cell-cycle state and niche describe how the same organismic world becomes different histories in different cells.

This architecture offers a solution to three problems.

First, it explains why recovery at one scale can coexist with non-return at another. The organism may appear behaviorally recovered while a neural circuit remains sensitized; tissue histology may normalize while stem cells retain chromatin memory; systemic physiology may later normalize while a pre-neoplastic field has acquired autonomy.

Second, it explains why irreversibility lacks a single marker. The transition is not one molecule but a change in dependency and trajectory. What matters is whether the previous state remains spontaneously accessible, whether a targeted intervention can restore it, and where the memory is currently maintained.

Third, it identifies a causal role for the emergent organism without introducing dualism. The Many constitute the integrated organism. The identity of that organism can then return upon the Many by

changing the conditions in which they act. The One does not own the cells and does not replace their agency. It constrains the field in which their histories are formed.

The proposal also refines the concept of pathological crystallization. A regulatory response becomes pathological not merely because it is intense, but because it loses the capacity to terminate with its cause. Emotion, neural representation, immune configuration and cellular memory may represent different scales of this same temporal failure. The response first persists within the organism; then it can become incorporated by the cells that have been adapting to it.

The deepest causal reversal is therefore possible:

> *Ω makes SI possible. SI may prevent return to the prior Ω regime. The resulting Ω-transition can become incorporated into the cellular populations that sustain SI.*

This recursion is the novel contribution of the paper. It transforms "organismic experience becomes cellular history" from a one-way descent into a closed, historically structured loop.

12. Conclusion

The paper proposes a testable class of multiscale causation:

event → individualized organismic experience → persistent identity-bearing state → recurrent systemic realization → reduced cellular return capacity → incorporated history → lower transformation threshold

The hypothesis does not compete with mutation, tissue organization or cellular autonomy. It specifies how their causal field may be altered by the organism that cells collectively constitute.

The nuclear formulation is:

> *A cell may fail to forget because the organismic identity within which it lives continues to realize a world in which the event has not ended.*

Initially, the cell may not be malfunctioning. It may be adapting accurately to persistent conditions. Pathology begins when the adaptation outlives those conditions, stabilizes across cellular or tissue memory, and acquires autonomy from the experience that produced it.

The preventive opportunity lies before that autonomy is complete. The crucial experimental task is to distinguish a cell or lineage that has lost intrinsic return capacity from one whose effective return is still being blocked by the organism, tissue or niche.

Declarations

Author contributions. Joaquim Santos Albino: conceptualization, theoretical development, literature synthesis, methodology, writing, review and final approval.

Funding. No external funding was received for this work.

Competing interests. The author declares no competing interests.

Data availability. No new datasets were generated or analyzed for this conceptual paper.

Ethics statement. This paper reports no original research involving human participants or animals.

Use of generative AI. Generative AI (OpenAI ChatGPT) was used during iterative conceptual dialogue, literature organization, language refinement, reference cross-checking, diagram preparation and document formatting. It was not treated as an author. Joaquim Santos Albino directed the argument, made all substantive decisions and accepts full responsibility for the manuscript.

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