

THE FRONTIER MEDICAL CONCEPT PAPERS

VOLUME 7 — THE BODY'S SECRET DECISIONS

Biological Triage, Compensatory Illness, Injury Memory
and the Limits of Recovery

20 independent theoretical essays · Prepared 16 October 2026

Mechanistic hypotheses, competing explanations and falsifiability. These are conceptual manuscripts, not clinical trials or systematic reviews.

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The Body Has a Hidden Triage System

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An injured organism may divert resources away from reproduction, growth or nonurgent performance toward maintaining perfusion, glucose supply, immune defense and immediate survival. This is not a claim that organs consciously vote; it is a proposal that interacting endocrine and neural signals create a functional hierarchy of demand. The critical novelty is to study failures of *return* to ordinary allocation after a threat ends.

The biological route

Inflammation can reduce appetite and increase hepatic acute-phase synthesis; sympathetic and HPA-axis output redistributes cardiovascular demand; severe illness changes muscle proteolysis and substrate allocation. A tissue-level signal can therefore have remote energetic consequences. One can imagine a regulatory network responding to inflammatory cytokines, leptin, insulin, glucocorticoids and autonomic afferents in concert, rather than each signal commanding a single organ. A persistent signal may stabilize an initially useful allocation at substantial long-term cost.

An illustrative scenario

Suppose a patient with a serious infection sleeps poorly, loses appetite and temporarily experiences reduced exercise capacity. Those changes can be maladaptive in excess but may also accompany cytokine-mediated prioritization of host defense. A useful causal diagram would chart the degree of infection, the autonomic and endocrine response, the cost in skeletal muscle and cognition, and the point at which recovery of ordinary function should begin. The critical observation is not that symptoms occur together, but whether their timing follows an identifiable regulatory response.

Alternative explanations and limits

The strongest objection is that what looks like strategic reallocation may simply be nonspecific sickness behavior, drug effects or organ injury. The metaphor of triage can conceal heterogeneity and lead to teleological claims that biology never demonstrated. Acute tradeoffs should not be assumed in all chronic diseases.

What would change the conclusion?

An informative comparison would track energy expenditure, hormone rhythms, muscle turnover and functional recovery across an acute illness and convalescence. The theory weakens if the presumed hierarchy cannot predict which functions are suppressed, or if each change is explained better by independent tissue injury. The timing of reversal is as important as the initial response.

The distinctive prediction

If this is a true allocation program rather than a coincidental cluster of symptoms, suppressed functions should return in a reproducible sequence as the threat subsides. That sequence would provide stronger evidence than simply observing temporary fatigue or appetite loss during infection.

Clinical interpretation

Clinically, distinguishing a protective allocation from a pathological continuation matters because neither indiscriminate stimulation nor deliberate deprivation is automatically appropriate. The concept is a map for questions, not a protocol or a justification for withholding treatment. It helps ask when the initiating threat is gone but an organism remains organized around emergency.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

Pyrkov TV et al. Longitudinal analysis of blood markers reveals progressive loss of resilience and predicts human lifespan limit. Nat Commun. 2021.
<https://www.nature.com/articles/s41467-021-23014-1>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 02 · THE BODY'S SECRET ECONOMY

The Organ That Steals Energy from the Others

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

A chronically inflamed organ could alter whole-body function by exporting endocrine, neural and inflammatory signals that change energy distribution elsewhere. Calling that organ an energy thief is metaphorical: a damaged liver or gut is not necessarily burning all the calories. The sharper question is whether a local pathology can bias systemic allocation enough to produce remote fatigue, muscle loss or cognitive effects.

The biological route

Persistent IL-6 or other inflammatory signaling can promote hepatic acute-phase production and muscle catabolism; cholestasis or renal dysfunction can change circulating metabolite pools; autonomic reflexes and vagal afferents can signal visceral distress centrally. This provides multiple paths by which a local organ becomes an input into whole-body homeostasis. The magnitude, timing and context of those paths would determine whether an organ-level lesion creates disproportionate systemic symptoms.

An illustrative scenario

A patient with inflammatory bowel disease and persistent fatigue could have several mechanisms in parallel: iron deficiency, disturbed sleep, nutritional restriction, inflammatory mediators and drug effects. The interesting question is whether gut-derived signaling adds something specific after those common contributors are measured. An observational association between bowel activity and fatigue would be expected even without a special energy-allocation program; the hypothesis must offer more than association.

Alternative explanations and limits

Advanced chronic disease commonly brings anemia, malnutrition, sleep disruption, infection, medications and inactivity. These could account for remote symptoms without invoking a coordinated priority shift. Demonstrating cytokine elevation alone would not identify the initiating tissue or prove that source removal would restore systemic energy.

What would change the conclusion?

Compare the same inflammatory burden across different organs, looking at downstream endocrine changes and function. If a proposed source-organ signature does not predict reversible remote impairment, the allocation model becomes descriptive rather than causal. Selective improvement after effective treatment of the source would be suggestive but still open to confounding.

The distinctive prediction

The distinctive prediction is asymmetry: two equally ill patients with different affected organs might exhibit different patterns of remote energy use even after controlling for systemic inflammation. That would suggest an organ-specific signaling contribution rather than a universal sickness response.

Clinical interpretation

This question could motivate fuller interpretation of unexplained multisystem symptoms without displacing ordinary differentials. The concept should never encourage unproven removal of organs or rejection of necessary anti-inflammatory, antimicrobial or disease-specific treatment.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

When Symptoms Become Load-Bearing Structures

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An abnormal physiological response may become essential compensation for an unresolved problem. Suppress the response abruptly and the hidden deficit may become more dangerous. Tachycardia supporting low stroke volume, high respiratory drive maintaining acid-base status and neuroendocrine activation during critical illness are familiar examples. The radical extension is to look for clinically important dependencies on persistent adaptations in chronic disease.

The biological route

Cardiac output is heart rate multiplied by stroke volume; when filling or contractility constrains stroke volume, tachycardia can be partly compensatory, although sustained tachycardia has costs. Similarly, compensatory hyperventilation can reduce carbon dioxide during metabolic acidosis. Feedback control makes an outward symptom both a signal of disorder and part of the current solution. Suppression can change the system more than expected because multiple variables were coupled.

A deeper interrogation

Consider an adult with severe left-ventricular dysfunction whose resting heart rate is high. The number itself is outside the usual desired range, but lowering it abruptly without appreciating the contribution of heart rate to cardiac output can be perilous in an unstable state. This does not mean tachycardia is always beneficial: sustained elevation increases myocardial oxygen demand and can worsen filling. The useful question is the sign of the *net effect at this point in time*. Another patient may have a high respiratory rate because metabolic acidosis has prompted compensatory carbon dioxide elimination. Interfering with the pattern before treating the acid-base disturbance would alter pH; treating the cause changes the need for compensation. The same symptom therefore cannot be assigned a universal biological value. A proposed general rule must begin with a local state equation, identify what is being defended, and then examine costs across time. This avoids the sentimental claim that every symptom is the body's wisdom. It also avoids the equally simplistic assumption that symptoms are meaningless outputs to silence. The deeper proposal is that some clinically visible signs are carrying information and work simultaneously. Care must distinguish that dual role using measurements and patient-specific evidence.

Alternative explanations and limits

This idea is easily misused. Fever, hypertension, anxiety, pain and inflammation are not automatically protective merely because they emerge during illness. Many cause direct injury and respond beneficially to treatment. A true compensatory role must be demonstrated at the particular severity, stage and patient rather than assumed from evolutionary language.

What would change the conclusion?

An explicit model should predict the direction and magnitude of downstream physiological change after modifying the sign. If adjustment improves organ-level performance and safety without unmasking the proposed deficit, the compensation hypothesis is weakened. Dynamic measures and counterfactual scenarios matter more than an isolated abnormal number.

The distinctive prediction

A compensation model makes a risky commitment: modify the abnormality while holding the upstream deficit fixed and a specific protected variable should worsen. If this does not occur, calling the symptom load-bearing is unjustified in that setting.

Clinical interpretation

The clinical message is not to tolerate dangerous symptoms: it is to distinguish the response from the constraint producing it, and to treat both with appropriate monitoring. In urgent contexts, established stabilization methods remain paramount.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

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VOLUME 7 · PAPER 04 · THE BODY'S SECRET ECONOMY

The Recovery Tax: Why Healing Can Temporarily Make You Feel Worse

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Repair has a metabolic price. The body must synthesize proteins, rebuild extracellular matrix, clear damaged cells and restore barriers; these processes draw on nutrients, oxygen and ATP. Could that demand temporarily reduce perceived energy available for other tasks during true convalescence? This is a plausible allocation question, not license to redescribe every adverse symptom as a desirable detoxification event.

The biological route

Wound healing transitions through hemostasis, inflammation, proliferation and remodeling, with changing cellular populations and substrate needs. Cytokines can alter appetite and sleep; mitochondrial and immune demands rise during repair. Physical fatigue may therefore persist after

the most visible sign of illness improves. However, actual deterioration, unstable vital signs, fever recurrence or neurological symptoms may reveal a complication rather than an acceptable recovery tax.

An illustrative scenario

Imagine a wound that is visibly smaller but a patient whose energy is not yet restored. Tissue reconstruction is one candidate explanation. Another is persistent infection, anemia or poor sleep. If new fatigue appears alongside tachycardia, hypotension or other signs of instability, it would be unsafe to label the syndrome an ordinary repair tax. The model gains credibility only when improvement in tissue integrity, energy expenditure and functional recovery can be followed together.

Alternative explanations and limits

Pain, deconditioning, anemia, infection, inadequate food intake, medication effects and mood changes often explain persistent fatigue. These alternatives must be sought before inferring hidden regenerative expenditure. Increased metabolic demand is not proof that repair is successful; tumors and pathological inflammation are also metabolically active.

What would change the conclusion?

The model should specify objective repair markers, resting energy expenditure, functional output and recovery trajectories, and distinguish improvement of tissue integrity from worsened patient outcomes. It fails if fatigue tracks only deconditioning or adverse effects, with no independently measurable association to tissue rebuilding.

The distinctive prediction

An authentic recovery tax should track objective tissue rebuilding and diminish as repair concludes. An illness that continues worsening, produces new organ impairment or lacks evidence of repair should not be protected by this interpretive label.

Clinical interpretation

For public education, the useful distinction is that recovery is active physiology but warning signs must not be celebrated as cleansing. The hypothesis favors careful pacing, adequate nutrition and clinical assessment when symptoms are disproportionate or escalating.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

Biological Bankruptcy: The Collapse of Physiological Reserve

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

A disease may appear to begin suddenly when many compensating systems reach their limits at nearly the same time. In such a model, the visible event is a threshold crossing, not the first moment of pathology. Biological bankruptcy is an analogy for exhausted reserve—not a quantifiable debt already established in medicine.

The biological route

Circulatory reserve, renal concentrating capacity, endocrine feedback, respiratory compensation and immune control each operate within finite ranges. Mild impairment in one compartment can be masked by another. If reserve narrows across several compartments, a relatively modest new challenge can produce a sharp clinical transition. Feedback loops explain why signs remain quiet until a constraint becomes binding.

An illustrative scenario

A person might maintain ordinary blood pressure through progressive sympathetic activation and decreasing vascular reserve. Another might have stable glycemia only through higher insulin secretion before dysglycemia becomes apparent. These examples suggest how one apparently normal output can conceal increasing control effort. But neither example proves a single hidden debt across organs. The model becomes useful when the cost of maintaining normality can itself be measured.

Alternative explanations and limits

Acute events are often genuinely acute: thromboembolism, infection, poisoning or hemorrhage may produce sudden injury without prolonged hidden exhaustion. The concept cannot be used to reinterpret every emergency as a failure of wellness practices.

What would change the conclusion?

Compare multiple reserve measurements with their trajectories rather than one laboratory snapshot. The hypothesis predicts that declining recovery speed, rising variability or increasing effort to maintain normal outputs precede at least some clinical decompensations. If there are no measurable precursors or cross-system interactions, the model contributes little beyond retrospective storytelling.

The distinctive prediction

The model predicts that combinations of declining reserve will forecast decompensation more accurately than their individual measurements. The threshold should also have a physiologically

interpretable limiting variable rather than a statistical cutoff selected after the fact.

Clinical interpretation

A practical consequence is respect for both acute diagnosis and longer history. Acute emergencies require immediate conventional evaluation; a reserve-oriented account can complement rather than replace the search for the precipitating event.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

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<https://www.nature.com/articles/s41467-021-23014-1>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 06 · THE BODY'S SECRET ECONOMY

Why Some People Survive Disease but Never Recover

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Surviving an insult is a different achievement from regaining baseline function. An adaptive state can persist when neural, immune and metabolic feedback has reorganized around a prolonged threat. The proposed explanation for a subset of post-illness syndromes is not an uncured pathogen or a single missing nutrient, but a path-dependent configuration that is slow or unable to unwind.

The biological route

Critical illness can produce muscular weakness, altered autonomic output, sleep disruption, microvascular injury and inflammatory changes. Each can reinforce the others: inactivity worsens conditioning, dysautonomia alters exertional tolerance, poor sleep impairs recovery and symptom fear changes behavior. The organism can become stable in a lower-performance state even after the original insult ends.

An illustrative scenario

After intensive care, a survivor may no longer have the infection that precipitated admission, yet remain severely weak. Muscle injury, sedation effects, neuropathy, poor sleep and delirium-related consequences may all matter. A recovery-state theory should first describe these well-characterized problems; only then should it investigate whether mutually reinforcing feedback loops explain

persistence beyond the sum of each injury. Recovery does not prove that the initiating disease was imaginary, and nonrecovery does not prove a hidden pathogen remains.

Alternative explanations and limits

Irreversible tissue injury, persistent infection, autoimmune disease, organ failure and psychosocial adversity may explain impairment more simply. Calling the condition an attractor does not tell us which mechanism exists, and should not excuse failure to diagnose treatable disease.

What would change the conclusion?

The theory should identify measurable feedback loops and predict the sequence of recovery under successful interventions. It would be weakened if performance changes were fully explained by irreversible structural injury without dynamic regulatory components, or if the proposed loops fail to show meaningful temporal relationships.

The distinctive prediction

A persistent-state hypothesis should forecast linked improvements: an intervention that improves sleep or autonomic function might permit additional functional recovery without erasing the old structural injury. If each outcome changes independently, the proposed shared attractor becomes less informative.

Clinical interpretation

It encourages a useful clinical distinction: preventing death, restoring organ integrity and recovering participation in life require different measures. It does not imply every patient can recover by changing mindset or lifestyle.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. Ann N Y Acad Sci. 1998.
<https://pubmed.ncbi.nlm.nih.gov/9629234/>

Pyrkov TV et al. Longitudinal analysis of blood markers reveals progressive loss of resilience and predicts human lifespan limit. Nat Commun. 2021.
<https://www.nature.com/articles/s41467-021-23014-1>

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VOLUME 7 · PAPER 07 · BIOLOGICAL GHOSTS OF PREVIOUS INJURY

The Wound That Never Received the Message to Stop

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Tissue repair requires as much termination as initiation. A wound might close visibly while persistent fibroblast, macrophage or neural signaling sustains abnormal physiology. The hypothesis is that in some disorders the missing step is not initiation of repair, but successful exit from the repair program.

The biological route

In healing tissue, leukocyte recruitment should give way to resolution, myofibroblast activity should decline, and matrix remodeling should reach an appropriate balance. Stiff extracellular matrix can reinforce YAP/TAZ-dependent fibroblast activation; persistent damage-associated signals may recruit innate immune cells. An initial scar can become an input to ongoing fibrogenesis. This is a physically plausible feedback loop rather than merely a metaphor.

An illustrative scenario

A healed skin wound provides a useful thought experiment: the epithelium has closed, but fibroblast activation may remain abnormal beneath it. If tissue stiffness keeps driving matrix synthesis, then manipulating the original inflammatory trigger may not reverse the new mechanical loop. If the tissue is simply a mature, inactive scar, the mechanism is different. The essay must therefore distinguish ongoing pathology from permanent but biologically quiet structural change.

Alternative explanations and limits

Continued inflammation may reflect a real ongoing insult such as infection, mechanical stress or autoimmunity. Fibrosis can also be an irreversible consequence rather than an active self-sustaining process. A visible scar does not prove pathological signaling is still occurring.

What would change the conclusion?

The model predicts persistent molecular or mechanical activation after the initiating lesion is controlled, and responsiveness to interruption of the feedback circuit rather than repeated removal of triggers. If signaling ends promptly yet dysfunction remains due solely to fixed architecture, the model needs revision.

The distinctive prediction

The most distinctive sign would be pathological fibroblast activation that persists after the primary inflammatory stimulus is controlled but decreases when a specific matrix-feedback pathway is interrupted. An inactive mature scar would not meet this definition.

Clinical interpretation

The concept reframes some recovery failures as termination failures while preserving the need to treat continuing infection, ischemia and other causes of tissue damage. Anti-fibrotic strategies must be disease-specific and clinically validated.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Liu F et al. Mechanosignaling through YAP and TAZ drives fibroblast activation and fibrosis. Am J Physiol Lung Cell Mol Physiol. 2015. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4329470/>

Fibroblast Yap/Taz Signaling in Extracellular Matrix Homeostasis and Tissue Fibrosis. Int J Mol Sci. 2024. <https://pubmed.ncbi.nlm.nih.gov/38929890/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 08 · BIOLOGICAL GHOSTS OF PREVIOUS INJURY

The Phantom Injury Hypothesis

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An injury can disappear on imaging while pain, guarding or threat responses continue. This paper asks whether peripheral sensory nerves, local immune cells and tissue mechanics sometimes preserve a response to an injury that is no longer present. Phantom injury is not a new diagnosis: it is a proposed multi-compartment account of selected persistent symptom patterns.

The biological route

Peripheral nerve injury may alter excitability; spinal and supraspinal networks can undergo sensitization; macrophages, mast cells and glial cells influence nociception; extracellular matrix stiffness can change mechanical input. Together, these interactions may produce a self-reinforcing loop in which expectation, afferent traffic and local biology each sustain the others. Central sensitization alone is not an explanation for every symptom, but it offers one mechanism for persistence.

A deeper interrogation

Imagine a patient whose old injury site becomes painful during stress, without new structural damage detected by standard examination. Several explanations remain possible: undetected peripheral injury, altered sensitivity of peripheral nerves, spinal amplification, changes in expectation, or combinations of these. An intellectually honest model cannot choose its preferred story from symptom persistence alone. The phantom-injury framework becomes useful only when it predicts which perturbation should change the symptom and which should not. It also must make sense of spatial specificity. Why would one shoulder or abdominal quadrant remain sensitized while adjacent tissue behaves normally? Resident immune-cell phenotypes, nerve branching, scar mechanics and cortical representation offer different causal routes. A strong paper should compare these possibilities rather than bundle them under “memory.” One possible discriminating prediction is that a subset of patients has persistent local nociceptive activity that can be observed independently of the central experience of pain. In another subset the dominant driver may be central gain or learned threat response. Neither patient is imagining the symptom. They inhabit different physiological explanations that should not be collapsed into one label.

Alternative explanations and limits

Ongoing occult structural disease, neuropathy, inflammatory arthritis, fracture and neuropathic injury must not be dismissed because imaging appears unremarkable. Imaging sensitivity, test selection and physical examination differ by disorder; absence of a visible lesion does not equal absence of pathology.

What would change the conclusion?

A discriminating account should separate tissue-derived nociceptive input from centrally maintained amplification using repeated objective evaluation and carefully interpreted response patterns. The hypothesis weakens if symptoms consistently map to an active peripheral pathology without independent evidence for persistent feedback.

The distinctive prediction

Spatially restricted symptoms create a stronger test than generalized discomfort: can local tissue measurements explain why one region retains the painful response? If not, circuit-level or systemic explanations may be more powerful.

Clinical interpretation

The clinical value is avoiding a false choice between “all tissue” and “all psychology.” Pain is experienced through a nervous system but can have complex and changing biological inputs. No single imaging result settles causation.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Astrocyte regulation of synaptic signaling in psychiatric disorders. Front Mol Neurosci. 2022.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC9700696/>

Fibroblast Yap/Taz Signaling in Extracellular Matrix Homeostasis and Tissue Fibrosis. Int J Mol Sci. 2024.
<https://pubmed.ncbi.nlm.nih.gov/38929890/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 09 · BIOLOGICAL GHOSTS OF PREVIOUS INJURY

The Body Remembers Where It Was Hurt

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Injury history may produce region-specific susceptibility years later. The proposed mechanism is local cellular memory in resident stromal, epithelial and immune populations, rather than a circulating toxin that affects every site equally. A tissue may respond differently to a second insult because earlier damage changed its epigenetic, vascular or mechanical context.

The biological route

Resident macrophages and fibroblasts can retain altered activation states; damaged basement membranes and persistent extracellular matrix changes can reshape local cytokine gradients. Chromatin remodeling may leave some genes easier to induce on re-exposure. An old injury site therefore represents a distinct ecological niche, even when circulating inflammatory markers are normal.

An illustrative scenario

A recurring patch of inflammation over a previously injured area could indicate biological memory. It could also indicate continued mechanical friction, a damaged local barrier or renewed exposure to the same irritant. The cleanest comparison would involve nearby tissue exposed to the same external conditions. A true site-specific cellular memory should predict a divergent response even after repeated insults and structural differences are accounted for.

Alternative explanations and limits

Local recurrence is also explained by repeated exposure, compromised anatomy, mechanical use, vascular insufficiency or infection in the same site. The theory should not be invoked without asking whether the environment simply continues to supply the original insult.

What would change the conclusion?

Paired sampling of previously injured and uninjured matched tissue under comparable exposures would be more convincing than anecdotal recurrence. One must demonstrate persistent site-specific changes that predict a different response to a new stress. Without predictive value, tissue memory may be just a descriptive label.

The distinctive prediction

Local history should matter even after matched current exposure. The model predicts that previously injured and neighboring uninjured tissue will respond differently to an equivalent new challenge through identifiable resident-cell programs.

Clinical interpretation

This perspective could refine thinking about recurrent dermatitis, fibrosis or regional pain, but remains an organizing hypothesis rather than proof of a universal stored injury program.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Liu F et al. Mechanosignaling through YAP and TAZ drives fibroblast activation and fibrosis. *Am J Physiol Lung Cell Mol Physiol*. 2015. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4329470/>

de Laval B et al. C/EBP β -dependent epigenetic memory induces trained immunity in hematopoietic stem cells. *Cell Stem Cell*. 2020. <https://doi.org/10.1016/j.stem.2020.01.017>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

The Extracellular Matrix as a Biological Hard Drive

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Tissues store a portion of their history outside cells. Collagen architecture, matrix stiffness, bound growth factors and basement-membrane composition persist and alter the behavior of incoming cells. The hard-drive analogy proposes that extracellular structure constitutes a durable local record of injury, capable of influencing future repair.

The biological route

Integrins transmit mechanical force into focal adhesion and cytoskeletal signaling; YAP/TAZ and other transcription factors respond to stiffness. A fibrotic matrix can therefore continue to instruct fibroblasts to synthesize matrix even after the original inflammatory trigger subsides. This yields an important asymmetry: rebuilding the cells alone may be insufficient if the environment instructs them to recreate disease.

A deeper interrogation

The matrix hypothesis becomes genuinely provocative when a cell replacement thought experiment is introduced. Suppose healthy fibroblasts enter a previously fibrotic tissue and begin to express a profibrotic program. Is their behavior caused by an intrinsic defect or by instructions from the environment? A stiff extracellular matrix can influence focal adhesion kinase, Rho-dependent cytoskeletal tension and YAP/TAZ nuclear localization. Binding and release of growth factors further create a biochemical component to the mechanical environment. If the same cell behaves differently when moved into a compliant matrix, the surrounding architecture is acting as a causal input. The implication is not that a scar literally contains sentences written in molecular language. It is that physical relationships can be stable, heritable across cell turnover in a limited sense, and sufficiently structured to constrain later outcomes. An important difficulty is circular causation: fibroblasts shape matrix, and matrix reshapes fibroblasts. Causal direction can be resolved only with perturbations that separate these components. Some chronic disease states might therefore require correcting both damaged cells and the environments that continuously induce damage-associated behavior.

Alternative explanations and limits

Matrix does not store memories as a brain does. Changes may be nonspecific signs of aging, scarring or mechanical use. The metaphor risks implying code-like instructions that have not been decoded or shown to be specific.

What would change the conclusion?

Strong evidence would transfer cells into matrices with different injury histories while controlling soluble factors, and show reproducible differences in fate and functional repair. A refutation would be that matrix differences disappear after controlling for ongoing cellular signals or simple tissue stiffness.

The distinctive prediction

If extracellular architecture carries persistent causal information, transplanting competent cells into an old fibrotic matrix should reproduce some abnormal behavior, while placing damaged cells into a corrected environment might attenuate it. Both directions are important for separating cause and effect.

Clinical interpretation

For clinical reasoning, this offers a mechanism of persistence and an argument for looking beyond blood tests. It is not an assertion that any manual therapy, collagen supplement or unproven matrix intervention repairs fibrotic disease.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Liu F et al. Mechanosignaling through YAP and TAZ drives fibroblast activation and fibrosis. *Am J Physiol Lung Cell Mol Physiol*. 2015. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4329470/>

Fibroblast Yap/Taz Signaling in Extracellular Matrix Homeostasis and Tissue Fibrosis. *Int J Mol Sci*. 2024. <https://pubmed.ncbi.nlm.nih.gov/38929890/>

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VOLUME 7 · PAPER 11 · BIOLOGICAL GHOSTS OF PREVIOUS INJURY

The Immunological Afterimage

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An infection may be microbiologically cleared while its immunological consequences persist. The afterimage hypothesis distinguishes several mechanisms often conflated: retained antigen, persistent tissue injury, altered trained immunity and self-sustaining autoimmune responses. None should be presumed without disease-specific evidence.

The biological route

Innate cells can undergo metabolic and chromatin-associated reprogramming; antigen may be retained or intermittently encountered; adaptive lymphocytes may recognize new epitopes after tissue damage. Altered tissue architecture can recruit immune activity independently of infection. These are distinct pathways with different predictions about organism detection, immune biomarkers and therapeutic response.

An illustrative scenario

Following infection, one patient might have persistent inflammatory laboratory findings while another has dysautonomia without comparable inflammatory changes. The phrase post-infectious illness describes timing, not a single mechanism. The immunological afterimage model would have explanatory value only for the subgroup in which a defined ongoing immune response can be observed and linked to function; it should not be used to erase heterogeneous causes of post-viral symptoms.

Alternative explanations and limits

Ongoing replicating pathogens, recurrent exposure and latent infections can produce similar symptoms. Conversely, fatigue after infection can arise through autonomic, endocrine, sleep, vascular or structural mechanisms that are not primarily immunological. It is easy to call everything “immune memory” without showing a relevant memory substrate.

What would change the conclusion?

The strongest analysis would demonstrate persistence of a defined immune state after clearance of a documented initiating pathogen, and then show that this state predicts a clinically relevant outcome. Mere antibody positivity is insufficient. A model weakened by null immune findings or better evidence of ongoing infection should yield to those alternatives.

The distinctive prediction

The model predicts a measurable immune phenotype with temporal continuity after pathogen elimination. Mere persistence of symptoms is not enough; a defined inflammatory program should help forecast course independently of continuing infection.

Clinical interpretation

Patients deserve evaluation of treatable infections and autoimmune disease, not dismissal as psychologically ill or automatic assignment to chronic infection. The proposed afterimage organizes investigation; it supplies neither a new diagnostic test nor a blanket immunosuppressive treatment.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

de Laval B et al. C/EBP β -dependent epigenetic memory induces trained immunity in hematopoietic stem cells. *Cell Stem Cell*. 2020. <https://doi.org/10.1016/j.stem.2020.01.017>

McEwen BS. Stress, adaptation, and disease: Allostasis and allostatic load. *Ann N Y Acad Sci*. 1998. <https://pubmed.ncbi.nlm.nih.gov/9629234/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

When Rejuvenation Erases Useful Biological Memory

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

If cells acquire maladaptive memory with age or injury, rejuvenation strategies might attempt to erase it. But some durable cellular adaptations protect against pathogens, carcinogenesis and repeated stress. The hypothesis is that indiscriminate resetting can remove beneficial as well as harmful information.

The biological route

Cell identity depends on chromatin organization and transcriptional programs. Innate immune training can enhance responses to certain later challenges, while exhaustion and maladaptive inflammatory training can be harmful. Cellular reprogramming changes epigenetic patterns but may also alter differentiation state and growth control. A purported youthful signature is thus not automatically equivalent to superior organism-level fitness.

An illustrative scenario

Imagine a program that makes a cultured immune cell express a more youthful epigenetic pattern. If the cell also becomes less capable of recognizing previously encountered pathogens, the cosmetic success of the molecular clock would conceal a functional loss. The reverse could occur too. To call the intervention rejuvenating requires a panel of biological functions, not one biomarker. A reset should be judged by what the cell can still do correctly.

Alternative explanations and limits

Some epigenetic changes may be passive correlates rather than memories with functional importance. Other states, such as terminally exhausted immune cells, might benefit from selective remodeling. The answer is likely cell type, duration and context dependent, not a simple ban on cellular resetting.

What would change the conclusion?

Test whether a reprogrammed cell retains appropriate lineage identity, pathogen defense, stress tolerance and tumor-suppressive responses after its molecular age metrics improve. If these functions consistently improve together, the fear of erasing adaptive memory would be weakened for that intervention and tissue.

The distinctive prediction

A reset that removes harmful age-associated programming might also diminish beneficial antigen-specific or lineage-specific responses. Successful rejuvenation should therefore demonstrate preserved function under challenge, not merely increased expression of youthful

markers.

Clinical interpretation

This is a theoretical safety framework, not a recommendation for or against any unapproved reprogramming procedure. It reinforces the distinction between an epigenetic clock moving backward and a person becoming healthier.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

de Laval B et al. C/EBP β -dependent epigenetic memory induces trained immunity in hematopoietic stem cells. *Cell Stem Cell*. 2020. <https://doi.org/10.1016/j.stem.2020.01.017>

Pyrkov TV et al. Longitudinal analysis of blood markers reveals progressive loss of resilience and predicts human lifespan limit. *Nat Commun*. 2021. <https://www.nature.com/articles/s41467-021-23014-1>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 13 · BRAIN AS AN ELECTRICAL ECOSYSTEM

The Ionic Psychiatry Hypothesis

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Could a subgroup of cognitive or affective symptoms arise from disrupted regulation of the extracellular ionic environment rather than from neurotransmitters alone? Neuronal membrane potentials require tightly regulated sodium, potassium, calcium, chloride and proton gradients. Severe electrolyte disease clearly affects mental status; the speculative question concerns subtler and chronic dysregulation within the brain.

The biological route

Astrocytes take up extracellular potassium during neural activity, coordinate water movement and regulate glutamate clearance. ATP-dependent transporters maintain ion gradients, while GABA signaling depends partly on chloride handling. Glial injury, inflammation or impaired perfusion could therefore influence excitability and network stability. The hypothesis asks whether measurable differences in ion buffering might characterize some patients whose standard serum values remain normal.

An illustrative scenario

A patient with fluctuating confusion and seizures may require evaluation for electrolyte disturbances, epilepsy, medication toxicity and encephalitis before anyone proposes a new psychiatric endotype. In contrast, a patient with stable depression and normal neurological findings provides a very weak basis for suspecting disrupted brain ion buffering. The hypothesis is strongest

where observable neural excitability and physiological disturbance occur together.

Alternative explanations and limits

Brain extracellular concentrations are not accurately inferred from ordinary blood electrolyte results. Common depression or psychosis cannot be attributed to hidden ionic imbalance simply because neurons require ions. Genetic channelopathies and epilepsy supply examples of mechanism, not evidence of a widespread nutritional ionic syndrome.

What would change the conclusion?

Discriminating evidence would link a validated measure of altered neural ionic regulation with a reproducible symptom phenotype and a targeted correction that changes function. If associations disappear after controlling for epilepsy, medication use and general brain illness, the wider psychiatric thesis would be substantially weakened.

The distinctive prediction

The proposed psychiatric subtype would require neural or glial ionic dysregulation that is reproducibly linked to symptoms despite ordinary blood electrolyte measurements. If no such neural measure exists, the hypothesis is not yet a diagnosable entity.

Clinical interpretation

The practical implication is a careful neurological and metabolic differential when there are red flags—seizures, confusion, acute fluctuations—not indiscriminate salt or mineral supplementation. Uncontrolled electrolyte treatment can itself produce serious harm.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Astrocyte regulation of synaptic signaling in psychiatric disorders. *Front Mol Neurosci*. 2022.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC9700696/>

Joshi R, Chou S-Y. Beer Potomania: A View on the Dynamic Process of Developing Hyponatremia. *Cureus*. 2018.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC6150768/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 14 · BRAIN AS AN ELECTRICAL ECOSYSTEM

The Brain's Electrical Noise Floor

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Neural circuits require signals to be distinguishable from background activity. This essay proposes that certain disorders may involve reduced reliability of neural signaling because the cellular

environment amplifies spontaneous activity, disrupts inhibitory timing or weakens sensory filtering. “Noise floor” is a technical analogy; it is not an accepted psychiatric diagnostic construct.

The biological route

Astrocytic glutamate transport, potassium buffering, interneuron inhibition, myelination, neuromodulatory gain and neurovascular support can all influence signal fidelity. Changes in one subsystem might increase trial-to-trial variability or promote network instability even without gross neuronal loss. The concrete hypothesis is that combined glial and metabolic stress can shift how strongly a circuit responds to irrelevant versus relevant inputs.

A deeper interrogation

The analogy to telecommunications is useful only if handled carefully. Every biological circuit contains spontaneous activity; in fact, stochasticity contributes to exploration and flexible behavior. A silent brain is not a healthy brain. What matters is the relation between fluctuations and information carried by signals needed for a particular task. An experiment could measure how reliably a sensory stimulus is encoded under varying metabolic or inflammatory states, not merely record that an EEG trace looks noisy. Astrocytes are candidate regulators because they clear synaptic glutamate and buffer ions; their performance influences the temporal and spatial boundaries of excitation. Yet the causal chain from a glial perturbation to depression or hallucination is long and uncertain. A biological change might influence information processing without determining symptom content. This framework should therefore separate cellular signal fidelity, circuit computation, conscious experience and clinical diagnostic categories. A convincing advance would demonstrate a phenotype-specific correspondence among those levels. Without it, “noise” remains a compelling metaphor rather than a psychiatric biomarker.

Alternative explanations and limits

Noise measures differ radically across EEG, neural recordings, behavior and subjective symptoms; some variability is adaptive and essential to learning. Claims about hallucinations or ADHD cannot be inferred from a generic increase in signal variance. Distinct psychiatric syndromes require separate explanatory evidence.

What would change the conclusion?

The proposal should specify a measurable network parameter, a circuit, a patient phenotype and a predicted dose-response relationship. If greater measured neural variability improves information processing, or is unrelated to clinical symptoms, the claim must be narrowed. It also must be compared against established neurotransmitter and network models.

The distinctive prediction

Signal fidelity should predict task-specific perception or cognition better than global labels such as “brain inflammation.” Different circuits may show different noise properties, and some increases in variability may improve rather than impair performance.

Clinical interpretation

This framework is useful for interdisciplinary hypothesis formation, but does not imply that “electrical noise” can be cured by detoxification, arbitrary electrolyte mixtures or commercially marketed brain devices.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Astrocyte regulation of synaptic signaling in psychiatric disorders. *Front Mol Neurosci*. 2022.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC9700696/>

Angleys H et al. The effects of capillary transit time heterogeneity on brain oxygenation. *J Cereb Blood Flow Metab*. 2015.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4420854/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 15 · BRAIN AS AN ELECTRICAL ECOSYSTEM

The Kidney–Brain–Mind Connection

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

The kidney governs more than excretion of creatinine. It helps regulate plasma tonicity, acid-base balance, erythropoietin, mineral metabolism and the circulating concentration of numerous solutes. The speculative frontier is whether modest or early disruptions in renal physiology can shape cognition and mood before overt uremic encephalopathy occurs.

The biological route

Chronic kidney disease is associated with cognitive dysfunction through combinations of vascular injury, anemia, disturbed mineral metabolism, retained solutes, inflammation, sleep disorders and medication exposure. Certain uremic toxins have mechanistic plausibility as neural stressors. In contrast, dietary oxalate is not established as an independent cause of common psychiatric syndromes in people with normal renal function.

An illustrative scenario

A person with declining kidney function and cognitive complaints may also have vascular disease, anemia or sleep apnea. If cognitive performance improves after addressing one of those conditions, that finding matters, but it need not identify an unnamed retained solute. Conversely, a specific metabolite candidate should demonstrate biological access, sufficient concentration and a reproducible effect. The kidney–brain axis is a family of mechanisms, not a single switch.

Alternative explanations and limits

The co-occurrence of kidney abnormalities and depressed mood could reflect shared diabetes, hypertension, socioeconomic stress, chronic pain or illness burden. An association between eGFR and cognition does not identify a specific causal metabolite or show reversibility after its removal.

What would change the conclusion?

A useful conceptual investigation would compare filtration markers, urine measures, albuminuria, vascular biomarkers and time course of cognitive change. A particular solute mechanism needs exposure, dose, biological access to neural tissue and a disease-specific effect—not just statistical association.

The distinctive prediction

A convincing early kidney–brain pathway needs to predict cognition from an identified retained solute, mineral disturbance or vascular mechanism rather than eGFR alone. The strongest candidate should be measurable before overt encephalopathy and remain relevant after common confounders are controlled.

Clinical interpretation

People with acute confusion, seizures, marked renal dysfunction or electrolyte changes require timely clinical evaluation. This hypothesis should encourage detection of defined treatable conditions rather than the marketing of unproven renal-cleansing products.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Joshi R, Chou S-Y. Beer Potomania: A View on the Dynamic Process of Developing Hyponatremia. *Cureus*. 2018. <https://pmc.ncbi.nlm.nih.gov/articles/PMC6150768/>

Angleys H et al. The effects of capillary transit time heterogeneity on brain oxygenation. *J Cereb Blood Flow Metab*. 2015. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4420854/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 16 · BRAIN AS AN ELECTRICAL ECOSYSTEM

The Misidentified Threat: When the Brain Cannot Read the Body

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An organism can experience fear without an external threat when internal bodily signals are interpreted as dangerous. The new synthesis asks whether altered interoceptive precision, autonomic variability and learned predictions can lock a person into recurrent physiological alarm even when the peripheral signal is benign.

The biological route

Baroreceptors, chemoreceptors, vagal afferents and spinal visceral pathways report internal state to the brainstem, insula and related networks. Predictions about body sensations influence what the

person notices and how they respond. Hyperventilation, palpitations or gastrointestinal sensations can be both consequences and triggers of fear, creating a loop between appraisal and autonomic output.

An illustrative scenario

A harmless increase in heart rate can become alarming to someone who interprets it as impending collapse; that fear may increase adrenergic output and magnify the sensation. But a genuine arrhythmia can produce a similar subjective experience. The investigative priority is to determine which physiology is present and whether prediction about the sensation contributes additional amplification. The model must be capable of recognizing both possibilities.

Alternative explanations and limits

Cardiac arrhythmias, thyroid disease, hypoglycemia, pulmonary disease and medication effects may produce genuine dangerous bodily signals. A predictive-processing framework should never be used to psychologize symptoms before appropriate differential diagnosis. Nor does every sensation arise from inaccurate perception.

What would change the conclusion?

One could compare symptom reports with objective physiology across controlled, clinically safe conditions and determine whether perception remains disproportionate after known medical causes are addressed. The model gains force if individual prediction errors prospectively forecast symptom escalation; it weakens if symptoms consistently correspond to identifiable untreated pathology.

The distinctive prediction

The key prediction is not simply that anxious people perceive their bodies differently. It is that a measurable mismatch between physiological state and perceived threat predicts recurrent symptoms and that reducing the mismatch changes both interpretation and function.

Clinical interpretation

The clinical importance is to treat body and interpretation as interacting levels rather than competing explanations. The framework may support interdisciplinary care, but claims of specific cure require disorder-specific outcome evidence.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. *Behav Brain Sci.* 2013. <https://www.cambridge.org/core/journals/behavioral-and-brain-sciences/article/abs/extending-predictive-processing-to-the-body-emotion-as-interoceptive-inference/A53E081B5EEBD7CF7658F3D484714AFE>

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Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

Carbon Dioxide: The Forgotten Regulator of Mental State

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Carbon dioxide is not only respiratory waste; it participates in acid-base physiology, cerebral vascular regulation and chemoreceptor signaling. Respiratory shifts can generate dizziness, tingling, feelings of unreality and panic symptoms. The provocative hypothesis is that respiratory control and sensitivity to carbon dioxide contribute to some recurrent psychiatric presentations more than their diagnostic labels reveal.

The biological route

Hyperventilation lowers arterial carbon dioxide, producing respiratory alkalosis and changes in cerebral blood flow and ionized calcium; chemoreceptor pathways also influence dyspnea and panic perception. The relationship is bidirectional: fear changes breathing, and altered breathing can magnify bodily sensations that increase fear. Carbon dioxide is therefore a plausible mechanistic bridge from behavior to neural physiology.

An illustrative scenario

Two patients who describe panic may have different respiratory patterns. One hyperventilates during attacks; another has no meaningful carbon dioxide change and experiences primarily cognitive fear. Even the first pattern may be consequence rather than cause. Careful measurement during symptoms could distinguish respiratory amplification from other panic mechanisms. It would not justify unsupervised carbon dioxide manipulation or disregard of cardiopulmonary symptoms.

Alternative explanations and limits

Hypoxemia, asthma, pulmonary embolism, seizure, cardiac disease and metabolic acidosis can also drive abnormal respiration. Symptoms during hyperventilation do not demonstrate that chronic anxiety is caused by a carbon dioxide disorder. Respiratory physiology is context-specific, and unsafe respiratory manipulations can create additional risk.

What would change the conclusion?

The question becomes whether repeatable respiratory phenotypes predict symptoms and treatment response better than subjective anxiety severity alone. If end-tidal carbon dioxide measurements do not track symptoms under carefully controlled conditions, this particular mechanistic model becomes less persuasive.

The distinctive prediction

The respiratory mechanism should be observable during symptom episodes and distinguishable from cardiac, pulmonary and metabolic drivers. Carbon dioxide changes that occur only after fear

begins would support amplification more readily than primary causation.

Clinical interpretation

Education should avoid telling patients to suppress breathing or perform risky rebreathing practices. Persistent dyspnea, chest pain, fainting or new neurological symptoms require conventional evaluation.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. Behav Brain Sci. 2013. <https://www.cambridge.org/core/journals/behavioral-and-brain-sciences/article/abs/extending-predictive-processing-to-the-body-emotion-as-interoceptive-inference/A53E081B5EEBD7CF7658F3D484714AFE>

Angleys H et al. The effects of capillary transit time heterogeneity on brain oxygenation. J Cereb Blood Flow Metab. 2015. <https://pmc.ncbi.nlm.nih.gov/articles/PMC4420854/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 18 · BRAIN AS AN ELECTRICAL ECOSYSTEM

Could Neuroinflammation Distort the Sense of Self?

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Depersonalization and dissociation are often discussed psychologically. Here the question is whether changes in inflammatory signaling, sleep and interoceptive network function can sometimes change the quality of embodied self-experience. This is a mechanistic hypothesis, not a claim that inflammatory biomarkers diagnose dissociative illness.

The biological route

Cytokine activity can affect sleep, motivation and neural signaling; astrocytes modulate excitatory synapses; and interoceptive processing contributes to the brain's model of bodily state. A combined disturbance might alter the integration of bodily sensations, memory, emotion and agency. Severe autoimmune encephalitis or delirium demonstrate medical causes of altered self and awareness, but they are not direct models of ordinary depersonalization.

An illustrative scenario

A person reporting depersonalization after sleep deprivation might experience symptom relief with restored sleep without any immune intervention. Another patient with fever, seizures, disorientation and rapidly evolving behavioral change needs medical assessment for encephalitis or other acute conditions. The proposed neuroimmune model should not merge these distinct presentations simply

because both involve altered consciousness or self-experience.

Alternative explanations and limits

Trauma history, panic, migraine, epilepsy, drug effects, sleep deprivation and primary dissociative disorders remain crucial alternatives. Inflammatory markers are nonspecific and may be elevated because of unrelated obesity, infection or stress. The presence of inflammation would not establish direction of causality.

What would change the conclusion?

The hypothesis needs prospective coupling of inflammatory changes with validated dissociation measures and independent neural or interoceptive markers, controlling for sleep and medication exposure. If the symptoms persist without any measurable immune change, the claim should narrow to selected subgroups.

The distinctive prediction

A biologically specific subgroup would require convergent evidence in immune measures, interoceptive processing and dissociative symptoms, not merely elevated C-reactive protein. Strong associations with sleep or trauma alone would favor narrower explanations.

Clinical interpretation

Clinical interpretation should remain respectful of lived experience, avoid simplistic immune treatments, and prioritize urgent assessment for acute confusion, seizures, fever or focal neurological symptoms.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Astrocyte regulation of synaptic signaling in psychiatric disorders. *Front Mol Neurosci*. 2022.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC9700696/>

Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. *Behav Brain Sci*. 2013.
<https://www.cambridge.org/core/journals/behavioral-and-brain-sciences/article/abs/extending-predictive-processing-to-the-body-emotion-as-interoceptive-inference/A53E081B5EEBD7CF7658F3D484714AFE>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 19 · GREAT CLEARANCE PARADOXES

The Redistribution Trap

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

A substance can leave one compartment and become more concentrated in another without leaving the body. This paper challenges claims that mobilization is synonymous with detoxification. The

theoretical question is when changes in adipose mass, protein binding, plasma volume or tissue perfusion alter exposure to bioactive compounds.

The biological route

Persistent lipophilic organic pollutants may accumulate in fat; weight loss can increase selected serum concentrations as tissue stores and distribution volumes change. Biliary and renal elimination, metabolism and tissue partitioning have separate kinetics. The compartment model therefore distinguishes release from storage, chemical conversion and verified excretion. A lower fat mass does not show that a toxic burden is harmless, and a higher blood concentration does not by itself prove clinical injury.

A deeper interrogation

Consider two patients who lose the same amount of fat mass. One has a measurable pre-existing body burden of a known persistent lipophilic compound; the other has negligible exposure. The second may experience no detectable chemical redistribution at all. Even for the first, several quantities must be distinguished: amount remaining in stored tissue, amount entering blood, concentration in circulating lipid fractions, metabolism to other species and total excretion. A rise in serum concentration does not answer how much of the original chemical has left the body. It can reflect release, a smaller distribution volume or changed lipid normalization. Nor does a rise prove symptoms are caused by that substance. The field would benefit from a simple accounting diagram with compartments and rates: input, exchange, transformation and verified exit. Different chemicals would demand different models, because water-soluble solutes, protein-bound drugs, calcium oxalate and persistent organic pollutants cannot be forced into the same “detox” equation. This paper's distinctive contribution is to resist both complacency and alarmism by insisting that biological clearance is a mass-balance question.

Alternative explanations and limits

Observed concentration increases may be partly mathematical consequences of changing lipid pools or specimen normalization. Exposures are chemically heterogeneous; one cannot extrapolate from chlorinated persistent pollutants to all metals, oxalate or ordinary dietary fats.

What would change the conclusion?

A valid analysis would measure absolute body burden when feasible, serum and tissue partitioning, excretion of identified compounds and clinically meaningful outcomes over time. If mass balance shows elimination outpaces release without adverse outcomes, the simple danger narrative loses force.

The distinctive prediction

The decisive observation is net mass balance: does a mobilized compound actually leave the body, remain circulating or move into another store? Confusion among those outcomes is precisely what the redistribution hypothesis is meant to prevent.

Clinical interpretation

The clinical lesson is not to fear medically indicated weight loss, but to resist unjustified claims of cleansing and to distinguish chemical kinetics from symptoms that may have unrelated causes.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Jansen A et al. Increased levels of persistent organic pollutants in serum one year after a great weight loss in humans. *Sci Total Environ.* 2018.

<https://pubmed.ncbi.nlm.nih.gov/29890598/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.

VOLUME 7 · PAPER 20 · GREAT CLEARANCE PARADOXES

The Liver That Looks Normal but Cannot Keep Up

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Routine aminotransferases are mainly markers of hepatocellular injury, not complete readouts of transporter activity, enzymatic flux, biliary excretion or metabolic reserve. This essay asks whether significant functional bottlenecks may exist despite unremarkable enzyme measurements—and when that possibility is clinically established versus conjectural.

The biological route

Bile formation requires canalicular transporters, hepatocyte polarity, energy-dependent transport and coordinated uptake/excretion systems. Drug metabolism involves multiple enzymes whose capacity can change with genotype, inflammation, interactions and liver disease. Albumin, bilirubin, INR and disease-specific tests offer different information from AST and ALT. The proposed systems lens emphasizes flux and throughput, not vague “liver congestion.”

An illustrative scenario

Consider a patient taking a drug cleared by a hepatic transporter who has normal AST and ALT. Drug exposure can still vary because of interactions, transporter genetics or changes in organ function. That is a tractable pharmacological question. It is very different from asserting that the patient has unspecified “liver congestion” because of a headache or food intolerance. Good terrain reasoning must preserve that difference.

Alternative explanations and limits

Normal enzymes with symptoms are common and rarely prove occult hepatic disease. Symptoms attributed to poor detoxification might instead reflect gastrointestinal, endocrine, neurological or psychological conditions. Claims about unidentified circulating toxins without analytical

measurements cannot substantiate the mechanism.

What would change the conclusion?

Discriminating evidence would be objective abnormalities in relevant transport, bile formation or drug metabolism pathways plus clinical response to correction of the demonstrated problem. Where tests and physiology remain normal, the hypothesis should not be used as a catch-all diagnosis.

The distinctive prediction

The claim becomes medically meaningful only when the suspected bottleneck is named: which enzyme, transporter, synthetic function or excretion route is impaired? Vague “congestion” without a measured pathway offers no testable explanation.

Clinical interpretation

The value is refined test interpretation: understand what a measurement assesses and what it does not. It does not justify liver flushing, indiscriminate supplements or stopping effective medication.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Jansen A et al. Increased levels of persistent organic pollutants in serum one year after a great weight loss in humans. Sci Total Environ. 2018.

<https://pubmed.ncbi.nlm.nih.gov/29890598/>

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