The Pre-tensioned Body: A Hypothesis Paper Grounding the Attractor Framework in ECM Mechanics [M] [F] (2026) Robert Galida – June 2026

Abstract

The attractor framework proposes that persistence under perturbation is the fundamental mark of reality—a property it terms *constraint navigation*. This paper proposes a biological grounding for the framework in the physical architecture of the body. From established biomechanical principles, the body is identified as a **pre-tensioned hydrophilic-collagenous composite**—a system where osmotic swelling pressure (from GAGs and proteoglycans) is actively constrained by collagen tensile strength. The difference between the calculated Water Holding Capacity (WHC) of the body's hydrophilic components and its actual water content is proposed as a **candidate surrogate signature** of this pre-tensioned state. Mechanotransduction is identified as a primary intercellular communication channel, and the ECM is shown to be a dissipative attractor that stores mechanical history and shapes cellular behaviour. The paper maps the attractor framework's core variables (κ, B, basin depth) onto measurable physiological quantities **as research hypotheses**: κ is proposed as a latent variable reflecting perturbation-recovery efficiency, estimated from candidate observables such as tissue recoil time, baroreflex sensitivity, and HRV recovery; B is proposed as a function of prestress, repair capacity, and network connectivity, with the WHC discrepancy as one candidate, non-exclusive proxy for its

prestress component; and basin transitions are proposed to correspond to crossing basin-specific thresholds, not a single uniform threshold. A research agenda is provided, including protocols for measuring κ and B non-invasively and testing the WHC-water content discrepancy as a candidate metric of basin depth.

Crucially, this paper does not revise the framework's ontological hierarchy. As established in *Intelligence is the Primitive* (Galida, 2026a), the primitive is **constraint navigation**—the capacity to detect perturbations, update internal states, and maintain persistent trajectories. Mechanotransduction is proposed as the *physical substrate* through which constraint navigation is implemented in biological systems. The nervous system and the ECM are complementary regulatory layers, not competing primitives.

All mappings from physiological variables to framework constructs are proposed as research hypotheses, not established conclusions.

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1. Introduction

The attractor framework defines intelligence as the ability to navigate a constraint field and distinguishes reality attractors (high κ, shallow basin, corrigible) from fantasy attractors (low κ, deep basin, sealed). The framework has been applied to physics, biology, cognition, AI, and social dynamics. However, its physical grounding in the body has remained implicit.

This paper proposes that grounding. It begins with an established biomechanical model of the body's architecture: a **pre-tensioned hydrophilic-collagenous composite**. It then proposes mappings from the framework's core variables onto

measurable physiological quantities, establishes mechanotransduction as a primary intercellular communication channel, and identifies the ECM as a dissipative attractor that stores mechanical history. The paper concludes with a research agenda and testable predictions.

A note on terminology: In the attractor framework's hierarchy, the primitive is *constraint navigation*—a domain-general property of any system that detects perturbations and maintains persistent trajectories. Mechanotransduction is proposed as the *physical substrate* through which constraint navigation is implemented in biological tissues. This paper proposes that substrate; it does not claim that mechanotransduction is a deeper primitive than constraint navigation. For the framework's ontological hierarchy, see Galida (2026a).

A note on scope: All mappings from physiological variables (prestress, mechanotransduction rate, WHC discrepancy) to framework constructs (B, κ, basin depth) are proposed as **research hypotheses**, not established conclusions. The biological claims are grounded in existing literature; the attractor mappings are the novel, untested component of this paper.

A note on the framework's strongest anchor: The framework's most direct empirical anchor is fibrosis, which exhibits classic attractor properties: self-reinforcement, hysteresis, path dependence, resistance to reversal, and threshold behavior. Fibrosis is therefore treated as a central demonstration of the framework's applicability to biological systems.

This paper is primarily a biological hypothesis paper. It proposes specific mappings from physiological variables to attractor-framework constructs. The broader philosophical claims of the attractor framework—about intelligence, consciousness, and reality—are discussed elsewhere (see

Galida, 2026a) and are not the focus of this paper. Where speculative extensions are made, they are clearly flagged.

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2. The Body as a Pre-tensioned System

2.1 The Established Biomechanical Model

We adopt the established biomechanical model of connective tissue as a composite material (Ingber's cellular tensegrity; Donnan osmotic swelling models). In this model:

Component	Role
Hydrophilic components (GAGs, proteoglycans)	Provide **osmotic swelling pressure** — a distributed, expansive force
Collagen	Provides **tensile strength** — the "rebar" that constrains the swelling pressure into a coherent, load-bearing architecture
The body	A **pre-stressed system** — like reinforced concrete, where the rebar (collagen) is under tension and the matrix (GAGs) is under compression

This is not a novel derivation from first principles; it is a reformulation of standard connective-tissue biomechanics in attractor-framework vocabulary.

2.2 The WHC-Water Content Discrepancy

The **calculated Water Holding Capacity (WHC)** of the body's hydrophilic components—the maximum water the tissue could hold if all GAGs and proteoglycans were fully hydrated and

unrestricted—exceeds the **actual water content**. This difference is proposed as a **candidate surrogate signature** of the pre-tensioned state. It represents the water that is being held back by the collagen network—the stored elastic + osmotic energy that defines the attractor basin.

Quantity	Meaning
Calculated WHC	The maximum water the tissue could hold under unrestricted swelling
Actual water content	The water the tissue actually contains
Difference	The water held back by collagen—a candidate surrogate for pre-tension

Operational definition: WHC is estimated via the Donnan equilibrium osmotic pressure:Π=RT∑(Cion,inside−Cion,outside)Π=*RT*∑(*Cion*,*inside*−*Cion*,*outside*)

where Cion*Cion* is determined by the fixed negative charge density of the GAGs. The WHC is the water content predicted under unconstrained free-swelling conditions. The discrepancy with measured water content is therefore a *candidate surrogate* for the mechanical work done by the collagen network to constrain this swelling.

Critical limitation: WHC discrepancy is a **model-derived construct**, not a direct observable. Its validity as a measure of prestress must be confirmed ex vivo by correlating the discrepancy with direct tensile/compressive stress-strain measurements. **We treat it as a candidate surrogate marker for prestress, not as prestress itself.**

WHC discrepancy is one candidate observable among several possible prestress proxies. Other candidates include tissue stiffness (measured by elastography), recoil dynamics (measured by indentation), hydraulic permeability (measured by perfusion), and poroelastic relaxation time (measured by

stress-relaxation tests). We do not claim WHC discrepancy is the preferred or exclusive measure; it is one candidate that warrants investigation.

Importantly, the relationship between WHC discrepancy and prestress is unlikely to be unique. Multiple states—edema, fibrosis, dehydration, inflammation, altered ionic composition, and altered GAG composition—could produce similar WHC-water discrepancies without representing the same prestress state. Prestress may be one contributor to the WHC discrepancy, but the relationship is unlikely to be one-to-one. WHC discrepancy is proposed as a starting point for investigation, not as a definitive measure.

2.3 The Functional Role of Pre-tension

At the scale of a whole organism, slow diffusion is solved by the cardiovascular system (convective bulk flow). However, once oxygen and nutrients leave the capillary bed, they must traverse the interstitial space to reach individual cells. Over distances of micrometers to millimeters, pure diffusion remains rate-limiting. The pre-tensioned ECM contributes to pressure gradients, fluid flow, and mechanical mixing that actively transport solutes through the interstitium. It is one of several contributors, alongside vascular pulsatility, lymphatic drainage, muscle contraction, respiration, and posture.

We propose that prestress is necessary for efficient mechanotransduction, but we do not claim it is the dominant driver of interstitial flow.

Problem	Pre-tensioned Contribution
Diffusion is too slow over tissue-scale distances	The pre-stressed ECM contributes to **pressure gradients**, **fluid flow**, and **mechanical mixing**

Problem	Pre-tensioned Contribution
Nutrients must reach cells deep within tissues	Osmotic pressure generated by GAGs contributes to **interstitial fluid flow**
Waste must be removed efficiently	Mechanical deformation acts as a **pump**, driving convection and mixing
Signalling molecules must propagate rapidly	Mechanotransduction transmits signals faster than diffusion alone

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3. Pre-tension as Stored Constraint History

The connective-tissue matrix carries a record of mechanical loading. Collagen fibers, proteoglycans, and crosslinks retain the geometry and tension that arose during development or past stresses. In effect, a pre-stressed ECM stores **constraint history**: cells continually read and update it. Cells respond to physical stimuli from their microenvironment, including ECM topography, composition, and stiffness (Discher et al., 2005; Engler et al., 2006), and remodel the matrix accordingly. The current structure of the ECM—fiber alignment, crosslink density, hydration patterns—encodes prior mechanical history.

“Constraint history” is more precise than “mechanical memory” because it refers to observable physical properties—fiber alignment, crosslink density, residual strain, anisotropy, and tissue architecture—rather than implying information storage in the cognitive or computational sense.

Hypothesis: Regions of ECM with higher collagen alignment or GAG concentration will correlate with the history of applied stress. Tendons remold to past loading, and scars “remember” tension by oriented fibers.

Experiment: Culture fibroblasts on 3D collagen gels under strain, then release the load and track collagen realignment over days. If the matrix "remembers," the network should remain partly aligned, and fibroblasts on this matrix will show different mechanosignaling (e.g., YAP nuclear localization) compared to naïve gels.

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4. Pre-tension and Free Energy Storage

A pre-tensed ECM is a far-from-equilibrium state that requires energy to maintain. More precisely, it stores free energy in the form of osmotic pressure (from GAGs) and tensile stress (from collagen). Negatively charged GAGs imbibe water and generate osmotic pressure; collagen fibers stretch to resist this swelling, creating tensional prestress. The result is a tension–compression balance that is thermodynamically high in free energy. When pre-tension is lost (e.g., by breaking crosslinks or GAG depletion), the system relaxes to a lower-energy, higher-entropy configuration.

Hypothesis: The water-holding capacity (WHC) gradient creates a free-energy gradient. A large WHC–actual water discrepancy (more bound water than free water) signifies a high osmotic tension and greater free energy storage.

Experiment: Use temperature ramps or chemical perturbations to alter ECM hydration in vitro, and measure work done (e.g., pressure-volume loops). Compare the change in free energy (via heat release or sorption isotherms) as pre-tension is varied.

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5. Thresholds and Phase Transitions in Pre-tension

Biological systems may exhibit a critical tension threshold below which mechanosignaling collapses. In a highly tensioned network, cells easily sense force via stretched fibers; if the network becomes too lax, mechanical signals dissipate before triggering cell responses. There may be a phase-like transition: above a certain pre-tension, the tissue acts as a coherent signal-transmitting medium; below it, the matrix cannot convey stiffness and mechanosensors fall silent.

Basin depth B is a dynamical concept—the energy barrier required to shift a system from one attractor state to another. Prestress is hypothesized to be **one contributor** to basin depth, not a direct measure of basin depth itself. Other contributors include repair capacity, energy availability, network connectivity, and hysteresis. Fibrosis illustrates this distinction: high prestress with low repair capacity yields a deep but pathological basin—a fantasy attractor.

κ is defined as responsiveness to perturbation per unit time—specifically, the inverse of the time (τ) required for a system to return to baseline after a standardized perturbation. In biological terms, κ is operationalized as **perturbation-to-state-update efficiency**. Candidate observables include tissue recoil time, baroreflex sensitivity, HRV recovery, and response latency in mechanosensitive signaling. The framework does not claim that any one of these *is* κ; it claims that they may correlate with κ under controlled conditions.

Hypothesis: There exists a tipping point in ECM tension where YAP/TAZ signaling drops sharply.

Experiment: Gradually digest collagen or GAGs in a tissue sample (using collagenase or hyaluronidase) and monitor

cellular mechanosignaling (e.g., YAP nuclear localization, calcium spikes). Plot signaling versus residual ECM stiffness to identify any sharp transition.

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6. Restoring Lost Pre-tension (ECM Plasticity)

The pre-tensioned state can be partially restored. Tissue remodeling is dynamic: fibroblasts and other cells continually synthesize new ECM and restore tension when stimulated. Exercise and mechanical loading promote this repair. Mechanistically, loading stimulates fibroblasts and chondrocytes to secrete collagen and hyaluronan, re-establishing the collagen–GAG tension balance. Early interventions seem most effective; once fibrosis (irreversible scarring) dominates, recovery is very slow.

Hypothesis: Moderate mechanical stimuli (stretching, cyclic loading) can induce cells to rebuild ECM prestress.

Experiment: In an animal model, apply controlled mechanical loading (e.g., vibration therapy or intermittent stretch) after an induced ECM insult (e.g., partial tendon cut). Monitor ECM markers (collagen I/III ratios, GAG content, tissue preload) over time. Compare to unloaded controls to see how much pre-tension is regained.

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7. The Nervous System as a Mechanosensitive Overlay

Mechanosensitivity is universal in biology. All cells,

including neurons, express mechanosensitive ion channels and attachments. The nervous system is best seen as a specialized extension of the general mechanotransductive framework. It aggregates and rapidly transmits information that is ultimately grounded in physical forces. The body's collagen/tissue network provides a basal "mechanical field," while the nervous system provides a faster, signal-amplified overlay.

Mechanosensitive channels (MSCs) are present in all domains of life—bacteria, archaea, and eukarya—and serve as sensors for touch, hearing, and balance (Martinac, 2004).

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8. Consciousness and Whole-Body Mechanotransduction — Speculative Implications

If mechanotransduction is foundational to biological intelligence, consciousness may not be confined to the brain alone. Embodied cognition theories suggest the sense of self arises from integrated body signals (proprioception, interoception, etc.). The pre-tensioned ECM constantly feeds mechanical inputs (from heartbeat, posture, respiration) into the nervous system. The sense of self—the unified bodily experience—could emerge from the pattern of tension and feedback in the entire body.

Note: This is a speculative extension of the framework, not an established finding. The hypothesis is included to provoke investigation, not to assert a conclusion.

The hypothesis generates specific predictions: altered interoceptive accuracy, altered mechanosensory integration, and altered body-schema stability should correlate with ECM

integrity. These predictions are testable, but the hypothesis itself remains speculative.

Hypothesis: Disorders of depersonalisation or sensorimotor neuropathy may be associated with altered ECM pre-tension and disrupted whole-body mechanotransduction.

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9. Anaesthesia and Mechanical Coherence – Speculative Implications

General anaesthetics profoundly relax muscle tone and reduce vascular tone, collapsing pre-tension throughout the body. This may contribute to loss of consciousness, but the primary mechanism is almost certainly CNS disruption (GABA-A potentiation, thalamocortical disruption). We propose that mechanical coherence may **modulate** conscious state transitions rather than being the principal mechanism.

Note: The mainstream account of anaesthesia attributes loss of consciousness primarily to direct CNS effects. The mechanical effects described here are a speculative, minority-view hypothesis.

Implication: Anaesthesia may not be only neural silencing; it also flattens the body’s mechanical context. This could provide a new perspective on anaesthesia depth and the transition to unconsciousness—but this remains speculative and secondary to the CNS mechanism.

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10. ECM and Neural Plasticity

The brain’s extracellular matrix (ECM) is a key regulator of plasticity. In the adult central nervous system, dense ECM structures (like perineuronal nets) enwrap neurons and stabilize synaptic connections. This stabilization preserves circuitry, but must be relaxed for learning. Neural plasticity is enabled by remodeling that ECM scaffold. Specialised proteases (MMPs) locally degrade ECM to allow synaptic growth. Disrupting ECM often reopens critical periods of plasticity.

The extracellular matrix stabilizes neural circuits while also retaining the ability to be remodeled, to allow synapses to be plastic (Dityatev et al., 2010).

Hypothesis: ECM stiffness, hydration, and organisation directly modulate learning and memory.

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11. ECM in Morphogenesis and Development

During embryonic development, the ECM’s mechanical properties actively guide tissue shaping. Cells use mechanosensation and mechanotransduction at every step of morphogenesis. Gradients of ECM stiffness, fiber orientation, and adhesion create a dynamic “morphogenetic field” of forces. This field adds an instructive layer on top of chemical morphogens.

The ability of a cell to sense and transduce mechanical signals is fundamental to biophysically guiding tissue morphogenesis (Mammoto et al., 2013).

The old idea of a morphogenetic field can be reinterpreted as the physical field of stress and strain in the ECM.

12. Reprogramming the ECM

Because the ECM retains mechanical history, it can also be re-programmed by new inputs. Chronic mechanical stimulation—like exercise, therapeutic stretching, or localized vibration—has been shown to remodel collagen networks and GAG content. The extent of reversibility likely diminishes with age and chronic pathology, but in principle the ECM can be "trained" to a more functional state.

Experiment: Compare young vs old animals subjected to identical mechanical therapy, measuring ECM markers (collagen crosslinking, HA content) before and after. Check if plasticity ("responsiveness") declines with age or disease.

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13. Evolutionary Origins: Ancient Mechanotransduction

Mechanotransduction is evolutionarily ancient. Mechanosensitive channels and adhesion complexes exist in bacteria, plants, fungi and all animals. Even simple multicellular organisms coordinate behaviour via tension. The nervous system likely evolved by layering fast electrical signaling on this existing mechanosensory scaffold.

Implication: Mechanical communication predated nervous networks. The nervous system is a specialised overlay on a more primitive, more global system.

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14. Fibrosis as a Fantasy Attractor

In fibrosis, the ECM enters a self-reinforcing rigid state. Activated fibroblasts lay down excess collagen and crosslinks. The stiff matrix further activates profibrotic signals, locking the tissue into a pathological attractor. Normal mechanotransduction amplifies the fibrotic feedback. Treating fibrosis is notoriously hard, consistent with escaping a deep attractor.

Fibrosis is a classic attractor phenomenon: self-reinforcement, hysteresis, path dependence, and resistance to reversal. It demonstrates the core dynamical properties of a fantasy attractor more directly than many of the consciousness sections. It is therefore treated as a central demonstration of the framework's applicability to biological systems.

Hypothesis: Fibrosis can be modelled as a dynamic system with a parameter (stiffness) that, when large, flips cell behavior to a new attractor.

Experiment: In vitro 3D cultures where stiffness is slowly increased and cell markers monitored.

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15. Cancer and ECM Degradation

Tumours often destroy or disorganise the ECM. Cancer cells secrete proteases (MMPs) that digest collagen and proteoglycans, releasing embedded growth factors. This degraded, low-tension environment may let cells escape normal constraints. ECM breakdown can free tumour cells from their normal niche attractors, allowing invasion and metastasis.

Implication: Normal ECM architecture constrains cellular behavior and tissue organization; disruption of those

constraints is frequently associated with tumor progression.

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16. Ageing as ECM Failure

Ageing appears as a gradual failure of ECM maintenance. Collagen becomes glycated and cross-linked, stiffening tissues but reducing dynamic range. GAG and proteoglycan levels decline, reducing water content and osmotic pre-tension. The net effect is loss of the coherent tension network. Cells in old ECM lose coherent mechanosignals, and stem cells in fibrotic niches lose potency.

Evidence: Ageing of the intervertebral disc is associated with a decrease in its hydration, which increases the compressive stiffness of the matrix (Maroudas et al., 1975). Similar water-content changes occur in articular cartilage with osteoarthritic degeneration (Mankin & Thrasher, 1975).

ECM deterioration may be one important contributor to systemic ageing, alongside genomic instability, mitochondrial dysfunction, epigenetic drift, stem-cell exhaustion, and immune dysregulation. The ECM is not the sole cause of ageing; it is one layer in a multi-factor process.

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17. The Heartbeat as a Global Periodic Perturbation

The cardiac pulse is a globally distributed periodic perturbation. Every cell experiences some aspect of it. The interesting question is whether biological regulation exploits the pulse as a synchronization carrier, rather than whether it is a “master signal.”

Hypothesis: The heartbeat entrains peripheral tissues.

Experiment: Compare mechanosensitive gene expression in pulsatile (arterial) vs non-pulsatile (venous or lymphatic) vessels under otherwise similar pressures.

Implication: The heartbeat is a global mechanical signal that all cells can feel—but we do not claim it is a "master" signal in any hierarchical sense.

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18. HRV and ECM Integrity

Healthy hearts display variability (HRV) that reflects adaptability. High HRV means the system can flexibly modulate pressure waves—effectively a more adaptable global mechanical coherence. Low HRV (as in ageing or disease) might mean a rigid, less coherent pulse.

Critical distinction: HRV is one possible observable among many, not the privileged readout of κ. Other candidate observables include tissue recoil time, baroreflex sensitivity, and skin turgor recovery. The framework's claim is not that HRV *is* κ, but that HRV may correlate with κ under controlled conditions. This is a hypothesis, not an established fact.

κ is not a single molecular mechanism. Mechanotransduction includes ion-channel gating (ms), calcium waves (seconds), YAP translocation (minutes), transcriptional remodeling (hours), and ECM remodeling (days). κ is proposed as a **latent variable**—a system-level correction coefficient estimated from recovery trajectories after a standardized perturbation—rather than directly identified with any single physiological process. Candidate observables for κ include tissue recoil time, baroreflex sensitivity, HRV recovery, and skin turgor

recovery. The framework does not claim that any one of these *is* κ; it claims that they may correlate with κ under controlled conditions.

Whole-body coherence requires both: signal quality (e.g., HRV) and signal transmission (healthy ECM).

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19. The Nervous System and the ECM as Complementary Regulatory Layers

The nervous system is often thought of as the body's primary communication and control network. This is true for rapid, point-to-point signaling. However, it is not the whole story.

Mechanotransduction is evolutionarily and developmentally prior to the nervous system—it appears in all cells, including bacteria and plants, and preceded the evolution of neural tissue by billions of years. However, it is not "the primitive" in the framework's ontological hierarchy. The primitive, as established in *Intelligence is the Primitive* (Galida, 2026a), is **constraint navigation**: the capacity of a system to detect perturbations, update its internal state, and maintain persistent trajectories.

Mechanotransduction is proposed as the **physical substrate** through which constraint navigation is implemented in biological systems at the tissue level. It is the mechanism by which cells sense and respond to mechanical forces—forces that are then integrated into the body's broader navigational repertoire.

This distinction is important for two reasons:

1. **It preserves the framework's domain-generality.** Constraint navigation applies to physical

systems (thermostats, electrons), biological systems (cells, organisms), cognitive systems (beliefs, learning), and artificial systems (LLMs, robots). Mechanotransduction applies only to biological systems.

2. **It clarifies the hierarchy.** The hierarchy is established in Galida (2026a) and reproduced here for reference:

Level	Description
Primitive	Constraint navigation — the capacity to detect perturbations, update internal states, and maintain persistent trajectories
Biological intelligence	Constraint navigation implemented in living systems
Cognitive intelligence	Constraint navigation involving representations
Reflective intelligence	Constraint navigation involving self-models
Linguistic intelligence	Constraint navigation involving symbols

In this hierarchy, mechanotransduction is proposed as the substrate of *biological intelligence*—not a separate, deeper primitive.

What does this mean for the body as a communication network?

The nervous system is a point-to-point system; it does not reach every cell. Neural conduction is fast (up to ~120 m/s), but mechanical wave propagation through a pre-tensioned, hydrated ECM is globally distributed. Mechanotransduction—present in every cell—provides a **complementary regulatory layer**: slower than the nervous system for point-to-point signaling, but more global and persistent. The ECM is best understood as a **constraint field and regulatory context** rather than a communication network in

the neural sense.

This does not mean the nervous system is “too sparse and too slow” in any absolute sense. It means that mechanotransduction and neural signaling are **complementary regulatory layers**, each solving different problems:

Layer	Speed	Reach	Function
Mechanotransduction	Slow (ms to hours)	Global (all cells)	Distributed mechanical history, homeostasis
Nervous system	Fast (ms)	Point-to-point	Rapid coordination, conscious regulation

The heart’s pulse is a global mechanical signal that every cell can feel. The nervous system is the fast, flexible overlay that can modulate this global signal. Whole-body coherence requires both: a healthy ECM (signal transmission) and a responsive nervous system (signal modulation).

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20. Imaging and Measuring the Pre-tensioned State

Noninvasive imaging of ECM tension and hydration is an active frontier. Magnetic resonance elastography (MRE) and ultrasound elastography can map tissue stiffness. MRI can measure water content and molecular environment via T1ρ and T2 mapping. Bioimpedance analysis (BIA) offers a simpler approach to gauge whole-body fluid compartments.

It is possible to detect changes in collagen, proteoglycan and water content—parameters that are associated with early

degradative changes in cartilage (reviewed in cartilage imaging literature).

Proposal: Combine modalities to estimate the WHC–water discrepancy. Over time, create whole-body "tension maps."

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21. Whole-Body Coherence and Measurement

Whole-body mechanical coherence might be measured by coupling between physiological rhythms. Record heart pulse waveforms at two distant sites and compute their synchronisation. Alternatively, measure the delay between the ECG R-wave and a mechanosensitive event (like a muscle stretch reflex) under varying postures.

Proposed metric: Develop a "mechanical coherence index" by measuring how simultaneously tissues stretch or respond to a controlled perturbation.

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22. WHC-Water Content Discrepancy as a Candidate Biomarker

The difference between a tissue's water-holding capacity (WHC) and its actual water content is proposed as a candidate health index. A large discrepancy may indicate lost tension and slack matrix.

Evidence: Ageing of the intervertebral disc is associated with a decrease in its hydration, which increases the compressive stiffness of the matrix (Maroudas et al., 1975). Similar water-content changes occur in articular cartilage with

osteoarthritic degeneration (Mankin & Thrasher, 1975).

Experiment: In a longitudinal cohort, use MRI or ultrasound to estimate WHC (by T1ρ for GAG) and actual water (by T2 or bioimpedance) in joints or muscles. Relate the WHC-water gap to measures like mobility, bone density, or metabolic health.

Prediction: The gap will widen with age and in connective tissue diseases (e.g. osteoarthritis, fibrosis), paralleling functional decline.

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23. Conclusion

The body is a pre-tensioned hydrophilic-collagenous composite. The WHC-water content discrepancy is proposed as a candidate surrogate signature of this pre-tensioned state. Pre-tension is not merely structural; it contributes to transport, mechanotransduction, and tissue organization at biologically relevant scales. Mechanotransduction is a primary intercellular communication channel, and the ECM is a dissipative attractor that stores mechanical history.

However, mechanotransduction is not "the primitive" in the attractor framework's ontological hierarchy. As established in *Intelligence is the Primitive* (Galida, 2026a), the primitive is **constraint navigation**—the capacity to detect perturbations, update internal states, and maintain persistent trajectories. Mechanotransduction is proposed as the *physical substrate* through which constraint navigation is implemented in biological systems.

The attractor framework's core variables (κ, B, basin depth) are **proposed to be** grounded in this substrate: κ is proposed as a latent variable reflecting perturbation-recovery efficiency, estimated from candidate observables such as

tissue recoil time, baroreflex sensitivity, and HRV recovery; B is proposed as a function of prestress, repair capacity, and network connectivity, with the WHC discrepancy as one candidate, non-exclusive proxy for its prestress component; and basin transitions are proposed to correspond to crossing basin-specific thresholds, not a single uniform threshold. These mappings require empirical validation through the measurement protocols outlined in the research agenda.

The strongest version of this paper's claim is not that ECM explains consciousness, aging, cancer, or intelligence. It is that the ECM is a **neglected dynamical layer** that may couple mechanics, signaling, adaptation, and long-term tissue memory. That claim is already significant and does not require overextension.

The nervous system and the ECM are complementary regulatory layers: the nervous system provides fast, point-to-point control; the ECM provides slow, globally distributed mechanical history and coherence. The ECM is best understood as a constraint field and regulatory context rather than a communication network in the neural sense.

Consciousness, in the framework's hierarchy, is a **second-order regulator** of intelligence—not of mechanotransduction directly. It can enhance or block biological intelligence (including mechanotransduction) via attention, stress, and intentional practice, but it operates *through* the same constraint-navigation architecture that governs all intelligence.

The biological program outlined here may occupy decades of empirical work. Extension to social and AI systems is speculative and outside the scope of this paper. We discuss these extensions elsewhere (see *Religions as Attractor Landscapes*, *Flatland to Reality*) but do not claim they are validated by the biological evidence presented here.

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