Where Aging Comes From

The Human as a Fixation in the Vacuum Net

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1. Three Reversals from One Cohort

Start with data.

In 1986 the city of Leiden enrolled almost every inhabitant aged 85 and over. The cohort was followed for a decade. Three findings came out of it. Each runs against the direction the textbooks give.

Thyroid. Gussekloo and colleagues found that a higher TSH and a lower free T4 went with *better* survival. The gland that looks like it is failing belongs to the people who live longest. Atzmon found the complement in centenarians: extreme longevity comes with a raised TSH.

Cholesterol. Weverling-Rijnsburger found that each rise of 1 mmol/L in total cholesterol corresponded to a 15 percent *lower* mortality. The excess deaths at the low end were from cancer and infection.

Blood pressure. Van Bemmel found no association at all between high blood pressure and mortality after age 85.

One nuance sharpens this rather than softening it. In nonagenarians, a higher fT3/fT4 ratio goes with lower mortality. So it is not that less is better. What tracks survival is the conversion capacity — a *relation* between two quantities. Not a level.

2. The Constant That Is Not Constant

Three markers, one cohort, three wrong readings. The error is not in the measurement.

A laboratory reference interval is a constant. It is derived from a healthy young population and then applied as though it held everywhere. That is the assumption doing the damage: that the value which is normal in one context is normal in all contexts.

The framework used here denies exactly that. Its fourth axiom — the context principle, $L(s) = \Phi(T|s)$ — states that what look like constants are state functions of the local medium. They are elastic moduli of a context, not universal numbers. This is the point where the framework departs from the alternatives, which all keep their constants universal.

Applied to a biological register, A4 makes a plain prediction. A regulated system in a changed context will settle at a changed set point, and that new value is not a defect. It is the setting the system chose under the load it now carries.

The coherence-medicine series already names this failure mode. Type III is reference drift: the system is coherent, but the reference against which it is judged has moved. The three Leiden reversals are Type III, measured in a whole city.

So the task of this essay is to separate three things which the word "aging" currently runs together. What genuinely wears. What merely stiffens. And what only appears to fail because it is being read against a constant that no longer applies.

3. The Net, and What a Human Is in It

Picture the vacuum as a net of knots. The condensate description is the collective, effective account of that network. Matter is not placed in the net; matter is frozen topology *of* the net.

A human being, in this reading, is not a body at coordinates. It is a fixation: an asymmetric enclosure that holds a basin on the vacuum manifold. The personal blueprint is N0 plus the local state, and the local state enters through A4. Dying is the fixation letting go. The address remains.

That single formula sets up the whole argument.

If the blueprint is N0 plus a local A4 term, then aging is a change in the A4 term. N0 does not age. The context does, and with the context the constants do.

This is not a picture imported from elsewhere. Ingber showed that cells and tissues are tensegrity structures: continuous tension stabilised by discontinuous compression, with force running from the extracellular matrix through integrins into the nucleus without a break. Cell shape, gene expression and cell fate are functions of the tension state. The body is measurably one continuum under prestress, which is what the net picture requires.

4. Three Degrees of Freedom

The dynamic net has three degrees of freedom: weather, tension, and fixation.

They read straight into biology.

Fixation is the knot that is held — cells, organelles, collagen fibrils, organs. Topologically it carries a conserved charge Q. This is what earlier clinical work called the *form* register.

Tension is the state of the medium — χ, the local stiffness of the substrate through which everything couples. Extracellular matrix, fascia, structured water, the conducting connective tissue. This is the *medium* register.

Weather is what passes through the net — the rhythms, the coupled oscillators, the traffic. Heart, breath, hormonal pulsing, cortical rhythm. This is the *rhythm* register.

Aging does something different to each. That is the whole argument, and it is why "aging" as a single word misleads.

5. Tension: the Medium Hardens

The thread hardens first, and here the damage reading is correct.

Advanced glycation end-products cross-link collagen. Elastin fragments. Fibroblasts become myofibroblasts and lay down more matrix. Tissue modulus rises measurably with chronological age.

It is not passive. Selman and Pardo describe fibroageing as a self-feeding loop. Stiffer matrix activates fibroblasts through mechanosensitive channels. Activated fibroblasts deposit more collagen. Stiffness promotes cellular senescence, and the senescent secretome drives further remodelling. Lampi and Reinhart-King showed that stiffness is an early driver of disease, not a late consequence.

In the framework this is a change in χ, the tension of the local medium. And the constitutive relation says that interaction strength is a composed function of context: $g = g(\alpha(\chi))$. Change the medium and you change the coupling — not because any knot was damaged, but because the substrate through which knots address each other has different properties.

That gives the biological observation a general form. Nothing is missing. The signal is unchanged. The transmission is not.

There is a second point here worth stating, because it turns "slack" from an image into a claim. The knot-network account gives the leading nonlinearity as topological, of order 2π, which forces the quadratic coefficient negative and puts the medium near a large cancellation — roughly 84 percent at $\gamma = 2\pi$. A medium in that condition sits close to criticality. It is poised: small inputs can reorganise it, and it can absorb without breaking.

Health is a medium that has kept its cancellation. Loss of slack is the ground state moving away from criticality. Not a net that has been pulled tight by an outside hand, but a medium whose own constitutive state has drifted.

6. Fixation: the Form Wears

The knots wear, and this too is real.

Nodules accumulate. Over half of people past sixty have thyroid nodules on ultrasound. Clonal drift proceeds. Autopsy series find papillary microcarcinomas in a large share of people who died of something else. Mutations pile up. Stem-cell pools deplete.

Here the classical account is right. Local errors accumulate in a bounded structure and are not repaired.

The framework adds a criterion rather than an image. Topological charge is conserved. A knot is not degraded continuously; it holds until it is untied, and then it is gone. So the register divides cleanly:

Changes in χ and in coupling are, in principle, recoverable. The knots are intact and the medium can be brought back toward its poised state.

Changes in Q are not. Destroyed follicles, lost neurons, an excised gland — the address no longer has a knot to hold it.

That criterion answers a practical question that is usually settled by intuition. Replacement therapy is right where Q has changed and something is genuinely absent. It is doubtful where only the A4 term has moved and the source is intact. The difference is not a matter of philosophy. It is visible in whether the tissue is still there.

7. Weather: the Rhythm Comes Apart

Here the standard reading breaks, and there is a measured quantity to put in its place.

Keenan and Veldhuis studied luteinising hormone in healthy men. Older men produced *more* pulses per day than younger men — about twenty-three against fifteen. The mass per pulse was smaller. Total secretion and elimination kinetics were the same.

Read that again. Same output. Broken structure. A level-based assay sees nothing at all.

The pattern generalises. Across the somatotropic, gonadotropic, corticotropic and insulin axes, the orderliness of release declines progressively with age. Insulin loses its regularity under a steady glucose stimulus.

Then the decisive measurement. Veldhuis applied cross-approximate entropy to *pairs* of coupled hormones — ACTH with cortisol, LH with testosterone. Their conditional synchrony falls with age. That is coupling loss, quantified, between two measured series.

This is not a new quantity. It is an existing estimator of something the framework already defines. The coherence functional measures how concentrated a register's state is, and how far it sits from its own set point. Cross-entropy between two registers is that functional evaluated across a pair. Endocrinology has been measuring the coherence functional since the 1990s without calling it that.

Lipsitz and Goldberger had proposed the general shape in 1992: aging as loss of physiologic complexity, with reduced capacity to adapt to stress. They already named heart-rate control, pulsatile hormone release and the EEG together.

So the weather register does not decline in amount. It loses organisation. The net keeps its threads and loses the ability to pull on several at once.

8. Reading the Signal Correctly

One thing must be got right, or this collapses into slogan.

Two literatures predict opposite things. Critical slowing down predicts rising variance and rising autocorrelation before a transition. The complexity account predicts falling variability. Heart-rate variability does fall with age. Hormonal irregularity rises.

The data do not simply pick a side. Gijzel and colleagues measured postural sway in high-functioning older adults, comparing walkers of the Nijmegen Four Days Marches with non-walkers. The walkers showed *lower* variance and *lower* autocorrelation, and lower variance went with successful aging at baseline and a year later. Rector and colleagues then applied both paradigms to geriatric inpatients and found the associations did not all run in the expected direction. Their conclusion: the assumptions of a paradigm must be matched to the homeostatic role of the variable being watched.

Vaillancourt and Newell had made the general point already. Complexity can rise or fall with age, depending on the intrinsic dynamics of the system and the task demand.

The resolution is not to choose a side. It is to stop treating variance as the quantity of interest.

Amount of fluctuation is not the measure. Structure of fluctuation is. A hormone axis losing orderliness and a heart losing variability are the same event seen from two directions. What disappears in both is the organised relation between parts. That is what the coherence functional is defined on, and what cross-entropy estimates. Raw variance does not estimate it at all.

This is also why the thyroid reads as it does in section 1. A raised TSH is a level, and A4 has already told us that levels are context functions. The fT3/fT4 ratio says something, because it is a relation between two registers rather than a number in one.

9. Mending

The therapeutic order follows from the ontology, and it is the same three-step cycle used elsewhere in this work for writing form into a medium.

Slacken. Take the standing load off, so the medium can reorganise. In the design language this is context normalisation: an address becomes transferable only after the local state has been brought back toward a neutral condition. Clinically: sleep, resolution of chronic inflammation, mechanical unloading, gentle mobilisation, periodic reduction of metabolic load. A taut net cannot be re-tied. It must be given slack first.

Re-tie. While the medium is pliable, present the healthier configuration. Progressive loading that restores fibre alignment. Movement that reopens redundant paths instead of concentrating function into a few overworked hubs. This is where the new arrangement is written, not where it is fixed.

Re-tension. Let the configuration set under controlled load, so it becomes the stable state rather than a passing fluctuation. Training. Progressive resistance. Restored redundancy — several thin paths rather than one thick one.

The order matters. Hardening a net that was never slackened only deepens the pattern already there.

There is direct evidence that the third step reaches the weather register. Endurance training raises heart-rate variability well past sixty, in a linear frequency-response relationship: more often, more gain. The largest change appears in nocturnal heart rate. Multicomponent training has been tested for restoring cardiac complexity in prefrail older adults.

That is the practical statement of the whole essay. The level may fall. The coupling is what to defend.

10. What Would Settle It

Four claims here are load-bearing. Each is checkable.

One. Coupling loss predicts decline better than level shift does. Cross-approximate entropy between paired axes is computable from endocrine datasets that already exist. What is missing is a study relating it to survival rather than to age alone. That single result would move this from reading to finding.

Two. Mending restores coupling and not only level. The exercise literature reports heart-rate variability. It should report entropy and cross-entropy on the same recordings.

Three — the sharpest. Reversibility follows the conserved charge. Where imaging or histology shows the structure intact, coherence measures should recover under the slacken–re-tie–re-tension cycle. Where the structure is gone, they should not, at any dose. This predicts *which* interventions can work in *which* patients, and it is wrong if recovery turns out to be independent of whether the tissue is still there.

Four. If tension is a genuine constitutive variable and not a metaphor, then stiffness measured directly — elastography, atomic-force microscopy on biopsy — should predict coupling loss in other registers before clinical disease appears. One medium, several registers, one shared signature. The damage account expects independent clocks per tissue. That difference is decidable.

A fifth question stays open. Pridham, Rockwood and Rutenberg modelled frailty in two large cohorts and found a tipping point near age seventy-five, where damage and repair rates cross. Their mechanism is a damage–repair balance, not a constitutive one. Whether the same transition shows up in coherence measures, at the same age, is unknown. If it does, the two accounts are describing one event from two sides.

The net does not empty as it ages. It goes slack where it should be taut, taut where it should be slack, and stops pulling together. Mending is bringing tension back where it belongs. Not adding thread.

Annotated References

The framework

J. Konstapel, "Fractal Context Dynamics: A Context-Dependent Framework for the Emergence of Physical Law" (Leiden, 2026). Source of the context principle A4, $L(s) = \Phi(T|s)$: what look like constants are state functions of the local vacuum. This is the axiom that makes a drifting reference interval expected rather than anomalous. Presented as a research programme, not settled physics.

J. Konstapel, "The Constitutive Equation of the Vacuum Condensate" (Leiden, 2026). Derives the composed form $g = g(\alpha(\chi))$ — context sets α, α sets the interaction. The technical basis for section 5, and for treating matrix stiffening as a change in a constitutive variable rather than as accumulated debris.

J. Konstapel, "How to Visualize the Vacuum" (Leiden, 30 July 2026). The seven images used throughout: fish, net, knot alphabet, taut and slack, weather in the net, the loaded spring, the mending of the net. Also the source of the $\gamma \approx 2\pi$ and ~84 percent cancellation reading that puts the medium near criticality.

J. Konstapel, "The Knot in the Blueprint" (Leiden, 31 July 2026). Defines the human as a fixation in the net — asymmetric enclosure, blueprint as N0 plus local state through A4, dying as the fixation releasing while the address remains. Section 3 is the aging case of that construction. Also the source of the slacken–re-tie–re-tension cycle used in section 9.

J. Konstapel, Coherence Medicine series, papers 1–4 (constable.blog and ResearchGate, 20 July 2026). Defines the coherence state C(t) from synchronisation, connectivity, information flow and adaptivity, and gives five failure types. Type III is reference drift, which is what sections 1 and 2 are about. Sections 7 and 8 use the same functional; cross-approximate entropy is an existing estimator of it.

The reversals

J. Gussekloo, E. van Exel, A. J. M. de Craen, et al., "Thyroid status, disability and cognitive function, and survival in old age," *JAMA* 292 (2004): 2591–2599. Prospective follow-up within the Leiden 85-plus Study, covering 87 percent of a two-year birth cohort. Raised TSH and lowered free T4 in the oldest old accompany better survival. The paper questions whether subclinical thyroid dysfunction should be treated at this age at all.

G. Atzmon, N. Barzilai, J. G. Hollowell, M. I. Surks, I. Gabriely, "Extreme longevity is associated with increased serum thyrotropin," *J Clin Endocrinol Metab* 94 (2009): 1251–1254. The same direction in a different population. Raised TSH as a feature of the very long-lived, not a defect survived in spite of.

A. W. E. Weverling-Rijnsburger, G. J. Blauw, A. M. Lagaay, et al., "Total cholesterol and risk of mortality in the oldest old," *The Lancet* 350 (1997): 1119–1123. 724 participants at median age 89. Each 1 mmol/L rise in total cholesterol corresponded to a 15 percent fall in mortality, driven by lower death from cancer and infection.

T. van Bemmel, J. Gussekloo, R. G. J. Westendorp, G. J. Blauw, "In a population-based prospective study, no association between high blood pressure and mortality after age 85 years," *J Hypertens* 24 (2006): 287–292. The plainest of the three. The association simply disappears.

"Thyroid status and mortality in nonagenarians from long-lived families and the general population," *Aging* (2017). The corrective to a lazy reading of the above. Higher free T3 and a higher fT3/fT4 ratio go with lower mortality. What tracks survival is a relation, not a level.

Coupling loss as a measured quantity

D. M. Keenan, J. D. Veldhuis, "Disruption of the hypothalamic luteinizing hormone pulsing mechanism in aging men," *Am J Physiol Regul Integr Comp Physiol* 281 (2001): R1917. The sharpest single result here. Roughly 23 LH secretory bursts per 24 hours in older men against 15 in the young, lower mean pulse mass, comparable total secretion. Same amount, degraded structure.

J. D. Veldhuis, "Nature of altered pulsatile hormone release and neuroendocrine network signalling in human ageing," *Novartis Found Symp* 227 (2000): 163–185; and "Altered pulsatile and coordinate secretion of pituitary hormones in aging: evidence of feedback disruption," *Aging (Milano)* 9 (1997). Orderliness of GH, insulin and LH release falls progressively with age. More important: cross-approximate entropy rises between coupled axes such as ACTH–cortisol and LH–testosterone. Coupling loss, measured, between two series.

G. S. Meneilly, J. D. Veldhuis, D. Elahi, "Disruption of the pulsatile and entropic modes of insulin release during an unvarying glucose stimulus in elderly individuals," *J Clin Endocrinol Metab* 84 (1999): 1938–1943. The same signature in a second axis with the input held flat. The ordering is lost even when nothing outside is varying.

L. A. Lipsitz, A. L. Goldberger, "Loss of 'complexity' and aging," *JAMA* 267 (1992): 1806–1809. The founding statement: aging as generalised loss of complex variability across cardiovascular control, pulsatile hormone release and EEG, with impaired adaptation as the consequence.

D. E. Vaillancourt, K. M. Newell, "Changing complexity in human behavior and physiology through aging and disease," *Neurobiol Aging* 23 (2002): 1–11. The necessary correction.

Complexity can rise or fall; direction depends on the system and the task. This is why section 8 abandons variance as the quantity of interest.

The medium

D. E. Ingber, "Tensegrity I. Cell structure and hierarchical systems biology," *J Cell Sci* 116 (2003). Continuous tension, discontinuous compression, force transmitted from matrix through integrins to nucleus, with shape and gene expression as functions of the tension state. The measured substrate for treating the body as one continuum.

M. Selman, A. Pardo, "Fibroageing: an ageing pathological feature driven by dysregulated extracellular matrix," *Ageing Research Reviews* (2021). Tissue stiffening as a general feature of chronological aging, with the self-amplifying loop between stiffened matrix and profibrotic programmes.

M. C. Lampi, C. A. Reinhart-King, "Targeting extracellular matrix stiffness to attenuate disease," *Science Translational Medicine* (2018). Stiffness as an early driver rather than a late consequence. Supports treating the medium as a primary variable.

Dynamics and the variance problem

M. Scheffer, J. Bascompte, W. A. Brock, et al., "Early-warning signals for critical transitions," *Nature* 461 (2009): 53–59. The generic signature proposed for systems near a tipping point. Included because the aging data do not straightforwardly show it, which is the point of section 8.

S. M. W. Gijzel, I. A. van de Leemput, M. Scheffer, et al., "Dynamical indicators of resilience in postural balance time series are related to successful aging in high-functioning older adults," *J Gerontol A* 74 (2019): 1119–1126. 240 older adults, mean age 84, including 94 walkers of the Nijmegen Four Days Marches. The walkers showed lower mediolateral variance and lower autocorrelation, and lower variance was independently associated with successful aging. Opposite to the naive early-warning prediction, and cited for that reason.

J. L. Rector, S. M. W. Gijzel, I. A. van de Leemput, et al., "Dynamical indicators of resilience from physiological time series in geriatric inpatients: lessons learned," *Experimental Gerontology* 149 (2021): 111341. Both paradigms applied to the same inpatient signals. The associations did not all run in the expected direction. The authors conclude that a paradigm's assumptions must match the homeostatic role of the monitored variable. A caution, not a confirmation.

G. Pridham, K. Rockwood, A. D. Rutenberg, "Dynamical modelling of the frailty index indicates that health reaches a tipping point near age 75," arXiv:2412.07795 (2024). Damage and repair transitions fitted to longitudinal data from the Health and Retirement Study and ELSA. Robustness and resilience decline continuously; a tipping point appears near 75, where damage rate equals repair rate. Note the mechanism is a damage–repair balance, not a constitutive one. It establishes that a transition exists without establishing what quantity crosses it.

Restoration

"Exercise frequency determines heart rate variability gains in older people: a meta-analysis and meta-regression" (2019). Endurance training raises heart-rate variability past sixty, in a linear frequency-response relationship. The empirical basis for the re-tensioning step.

"Effect of exercise training on heart rate variability in healthy older adults," ***American Heart Journal*** **(1999).** Training raises total variability, with the most marked change in nocturnal heart rate. Variability is stable over a year in those who do not change activity level, so the gain is attributable to the training.

"Multicomponent exercise training in cardiovascular complexity in prefrail older adults: a randomized blinded clinical pilot study" (2021). Tests whether cardiac complexity, not merely variability, can be restored. The right endpoint, at pilot scale. Still missing is a trial reporting cross-entropy between coupled axes.