The Self-Forging Fire

Stress-Driven Phase Transitions as a Scale-Invariant Mechanism of Collective Intelligence Development

tratium

Abstract. Systems under stress either fragment or reorganize at higher capacity. We argue that this contrast can be modeled as a regime transition in which stress functions simultaneously as perturbation threatening coherence and, under constrained conditions, as cognitive glue enabling higher-order reorganization. Three independent bodies of evidence converge: (1) biological hormesis, where sub-lethal stressors upregulate adaptive capacity through biphasic dose-response dynamics (Calabrese, 2004); (2) organismic stress adaptation, where moderate adversity, exercise supercompensation, trained immunity, and allostatic load show the transition from adaptive challenge to structural degradation (Seery et al., 2010; Viru, 1984; Arts et al., 2018; McEwen, 1998); and (3) collective intelligence research, where stress sharing between cells enlarges cognitive light cones and enables morphogenetic problem-solving inaccessible to individual agents (Levin & Shreesha, 2024). We formalize the relationship between hormesis and antifragility: hormesis supplies the empirical dose-response curve, while antifragility supplies a curvature criterion for when stress variability improves expected function (Taleb & Douady, 2013). We connect these to the criticality literature: living systems that thrive under stress often operate near phase transitions where sensitivity and adaptiveness are maximized (Muñoz, 2018; Toker et al., 2022). The bidirectional outcome — growth or fragmentation from the same stressor — is conditioned by dose, recovery, prior state, and the system's proximity to criticality at the moment of perturbation. The scale-invariance claim is formal rather than literal: the pattern recurs across domains, but the mechanism may not. We propose a unified phase diagram, introduce domain-specific anti-fragility indices, and generate testable predictions across scales.

Keywords: antifragility, hormesis, criticality, phase transitions, collective intelligence, post-traumatic growth, allostatic load, stress sharing, self-organized criticality

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1 Introduction

The observation that stress can produce growth is ancient. "What does not destroy me makes me stronger," wrote Nietzsche in 1888, compressing into an aphorism a pattern recognized across philosophical, contemplative, and martial traditions. The observation that stress can produce destruction is equally obvious. What has not been established is whether these two outcomes — growth and fragmentation — represent a single formal pattern operating in different regimes, and whether that pattern is scale-invariant: the same mathematical structure recurring at the level of cells, organisms, and populations.

This paper argues for a weak scale-invariance claim: not that cells, organisms, and societies are literally the same system, but that a common formal structure recurs across them. The proposed mechanism is a regime transition in the information-processing capacity of a system under stress. Below a critical stress threshold, the system's adaptive machinery is activated without being overwhelmed — the system may reorganize at higher capacity. Above the threshold, the same machinery is overwhelmed and the system fragments — losing coherence, reverting to lower-order dynamics, or dissolving entirely. The transition between these regimes is nonlinear and, in the strongest cases, formally analogous to phase transitions in physical systems.

The claim that this formal pattern is scale-invariant rests on the convergence of three independent research programs that have not previously been unified:

At the cellular level, the hormesis literature (Calabrese, 2004; Calabrese & Blain, 2005) documents a biphasic dose-response relationship in which sub-lethal stressors — toxins, heat, reactive oxygen species, caloric restriction — upregulate adaptive capacity through heat shock proteins, mitochondrial biogenesis, and DNA repair enzymes. The hormetic zone is narrow: typically 30–60% stimulation above control at doses below the no-observed-adverse-effect level (Calabrese, 2004). Above this zone, the same stressors produce damage. Levin and Shreesha (2024) demonstrated computationally that stress sharing between cells functions as "cognitive glue" for collective intelligences — the mechanism by which individual cellular agents with small cognitive light cones merge into a collective capable of solving morphogenetic problems none of them can solve alone.

At the organismic level, the strongest evidence comes from functional stress-adaptation curves rather than self-reported growth narratives. Seery, Holman, and Silver (2010) document a U-shaped relationship between lifetime adversity and resilience: individuals with moderate adversity show better mental health outcomes than those with either no adversity or high adversity. Exercise training shows the same temporal logic in supercompensation: stress temporarily depresses capacity, recovery restores it above baseline, and insufficient recovery produces overtraining rather than growth (Viru, 1984). Trained immunity provides an immunological example: BCG vaccination can epigenetically reprogram monocytes and improve resistance to unrelated viral challenge (Arts et al., 2018). McEwen's allostatic load model (1998, 2017) provides the mechanism for the upper boundary: when chronic stress exceeds the organism's adaptive capacity, the stress response system itself becomes pathological — structural brain changes, chronic inflammation, autonomic rigidity. The transition from adaptive allostasis to allostatic overload is characterized by nonlinear dynamics and hysteresis. Post-traumatic growth is discussed below as a weaker, contested candidate rather than as load-bearing evidence.

At the collective level, criticality in biological networks is now well established. Neuronal avalanches follow power-law distributions consistent with systems at a critical phase transition (Beggs & Plenz, 2003). Biological regulatory networks are significantly closer to criticality than random networks (Daniels et al., 2018). Starling flocks exhibit scale-free correlations spanning the entire collective (Cavagna et al., 2010). Mühlmann (2005) argues that human cultures are shaped by "maximal stress cooperation" events — population-scale stressors that drive collective reorganization. This civilizational extension is more speculative, but it connects to formal work on complex adaptive systems (Holland, 1992), cliodynamic cycles of demographic stress and state instability (Turchin & Nefedov, 2009), and collapse as diminishing returns to social complexity (Tainter, 1988).

We propose that these observations describe a single formal pattern across scales: stress can drive a system toward a regime boundary. Systems near criticality — poised between order and disorder — can sometimes use the perturbation to reorganize at higher capacity. Systems far from the adaptive range fragment. The golden ratio's anti-resonance properties, which we analyzed in a companion paper (tratium, 2026), may provide one mechanism by which living systems maintain themselves near the critical point where stress produces reorganization rather than collapse.

2 Cellular Scale: Hormesis and Stress Sharing

2.1 The Hormetic Dose-Response

Hormesis — the biphasic dose-response in which low-dose exposure to a stressor produces beneficial effects while higher doses produce harm — was marginalized in toxicology for decades but is now recognized as a general biological phenomenon. Calabrese's systematic review of the toxicological literature identified hormetic responses across chemical classes, biological models, and endpoints (Calabrese & Blain, 2005). The hormetic zone is remarkably consistent: stimulatory responses typically peak at 130–160% of control values at doses below the NOAEL.

The molecular mechanisms of hormesis are well characterized. Heat shock proteins (particularly Hsp70) are upregulated in response to thermal, oxidative, and ischemic stress; prior stress exposure ("preconditioning") upregulates the chaperone network, providing enhanced tolerance to subsequent stress (Calabrese et al., 2015). Caloric restriction — a 30–40% reduction in caloric intake — extends lifespan across species through hormetic mechanisms: upregulation of antioxidant defenses, DNA repair, neurotrophic factors, and mitochondrial biogenesis (Mattson, 2008; Masoro, 2007). Exercise generates reactive oxygen species that at moderate levels trigger adaptive responses; antioxidant supplementation blocks these adaptations, demonstrating that the stress itself is the necessary signal (Ristow & Schmeisser, 2014). The CALERIE trial confirmed hormetic benefits of moderate caloric restriction in non-obese humans over two years (Most et al., 2017; Spadaro et al., 2022).

The hormetic curve has a characteristic mathematical form: a biphasic function with an overcompensation region at low doses superimposed on a monotonic dose-response at higher doses. The Brain-Cousens model (1989) adds a hormetic term to the standard log-logistic function, capturing both the stimulatory and inhibitory phases within a single parametric framework. This is not a threshold effect. It is a region of positive slope — active improvement — before the system transitions to the familiar dose-dependent decline.

2.2 Stress Sharing as Cognitive Glue

Levin and Shreesha (2024) extended the hormetic framework from individual cells to collective morphogenesis. Their computational model demonstrates that when stressed cells can export their stress molecules to neighbors — making "my problem your problem" — the collective achieves morphogenetic outcomes that are impossible without stress sharing. The mechanism requires no altruism: each cell simply minimizes its own local stress, but the leaking stress signal bends

the energy landscape for neighboring cells, inducing them to rearrange in ways that resolve the collective problem.

The key result is that stress sharing enlarges the cognitive light cone — the radius of concern, the scale of goals the system can represent and pursue. Without stress sharing, each cell optimizes only its own local state. With stress sharing, the cell's effective optimization domain extends to include its neighbors' states, because their stress has become its stress. The simulated embryos with stress sharing achieved perfect morphogenesis earlier and more reliably than those without (Shreesha & Levin, 2024).

This result connects to Levin's broader TAME framework (2022), which treats intelligence as a multiscale phenomenon: molecular networks, cells, tissues, organs, organisms, and swarms each solve problems in distinct "problem spaces." The transition from cellular to collective intelligence requires cognitive glue — mechanisms that align individual agents toward shared goals. Bioelectric networks provide one form of glue through memory anonymization: gap junctions allow physiological states to propagate between cells, blurring individual identities into collective ones. Stress sharing provides another — one mediated by the ancient, conserved stress-response machinery.

Cancer, in Levin's framework, is a failure of collective intelligence: "When cells become isolated from the information structure of the tissue, they revert back to an ancient, unicellular transcriptional and behavioral phenotype" (Levin, 2024). Normalizing the bioelectric microenvironment can revert cancer cells to normal behavior — the collective information field overrides individual cellular defects (Chernet & Levin, 2013, 2014). Cancer is fragmentation. Health is maintained collective coherence under stress. This is the cellular-scale instance of the bidirectional outcome: the same environmental perturbation produces either reorganization (morphogenesis, wound healing, regeneration) or fragmentation (cancer, developmental defect) depending on the integrity of the collective information architecture.

3 Organismic Scale: The Adversity Curve and Allostatic Load

3.1 The U-Shaped Adversity Response

Seery, Holman, and Silver (2010) demonstrated a U-shaped relationship between cumulative lifetime adversity and multiple health and well-being outcomes in a nationally representative U.S. sample ($N = 2,398$). People with some lifetime adversity reported better mental health, lower functional impairment, higher life satisfaction, and fewer PTSD symptoms following subsequent stressful events than either people with no adversity or people with high adversity. The quadratic effects were statistically robust and held across outcome measures.

This finding is the organismic analogue of the hormetic dose-response: moderate stress builds capacity; zero stress leaves the system undeveloped; excessive stress overwhelms the adaptive machinery. The parallels to biological hormesis are structural: the same biphasic pattern, the same narrow optimal zone, the same transition from beneficial to harmful at a dose threshold.

The post-traumatic growth literature (Tedeschi & Calhoun, 1996, 2004) provides a possible psychological analogue of the beneficial regime: growth requires "seismic" disruption to core beliefs,

followed by deliberate cognitive processing that revises rather than merely repairs the prior schema. The curvilinear PTG-distress relationship — moderate distress associated with maximal reported growth, low or high distress associated with minimal reported growth (Shakespeare-Finch & Lurie-Beck, 2014) — maps onto the phase-transition model, but it should not carry the argument. The stronger organismic examples are Seery's functional U-curve, exercise supercompensation, trained immunity, and allostatic overload.

Critical caveats are necessary. Boals (2023) presented a comprehensive case that self-reported PTG is largely illusory — driven by positive cognitive biases, demand characteristics of measurement instruments, and cultural narratives that valorize growth through suffering. The PTGI's unipolar scale (all items are positively worded) and suspiciously high internal consistency across supposedly distinct domains suggest response bias. Genuine PTG, Boals argues, is "very rare." This critique is essential for the present framework: it suggests that the "reorganize at higher capacity" outcome at the organismic level is rare precisely because the critical conditions are stringent. Most organisms under stress do not reorganize. They survive, cope, or fragment. Genuine reorganization at higher capacity requires specific conditions — sufficient but not overwhelming disruption, adequate processing resources, and access to schemas flexible enough to accommodate revision. The rarity of the outcome does not undermine the mechanism; it specifies the boundary conditions.

3.2 Allostatic Load as Phase Transition

McEwen's allostatic load model (1998, 2017) provides the mechanistic basis for the upper boundary of the adversity curve. Allostasis — "stability through change" — is the body's adaptive stress response: the HPA axis, the sympathetic nervous system, and the immune system coordinate to meet challenges and restore homeostasis. The response is adaptive when it activates efficiently and deactivates cleanly. The response becomes pathological — allostatic overload — under four conditions: repeated stressors without recovery, failure to habituate, failure to terminate the response, and inadequate response triggering compensatory hyperactivation.

The transition from adaptive allostasis to allostatic overload has the hallmarks of a dynamical phase transition. The system has two attractor states: (1) healthy allostasis, characterized by efficient activation-deactivation cycles, high heart rate variability, preserved hippocampal volume, and metabolic flexibility; and (2) allostatic overload, characterized by chronic activation, reduced HRV, hippocampal atrophy, amygdala hypertrophy, prefrontal cortex thinning, chronic inflammation, and metabolic rigidity (McEwen & Gianaros, 2011). The transition exhibits nonlinearity — small additional stressors can tip a near-threshold system into overload — and hysteresis — recovery from overload requires substantially more favorable conditions than the conditions that produced the degradation.

Heart rate variability is a candidate order parameter for this transition. HRV — the beat-to-beat variation in cardiac intervals — indexes autonomic flexibility: the system's capacity to modulate its state in response to changing demands. High HRV reflects a system with access to both sympathetic and parasympathetic resources, capable of fluidly shifting between activation and rest. Low HRV reflects a system locked into a defensive state, unable to modulate. HRV decreases with allostatic load (Jarczok et al., 2015) and predicts cardiovascular mortality, depression, and

all-cause mortality (Thayer et al., 2010). In the present model, HRV is not assumed to be the phase boundary itself; it is an observable summary of autonomic flexibility whose level and complexity should change nonlinearly as allostatic load approaches overload. The stronger claim — that HRV behaves as an order parameter — requires evidence of critical scaling, hysteresis, or abrupt loss of variability across the transition.

Rao and Bhatt (2019) developed a semi-mechanistic mathematical model of the HPA axis under chronic stress that demonstrates formally that chronic stress alters circadian rhythm parameters and shifts the system to a new steady state — a bifurcation in dynamical systems terms. This is the closest the allostatic literature has come to an explicit phase-transition formalization.

We propose that allostatic load represents a continuous control parameter driving the autonomic system through a phase transition. Below critical load, the system operates in the adaptive regime: stress activates the response, the response resolves, the system returns to baseline with enhanced capacity (hormesis). Above critical load, the system transitions to the overload regime: the stress response fails to deactivate, structural changes accumulate, and the system locks into a rigid defensive configuration from which recovery is costly and uncertain. The phase boundary is the point of maximum sensitivity — where the system is most responsive to both beneficial stressors (which push it toward reorganization) and harmful stressors (which push it toward fragmentation). This is criticality at the organismic scale.

4 Collective Scale: Criticality and Maximal Stress Cooperation

4.1 Criticality in Biological Networks

The evidence that biological systems operate near critical phase transitions is now extensive. Beggs and Plenz (2003) discovered neuronal avalanches in cortical tissue whose sizes follow power-law distributions with exponent $\alpha \approx -\frac{3}{2}$, matching the mean-field prediction for a system at a critical branching point. At criticality, neural networks maximize dynamic range (Kinouchi & Copelli, 2006), information transmission (Shew & Plenz, 2013), and sensitivity to inputs across scales.

Toker et al. (2022) found that consciousness is associated with near-critical cortical dynamics: waking consciousness is associated with edge-of-chaos dynamics, while loss of consciousness (anesthesia, deep sleep) corresponds to movement away from the critical point. Daniels et al. (2018) showed that biological regulatory networks — not just neural networks — are significantly closer to criticality than random networks, suggesting that natural selection has tuned regulatory architecture toward the phase boundary.

At the collective animal level, Cavagna et al. (2010) measured velocity correlations in starling flocks of up to 2,600 birds and found them to be scale-free: the correlation length scales with flock size, meaning the entire flock is correlated regardless of its extent. This is the signature of a system at a critical point, where correlation lengths diverge. The flock achieves maximum collective responsiveness — the capacity to respond as a unit to predator threats — by operating near the phase transition.

Mora and Bialek (2011) reviewed evidence across neural networks, gene regulation, flocking, and protein interactions, concluding that biological systems may generically evolve toward critical points because criticality maximizes dynamic range, information capacity, and sensitivity simultaneously.

4.2 Maximal Stress Cooperation

Mühlmann (2005) proposed that human cultures are shaped by "maximal stress cooperation" (MSC) events — population-scale stressors that trigger collective reorganization. The stress response at the population level follows the same biphasic logic as cellular hormesis: the stressor activates the HPA axis across the population, redirecting collective resources toward threat response. If the population successfully evaluates the stress — determines that the threat has been survived — the system accesses a post-stress relaxation phase characterized by hormonal recovery, immune restoration, and cultural consolidation. If the evaluation fails, the population remains in chronic collective stress, producing immunosuppression, cognitive impairment, and cultural fragmentation.

The evolutionary cooperation literature provides independent support. Ibbotson, Jimenez-Romero, and Page (2022) combined agent-based evolutionary modeling, mathematical analysis, and human experimental data in a Stag Hunt setting, finding that environmental harshness shifts cooperative decision-making and that cooperative foraging phenotypes survive harsh environments while paying miscoordination costs in favorable environments. Torney, Berdahl, and Couzin (2011) similarly showed that cooperative signaling can become stable when resources are sparse, ephemeral, and locally abundant. These results support a population-scale U-curve: moderate environmental stress can select for cooperation and collective intelligence; insufficient stress allows free-riding or undercoordination; excessive stress overwhelms cooperative capacity.

Levin's stress-sharing mechanism, demonstrated at the cellular level, has a structural analogue at the population level. When one group member's stress propagates to others — through empathy, social contagion, shared narrative, or institutional transmission — the group's effective cognitive light cone may enlarge. Individual members begin to optimize for collective goals because the collective's stress has become their individual stress. The mechanism is not identical across scales: cells exchange molecular stress signals through local biophysical channels, while human collectives exchange stress through cognition, institutions, media, and norms. The proposed commonality is formal rather than material: stress sharing bends the incentive landscape from individual optimization toward collective coordination, without requiring altruism.

This collective-scale claim is best understood against established theories of complex social dynamics. Holland's complex adaptive systems framework treats adaptation as an emergent property of interacting agents, tags, feedback, and recombination (Holland, 1992). Turchin and Nefedov's secular-cycle model identifies recurrent couplings among population pressure, elite competition, fiscal stress, and sociopolitical instability (Turchin & Nefedov, 2009). Tainter's collapse theory argues that societies often solve problems by adding complexity, but that added complexity eventually faces diminishing marginal returns, making further stressors more likely to produce simplification or collapse (Tainter, 1988). These frameworks do not prove the present phase diagram, but they define the empirical terrain any civilizational extension must satisfy.

5 The Unified Phase Diagram

5.1 Hormesis and Antifragility

Taleb and Douady (2013) provided the most rigorous mathematical formalization of antifragility: a system is antifragile if its transfer function is convex in the domain of stressors. By Jensen's inequality, a system with a convex response function benefits from volatility: $E[f(x)] > f(E[x])$ when f is convex. The system gains more from favorable fluctuations than it loses from unfavorable ones.

We observe a precise but conditional relationship between hormesis and antifragility. The hormetic dose-response curve (Calabrese, 2004) is characterized by low-dose improvement followed by high-dose impairment. Antifragility, however, is not defined by improvement alone. It is defined by convexity of the response function with respect to variation in the stressor. A positive first derivative, $f'(\sigma) > 0$, means that more stress improves output at that operating point; it does not by itself imply antifragility. Antifragility requires positive curvature, $f''(\sigma) > 0$, so that variable exposure at a fixed mean produces higher expected output than constant exposure at that mean. The hormetic zone is therefore not automatically identical to the antifragile zone. It is the empirical region in which antifragility can be tested by estimating local curvature.

This bridge, while structurally obvious once stated, has not been cleanly established in the peer-reviewed hormesis literature. Taleb discusses hormesis as a biological instance of antifragility in his popular work (2012), and Taleb and Douady (2013) define antifragility mathematically, but Calabrese's formalization of the dose-response curve (the Brain-Cousens model and its variants) does not frame hormesis through convexity or Jensen's inequality. Recent work has begun to operationalize antifragility in biological networks: Kim et al. (2020) measured antifragility in Boolean models of biological networks and used it to predict robustness and evolvability, while López-Díaz, Sánchez-Puig, and Gershenson (2023) found that heterogeneity broadens parameter regions associated with both criticality and antifragility in random Boolean networks. These papers support the measurability of biological antifragility, but they do not reduce hormesis to antifragility. The present contribution is the curvature criterion linking the two.

The unification is testable. Let $f(\sigma)$ represent the system's functional output (growth rate, information-processing capacity, fitness) as a function of stress dose σ. A hormetic response is characterized by a region where output exceeds baseline:

$$f(\sigma) > f(0) \quad \text{for some } \sigma \in (0, \sigma_{\text{harm}})$$

An antifragile response at operating point σ_0 requires:

$$f''(\sigma_0) > 0$$

By Jensen's inequality, for a zero-mean perturbation ε around σ_0:

$$E[f(\sigma_0 + \varepsilon)] > f(\sigma_0) \quad \text{when } f''(\sigma_0) > 0$$

Conversely, where $f''(\sigma_0) < 0$, stress variability lowers expected output even if the mean dose remains in a region where $f(\sigma) > f(0)$. This distinction matters. A system may be in the beneficial

hormetic range but already concave near the peak, meaning additional volatility is harmful. The antifragile portion of a hormetic curve is the locally convex subregion, not the entire region of above-baseline performance.

The phase-transition analogy attaches to the loss of curvature and the rapid change in susceptibility, not simply to the maximum of the response curve. In statistical mechanics, a critical point is defined by diverging susceptibility and correlation length; in biological dose-response data, the corresponding claim must be operationalized by measurable signatures such as abrupt changes in slope, hysteresis, critical slowing down, or scaling behavior. Systems that maintain themselves near such a boundary — through the anti-resonance mechanisms described in our companion paper (tratium, 2026) or through other homeostatic processes — are candidates for extracting developmental benefit from environmental volatility. The claim is therefore falsifiable: if fitted dose-response functions are not locally convex, or if volatility at fixed mean dose does not improve expected function, the hormesis-antifragility bridge fails for that system.

5.2 The Phase Diagram Across Scales

The unified framework maps onto a phase diagram that holds across scales:

Regime	**Stress response**	**System behavior**	**Outcome**
Understressed (subcritical)	Insufficient activation	Rigidity, underdevelopment, competitive exclusion	Stagnation
Optimally stressed (near-critical)	Hormetic benefit with local convexity	Reorganization, enhanced adaptive capacity	Growth
Overstressed (supercritical)	Concave response, allostatic overload	Fragmentation, loss of coherence, dissolution	Collapse

Table 1: The three-regime phase diagram. Growth is expected where hormetic benefit coincides with local convexity. Stagnation and collapse occupy the subcritical and supercritical regimes respectively.

This phase diagram instantiates differently at each scale but maintains the same formal structure:

Scale	Stressor	Growth regime	Fragmentation regime
Cellular	Toxins, heat, ROS, CR	Hormesis: HSP upregulation, mitochondrial biogenesis	Apoptosis, necrosis
Neural	Input variation, metabolic demand	Critical dynamics: neuronal avalanches	Seizure (supercritical) or quiescence (subcritical)
Organismic	Trauma, adversity	Post-traumatic growth, increased HRV	PTSD, allostatic overload, structural brain changes
Collective	Predation, environmental change, social disruption	Scale-free coordination, cooperative phenotypes	Institutional overload, social fragmentation

Table 2: Scale-specific instantiation of the phase diagram. The proposed commonality is formal: local convexity supports growth under variability, while departure from the adaptive regime produces fragmentation or overload.

5.3 Anti-Fragility Indices

To prevent the framework from becoming an unfalsifiable narrative ("everything is a phase transition"), each domain requires measurable indices tied to the predicted transition. Some indices estimate curvature directly; others estimate distance from criticality or loss of adaptive flexibility:

Scale	Anti-fragility index	Measurement
Cellular	Hormetic quotient	Ratio of stimulatory response to control at sub-NOAEL doses
Neural	Criticality distance	Avalanche exponent deviation from $-3/2$; branching ratio
Cardiac	HRV complexity	$1/f$ spectral exponent; multiscale entropy
Organismic	Allostatic load index	Composite biomarker score (McEwen's 10-item index)
Collective	Correlation length	Spatial extent of behavioral correlations relative to group size

Table 3: Domain-specific indices. A system is predicted to be in the growth regime when its index indicates local convexity, near-critical dynamics, or preserved adaptive flexibility; in the fragmentation regime when the index indicates departure from the adaptive range toward either pole.

6 Predictions

The framework generates testable predictions:

1. **Hormesis-antifragility bridge**: The convexity of the hormetic dose-response curve, measured by the second derivative of the Brain-Cousens model fit, should predict the system's benefit from dose volatility (measured by comparing outcomes under constant vs. variable dosing schedules

at the same mean dose). If the framework is correct, organisms receiving variable sub-NOAEL doses should outperform organisms receiving constant doses at the same mean only where the fitted response is locally convex — because Jensen's inequality favors convex payoffs under variance.

2. **HRV as phase-transition order parameter**: Heart rate variability should exhibit critical scaling (power-law fluctuations, long-range correlations, $1/f$ spectral character) in healthy systems and lose these signatures in both understressed (sedentary, unchallenged) and overstressed (allostatic overload) systems. The relationship between allostatic load score and HRV complexity should be nonlinear, with a critical threshold separating the adaptive and overload regimes.

3. **Stress sharing and collective problem-solving**: In Levin's morphogenetic model, blocking stress propagation (e.g., gap junction inhibitors in developing tissue) should reduce the effective cognitive light cone and impair collective morphogenetic accuracy. Enhancing stress propagation should enlarge it — up to a point, beyond which excessive stress sharing should produce collective overload (the concave regime at the collective scale).

4. **Cross-scale consistency**: Interventions that shift a system from the fragile to the antifragile regime at one scale should produce measurable effects at adjacent scales. Exercise (cellular hormesis) should increase HRV (organismic flexibility). High HRV should predict better functional recovery and resilience outcomes after stress exposure. Collective stress-sharing capacity should predict group-level adaptiveness.

5. **Anti-resonance and the maintenance of criticality**: Systems whose internal frequencies are tuned to golden-ratio-spaced (φ-spaced) ratios should show enhanced maintenance of near-critical dynamics under perturbation, relative to systems with harmonic (2:1) or random frequency spacing — the prediction from our companion paper (tratium, 2026) applied to the present framework.

7 Discussion

7.1 Limitations

The most significant limitation is that the scale invariance claimed here is demonstrated by structural analogy across independently studied systems, not by a single experiment spanning multiple scales. The claim is therefore weak scale invariance: shared mathematical form, not shared substrate or identical mechanism. Each link in the chain — hormesis, PTG, allostatic load, stress sharing, criticality — is independently supported to different degrees, but no experiment has manipulated stress at one scale and measured phase-transition effects at another. The cross-scale predictions in Section 6 are designed to test this gap.

The hormesis-antifragility bridge depends on curvature, not merely on above-baseline benefit. A reviewer could correctly object that many hormetic curves are concave near their peak, in which case variable dosing at a fixed mean would reduce rather than improve expected output. The framework therefore predicts local antifragility only in convex regions of fitted dose-response

functions. It also inherits the standard criticisms of hormesis: uncertain generalizability, risks of post hoc curve selection, incomplete mechanistic grounding across endpoints, and poor suitability as a default assumption in human risk assessment (Kitchin & Drane, 2005; Thayer et al., 2005; Mushak, 2007). These criticisms do not defeat the narrower claim that some biological stress responses are locally convex, but they rule out treating hormesis as universal or automatically beneficial.

The phase-transition language is also stronger than the evidence in several domains. In neural avalanches and flocking, scaling behavior and diverging correlation lengths make the analogy relatively precise. In allostatic load, the evidence is more indirect: nonlinear degradation, attractor-like pathology, and possible hysteresis are consistent with a dynamical transition, but HRV has not yet been shown to function as a strict thermodynamic order parameter. The paper therefore treats HRV as a candidate order parameter whose status must be established by the critical-scaling prediction in Section 6.

The post-traumatic growth literature is the weakest empirical link. Boals (2023) presents a compelling case that self-reported PTG is largely illusory. The Seery (2010) U-curve is more robust because it measures functional outcomes rather than self-reported growth, but the effect sizes are modest. The framework's prediction — that genuine reorganization at higher capacity is rare and requires specific boundary conditions — is consistent with Boals's critique but means the "growth" pole of the phase diagram is empirically thinner than the "fragmentation" pole.

Polyvagal theory, which provides a clinically influential model of autonomic state transitions under stress, is scientifically contested. Grossman (2023) challenges all five foundational premises of the theory. The dissolution hierarchy — the most relevant concept for the present framework — is the most contested element. We have used the polyvagal framework as a clinically observed pattern rather than an established mechanism, and the paper's formal claims do not depend on the specific phylogenetic narrative Porges proposes. What is well established is that autonomic flexibility (indexed by HRV) predicts stress resilience, and that chronic stress reduces autonomic flexibility. The phase-transition formalization does not require the polyvagal hierarchy to be correct — only that the autonomic system exhibits regime transitions between adaptive and maladaptive configurations.

The civilizational scale is the least empirically grounded. Mühlmann's MSC framework is theoretical, not experimental. Turchin and Nefedov's secular cycles and Tainter's collapse theory provide more rigorous historical frameworks, but they explain social instability through demographic-structural pressure, elite competition, fiscal stress, and diminishing returns to complexity rather than through biological stress sharing. The present paper can at most claim compatibility with those theories, not derivation from them. Extending cellular and organismic phase transitions to human societies requires bridging assumptions that the present data cannot fully justify.

7.2 Relation to Existing Frameworks

The framework proposed here connects to several existing theoretical programs:

Taleb's antifragility (2012) provides the general conceptual framework but lacks biological formalization. The present paper supplies the connection to hormesis and criticality that makes antifragility empirically tractable in biological systems.

Prigogine's dissipative structures (1977) establish the thermodynamic precondition: life exists far from equilibrium, and order arises from energy throughput. The present framework adds the informational complement: the system must not only be far from equilibrium but must be structured to resist falling back toward either equilibrium (order/death) or maximum entropy (chaos/death). The anti-resonance mechanisms described in our companion paper may provide one such structure.

Levin's TAME framework (2022) provides the biological foundation for scale-invariant intelligence. The present paper extends TAME's insights by proposing that stress sharing is one mechanism by which cognitive light cones can enlarge — not merely a perturbation to be survived but, under constrained conditions, an active ingredient in the transition from individual to collective intelligence.

Kauffman's edge-of-chaos framework (1993) and Bak's self-organized criticality (1987) provide the mathematical setting. The present contribution is the identification of a testable hormesis-antifragility bridge — local convexity in the stress-response function — as one mechanism by which biological systems may extract benefit from perturbation near critical regimes.

Holland's complex adaptive systems framework (1992) provides the general agent-based substrate for this argument: adaptation arises through distributed agents, feedback, and recombination rather than centralized control. Recent Boolean-network models of antifragility and criticality (Kim et al., 2020; López-Díaz et al., 2023) show that antifragility can be operationalized in biological network models. The present framework differs by linking that operationalization to empirical dose-response curves and allostatic physiology.

7.3 Broader Implications

If the framework is correct, it reframes the relationship between adversity and development. Stress is not merely something to be minimized. Below a threshold, stress is the mechanism by which systems develop capacities they could not develop in its absence. The cellular evidence is unambiguous: exercise, caloric restriction, and thermal stress all improve health through hormetic mechanisms that require the stress itself as the trigger. Blocking the stress (e.g., antioxidant supplementation during exercise) blocks the adaptation.

The framework also has implications for collective intelligence design. If stress sharing is one form of cognitive glue that binds individual agents into higher-order collectives, then systems designed to minimize individual stress may inadvertently prevent the emergence of collective intelligence. However, stress sharing has a dark twin: unbounded stress propagation produces not collective intelligence but collective seizure — epileptic synchrony in neural networks, cytokine storm in immune systems, mob panic in social groups, trauma synchronization in institutional settings. The optimal design therefore requires **bounded stress transmissibility**: a coupling strength parameter κ that determines how readily one agent's stress becomes another's. Too little coupling ($\kappa \approx 0$): isolated agents, no collective intelligence. Too much ($\kappa \to \infty$): collective seizure,

every agent overwhelmed by the system's aggregate stress. The optimal κ is itself conditioned by the system's anti-closure capacity — systems with stronger anti-resonance structure (higher resistance to pathological synchronization) can tolerate greater stress coupling before seizing. This interaction between anti-closure and stress coupling is developed further in the companion paper (tratium, 2026a) and will be formalized in a forthcoming synthesis.

The design principle is therefore not stress minimization but stress calibration: enough stress to activate locally convex adaptive responses, delivered with sufficient recovery time for consolidation, bounded coupling to prevent collective seizure, and maintained within a system whose anti-closure architecture preserves the near-critical operating regime. The integrated model requires two coupled parameters: an anti-closure index (α, measuring resistance to premature synchronization) and a stress load (σ, measuring perturbation intensity). Adaptive development occurs only in a bounded zone of $\alpha \times \sigma$ space: high enough α to prevent lock-in, calibrated σ to force reorganization, low enough σ to avoid overload. The full two-parameter framework is forthcoming.

For AI alignment, the framework suggests that artificial systems designed to be robust (indifferent to perturbation) may be suboptimal compared to systems designed to be antifragile (improved by perturbation). The criticality literature already suggests that neural networks at the edge of chaos exhibit superior computational properties. The present framework adds that one possible mechanism for maintaining edge-of-chaos dynamics is calibrated perturbation followed by consolidation, analogous to stress-driven reorganization in living systems.

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