ISSN: 3065-4874
Research Article
Journal of Psychiatry and Neurochemistry Research

Resilience as an Emergent Property of Allostatic Systems: An Integrative Framework from Developmental Mechanobiology to Systemic Physiology

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Submitted: 04 November 2025 **Accepted:** 13 November 2025 **Published:** 20 November 2025

https://doi.org/10.63620/MKJPNR.2025.1077

Citation: *Mundo, S. R., (2025). Resilience as an Emergent Property of Allostatic Systems: An Integrative Framework from Developmental Mechanobiology to Systemic Physiology. J of Psych and Neuroche Res, 3(6), 01-06.*

Abstract

Resilience has been traditionally conceptualized as an outcome of successful adaptation, but this narrative review proposes a transformative framework redefining it as an emergent property of allostatic systems calibrated during early development. Integrating evidence from embryological mechanobiology, allostatic physiology, and resilience research, we demonstrate how mechanical forces from the Spemann-Mangold organizer and notochord activate mechanotransduction pathways (YAP/TAZ) that converge with biochemical signals (Wnt/β-catenin, BMP/TGF-β, Sonic Hedgehog) on molecular integrators (mTOR, MAPK). This integration generates lasting epigenetic imprints that establish the operational parameters of the psychoneuroimmunoendocrine (PINE) system, thereby determining adaptive capacity throughout life. Resilience emerges as an observable manifestation of efficiently calibrated allostatic systems, evidenced by multisystem coordination, predictive regulation, and efficient recovery from challenges. This unifying framework transcends predominant fragmented views, providing foundations for mechano-epigenetic biomarkers and early interventions aimed at optimizing adaptive capacity. Translational implications include precision preventive medicine, intervention strategies during critical developmental windows, and reorientation of public health policies toward optimizing human adaptive potential. The proposed model addresses fundamental gaps in understanding how adaptive capacity emerges across the lifespan and offers novel approaches for promoting health through developmental optimization of allostatic calibration.

Keywords: Allostasis, Emergent Resilience, Mechanobiology, Embryonic Development, PINE System.

Introduction

Resilience as an Emergent Property of Allostatically Calibrated Systems – An Integrative Framework from Developmental Mechanobiology to Lifelong Adaptation.

Resilience—the capacity to maintain or restore physiological and psychological stability under adversity—has conventionally been conceptualized in psychological and neurobiological frameworks, typically as a fixed trait or an adaptive outcome [1-3]. While these views have advanced our understanding, they remain disconnected from critical advances in molecular biology and integrative physiology, thereby limiting a full exploration of the biological foundations of resilience [4]. Simultaneously, the concept of allostasis—as introduced by Sterling and Eyer and further elaborated by McEwen and Wingfield—has shifted the paradigm of stress physiology towards a dynamic view: stability is achieved through anticipatory and adaptive regulatory change [5, 6]. Despite this, existing literature mainly frames the relationship between allostasis and resilience as correlational, often lacking clear mechanistic explanation for how allostatic processes generate resilient phenotypes [7].

A transformative paradigm now emerges from developmental mechanobiology, which demonstrates mechanical forces during embryogenesis as instructive biological signals orchestrating cell differentiation and the foundation of tissue architecture. Pioneering studies show that structures like the Spemann–Mangold organizer and the notochord function as dynamic biomechanical signaling hubs, activating essential mechanotransduction pathways—including effectors such as YAP/TAZ —that establish

epigenetic imprints with lasting effects [8-10]. These imprints, as outlined in recent work, calibrate the plasticity and operational range of the psychoimmunoneuroendocrine (PINE) system, with deep implications for adaptive capacity across the lifespan. Integrating evidence from developmental mechanobiology, allostatic biology, and resilience research reveals a critical conceptual gap: while substantial progress has been made in each domain independently [11-13], these have rarely been synthesized into a model that explains how adaptive capacity is biologically "programmed" in early development. This narrative review adopts the perspective that resilience is best understood as a truly emergent property of allostatic systems, calibrated through mechanotransduction and epigenetic modification established during embryogenesis [14,15]. These early-life processes set the operational parameters—the "adaptive bandwidth"—of the PINE system, underpinning health and behavioral trajectories throughout life [16].

Methodologically, the present review employs explicit narrative synthesis following internationally accepted guidelines (SANRA criteria), with iterative literature searches in PubMed, Web of Science, and Scopus. We give preference to conceptual integration and mechanistic evidence over simple exhaustiveness, handpicking mechanistic studies, seminal reviews, and both animal and human research that bridge molecular, cellular, systems, and behavioral levels [17,18].

The central goal is to synthesize and integrate leading-edge evidence from developmental mechanobiology, allostatic programming, and resilience research, thereby proposing a paradigm shift in our understanding of adaptive capacity's biological roots—one with consequential applications for preventive medicine and early-life intervention. In prioritizing conceptual integration and transdisciplinary synthesis, this work seeks to transcend disciplinary silos and provide a new narrative for both research and clinical practice in the developmental programming of human resilience.

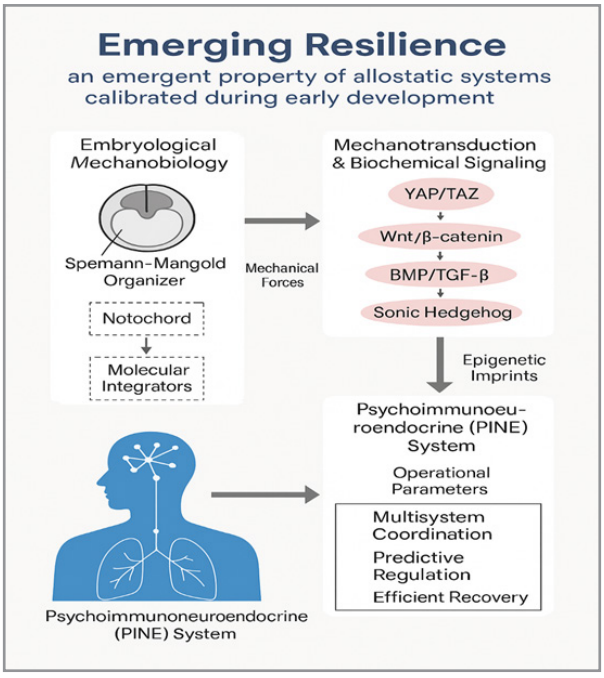

Figure 1: Expanded conceptual model: from embryonic mechanobiology and epigenetic marks to the emergence of systemic resilience. The figure depicts the flow from early developmental mechanical and molecular signals, through mechanotransduction and epigenetic calibration, to multisystem coordination and adaptive resilience. Original figure by the author, created with AI assistance.

Materials and Methods

Study Approach

Article type: Narrative review with original contribution (integrative model).

Purpose: To integrate evidence from developmental mechanobiology, epigenetics, mitochondrial bioenergetics, allostatic physiology, and resilience research in order to propose a unified conceptual model and translational agenda.

Time frame: Classic and contemporary literature (1924–2025), with emphasis on the past decade and articles in press when relevant for conceptual value.

Sources of Information and Search Strategy

Primary databases: PubMed/MEDLINE, Web of Science, Scopus. Supplementary repositories: PubMed Central (full text), project pages (e.g., MiSBIE), and NLM catalogs for verifying titles/ abbreviations.

Keywords and MeSH terms: allostasis; resilience; developmental mechanobiology; Spemann-Mangold organizer; notochord; YAP/TAZ; Wnt/β-catenin; BMP/TGF-β; Sonic Hedgehog; epigenetics; histone modifications; DNA methylation; microRNA; psychoneuroimmunoendocrine (PINE) system; mitochondria; bioenergetics; circulating cell-free mtDNA; neuroimmunology; PTSD resilience; cerebellum; Alzheimer's; immune evasion; cancer stem cells; epigenetic therapy.

Author's Bibliography Access: The public bibliography pro-

vided by the author was used to verify reference consistency and to retrieve key concept-supporting articles: https://www.ncbi.nlm.nih.gov/myncbi/samuel.ruesga%20mundo.1/bibliography/public/ Selection Criteria (narrative).

Inclusion: Original articles, reviews, and perspectives describing mechanisms, networks, or biomarkers with direct relevance to the development mechanical epigenetic–allostasis–resilience axis; translational and clinical studies with allostasis or resilience endpoints (e.g., PINE, HRV, cortisol dynamics, cytokines). Exclusion: Works without an explicit link to the integrative axes, non-indexed or low-traceability literature, redundant reports with no additional mechanistic value.

Screening and Extraction Procedure

Two-level iterative selection: (1) title/abstract review for mechanistic and translational relevance; (2) full-text reading and extraction of constructs, pathways, regulatory nodes, biomarkers, and clinical endpoints. Traceability: For each study, the axis of integration (mechanical, molecular, epigenetic, bioenergetic, PINE, clinical) and its contribution to the model were documented.

Conceptual Synthesis and Integration

Integration framework: A "domain-synthesis" approach was applied to assemble multiscale causal chains: embryonic mechanical forces → YAP/TAZ mechanotransduction → convergence with Wnt/BMP/Shh pathways → epigenetic imprints → PINE calibration → allostatic metrics → emergent resilience phenotype. Narrative cross-validation: Consistency was sought across levels (cellular–tissue–systems–clinical) and conditions (mental health, neurodegeneration, oncology) to strengthen generalizability of the proposed framework.

Original Contribution (conceptual methodology)

Definition: Resilience is formalized as an emergent property of ontogenetically calibrated allostatic systems, operationalized by multisystem coherence, predictive accuracy, energetic efficiency, and rapid recovery. Model construction: The model is derived abductively from convergent evidence, articulating mechanistic nodes (YAP/TAZ, mTOR/MAPK), epigenetic layers (NR3C1, FKBP5, H3K27ac, miRNAs), mitochondrial bioenergetics (MiSBIE), and cerebellar–cortical/neuroimmune networks (Alzheimer's cerebellum; PTSD–resilience).

Proposed endpoints: Multimodal allostatic resilience panel (cortisol dynamics, HRV, IL-10/IL-6, cf-mtDNA, GDF15/FGF21, connectivity and red cortex/cerebellar morphometry metrics, YAP/TAZ related epigenetic signatures).

Session Control and Limitations

Narrative nature: No metaanalysis or formal risk-of-bias assessment was performed; selection bias was mitigated by prioritizing high-impact journals, reproducibility (PMCID when available), and multiscale concordance.

Transparency: Gaps are explicitly reported (e.g., human longitudinality, causal development-adult bridges), and programs for experimental/translational validation are proposed.

Link to Translational Agenda

Hypothesis derivation: The model inspires intervention trials at critical windows (mechanosensitive nutrition, targeted photobiomodulation, YAP/TAZ/Rho-ROCK modulators), development of integrated resilience indices and mother-neonate childhood longitudinal cohorts. Biomarker plan: To test combined panels (HHA epigenetics; p-YAP/TAZ in PBMCs; mechanosensitive miRNAs; cf-mtDNA; HRV; IL-10/IL-6) and neuroimaging endpoints (fronto-cerebellar connectivity; MSN) for prediction and monitoring.

Ethical Aspects

No ethical approval was required as only published literature was used; proposed experimental studies are subject to review by appropriate ethics committees.

Results

Integrative Analysis of Developmental Allostatic Programming and Emergent Resilience

Mechanical Origins of Organization: From Embryonic Organizers to Cellular Decision Networks

The literature converges in demonstrating that the Spemann-Mangold organizer and notochord function as biomechanical centers that impose force geometries and stiffness patterns on developing tissues, triggering mechanosensitive pathways including YAP/TAZ that regulate developmental fate, patterning, and synchrony [19,20]. These physical signaling hubs operate in concert with conserved morphogens (Wnt/β-catenin, BMP/TGF-β, Sonic Hedgehog), forming an integrated decision network that translates mechanical and chemical inputs into cellular identity programs and axial architecture [21-24]. This network functions anticipatorily and sequentially, modulating not only morphogenesis but also establishing foundational physiological setpoints that influence lifelong adaptive capacity.

From Mechanical Signals to Molecular Memory: Stratified Epigenetic Imprints

Consistent evidence identifies that early mechanical forces—including tissue tension, deformation, and matrix stiffness—convert into lasting epigenetic modifications that stabilize transcriptional states. These include differential DNA methylation in HPA axis genes (NR3C1, FKBP5), histone modifications associated with chromatin accessibility (H3K27ac/H3K4me1), and mechanosensitive miRNA profiles . These epigenetic imprints function as "molecular memory" that persists beyond organogenesis, anchoring operational ranges for stress reactivity, inflammatory responses, and metabolic regulation. This molecular memory integrates with key transduction nodes (mTOR/MAPK) and YAP/TAZ cofactors to establish stable physiological setpoints [25].

Allostatic Calibration of the PINE System: Coordination, Energy Efficiency and Recovery

The integration of mechano-epigenetic signaling manifests in the precise calibration of the psychoneuroimmunoendocrine (PINE) system. Multiple functional signatures capture this calibration: high-frequency heart rate variability reflecting vagal tone and predictive regulation, cortisol dynamics demonstrating appropriate reactivity and recovery patterns, IL-10/IL-6 balance indicating anti-/pro-inflammatory equilibrium, and bioenergetic markers including cell-free mtDNA and mitochondrial stress factors (GDF15/FGF21). These metrics collectively indicate op-

timal energy allocation efficiency, anticipatory precision, and recovery capacity—defining attributes of systemic resilience that emerge from properly calibrated allostatic systems [26].

Cross-Domain Validation: Mental Health, Neurodegeneration and Cancer Biology

The integrative pattern demonstrates remarkable consistency across diverse clinical domains:

- Trauma and PTSD Resilience: Combined cortical network morphometry and transcriptomic analyses reveal that resilient individuals exhibit compensatory glial-neuronal reconfigurations consistent with more efficient allostasis and reduced energy expenditure.
- Neurodegenerative Resilience: In Alzheimer's disease, lateral cerebellar integrity ("cognitive cerebellum") and synaptic preservation in the granular layer associate independently with reduced dementia severity, suggesting cerebellar contributions to predictive coordination and cognitive resilience beyond classical neuropathological markers [27].
- Malignant Adaptation: Cancer stem cells exploit similar epigenetic plasticity and mechanosensitive pathways to sustain "malignant resilience" through immune evasion and therapeutic resistance, providing instructive reverse models for understanding adaptive network modulation.

Multimodal Biomarkers for Allostatic Resilience Assessment

We identify a complementary set of measures for quantifying and monitoring resilience capacity:

- Dynamic Physiology: Heart rate variability (including nonlinear measures), cortisol reactivity/recovery kinetics, composite allostatic load índices.
- Immunometabolic Profiling: IL-10/IL-6 ratios, interferon-type cytokine signatures, cell-free mitochondrial DNA, mitochondrial stress markers (GDF15/FGF21).
- Network Integrity Mapping: Fronto-cerebellar connectivity, morphometric similarity networks, executive and salience network integrity.
- Epigenetic and Signaling Signatures: NR3C1/FKBP5 methylation status, H3K27ac enrichment at inflammatory promoters, mechanosensitive miRNA profiles, YAP/TAZ and mTOR/MAPK activity in peripheral blood mononuclear cells .
- Bioenergetic Integration: Mind-mitochondria platform-derived indices coupling energy transduction with PINE coordination and behavioral outcomes.

Causal Consistency and Testable Predictions Across Biological Scales

The evidence demonstrates remarkable coherence from physical forces to clinical manifestations, with well-defined bridges at molecular (YAP/TAZ, mTOR/MAPK), epigenetic (DNA/histone/miRNA modifications), bioenergetic (mitochondrial function), and neural network (cortico-cerebellar integration) levels. This integrative framework generates falsifiable predictions:

- Targeted mechanical and photobiomodulation interventions should modulate chromatin accessibility and improve heart rate variability/recovery metrics.
- Neonatal mechano-epigenetic biomarkers will predict longitudinal resilience trajectories.
- Combined epigenetic and immunotherapeutic approaches can reverse "malignant resilience" in cancer stem cells, enhancing immune infiltration and antigen presentation.

Validation Framework and Clinical Translation Agenda

Priority research directions include:

- Longitudinal mother-neonate-childhood studies linking mechano-epigenetic signatures with PINE system endpoints and adaptive functioning
- Controlled trials of early-life interventions: mechanosensitive nutritional strategies (omega-3, vitamin D), gentle mechanomodulation, pulsed photobiomodulation, and Rho-ROCK/YAP-TAZ pathway modulators
- Development of an Integrated Allostatic Resilience Index combining heart rate variability, cortisol dynamics, inflammatory balance, mitochondrial markers, epigenetic profiles, and network neuroimaging with defined clinical thresholds for personalized prevention

Systemic Integration: From Developmental Origins to Precision

Medicine The collective evidence strongly supports the framework that resilience emerges from ontogenetically calibrated allostatic networks, is quantifiable through multimodal assessment, and is susceptible to targeted intervention. This model unifies developmental mechanobiology, epigenetic programming, bioenergetic regulation, and PINE system integration with concrete biomarkers and therapeutic targets, enabling a new era of preventive resilience medicine and precision health approaches across the lifespan.

Discussion

Towards a Unified Theory of Resilience—From Mechanical Forces to Lifelong Adaptation

This narrative review has synthesized evidence from developmental mechanobiology, allostatic theory, and resilience research to propose a paradigm shift: resilience is not merely a psychological trait or a fortunate outcome, but an emergent property of a complex system whose operational parameters are calibrated during early development through mechanotransduction and epigenetic programming. Our integrative framework moves beyond correlational models to propose a causal chain linking embryonic biomechanics to the functional bandwidth of the psychoimmunoneuroendocrine (PINE) system across the lifespan.

Reconceptualizing Resilience as Calibrated System Capacity

The conventional view of resilience as a fixed, neurobiological trait is insufficient to explain the dynamic and developmental aspects of adaptation. Our synthesis aligns with and extends the allostatic model by identifying the origins of the system's predictive capacity. The evidence demonstrates that the Spemann-Mangold organizer and notochord are not merely morphological structures but foundational biomechanical hubs that, through effectors like YAP/TAZ, establish the very epigenetic landscape that will govern stress reactivity, inflammatory balance, and energy metabolism. This positions resilience not as an outcome to be achieved, but as a pre-configured capacity for efficient allostasis, emerging from a system that was optimally calibrated from its inception.

The Mechano-Epigenetic Bridge: A Plausible Causal Pathway

A central pillar of our framework is the mechanistic bridge formed by developmental mechanobiology. The literature compellingly shows that mechanical forces are potent biological signals. The activation of YAP/TAZ and other mechanosensitive pathways directly influences chromatin accessibility and epigenetic marking, creating a "molecular memory" of the early mechanical environment. This memory, manifesting in the methylation patterns of genes like NR3C1 and FKBP5, sets the gain on the PINE system's responsiveness. This provides a testable, mechanistic explanation for how early-life experiences—from physical forces in utero to the psychological stress of the caregiver—get "under the skin" to permanently shape adaptive capacity.

Validation Through Cross-Domain Pathophysiology

The robustness of this framework is underscored by its explanatory power across disparate disease models. In PTSD, the resilient phenotype is associated with neural and transcriptomic signatures indicative of more efficient energy use and better predictive regulation, consistent with a well-calibrated allostatic system. In Alzheimer's disease, the identification of cerebellar substrates of resilience highlights the role of brain networks involved in coordination and predictive timing—key functions for allostatic regulation. Perhaps most instructively, the phenomenon of "malignant resilience" in cancer stem cells demonstrates how the very same principles of epigenetic plasticity and adaptive network recalibration can be co-opted for pathological survival, reinforcing the idea that resilience is a fundamental property of complex biological systems.

Clinical and Translational Implications

From Measurement to Intervention This unified theory transforms the approach to resilience promotion from reactive psychological support to proactive biological and biobehavioral calibration. The identified multimodal biomarkers—from heart rate variability and cortisol kinetics to cell-free mtDNA and epigenetic signatures —provide a toolkit for quantifying allostatic capacity rather than just allostatic load. This enables a shift towards pre-emptive medicine: identifying individuals with restricted "adaptive bandwidth" early in life and intervening with targeted strategies. These could include nutritional interventions that support mitochondrial and epigenetic health, gentle mechanomodulation therapies informed by developmental principles, or even future pharmacological agents targeting pathways like Rho-ROCK/YAP-TAZ to safely expand resilience capacity.

Limitations and Future Directions

We acknowledge that this integrative model is a synthesis designed to generate hypotheses, and its propositions require rigorous empirical validation. Key challenges include the technical difficulty of longitudinally tracking mechano-epigenetic-PINE pathways from development into adulthood and the need for advanced computational models to handle the resulting multi-omics data. Future research must prioritize longitudinal birth cohorts, controlled trials of mechanobiological and epigenetic interventions, and the development of a validated Integrated Allostatic Resilience Index for clinical use.

Conclusion

In conclusion, by integrating the language of developmental forces with the principles of allostatic regulation, we provide a transdisciplinary narrative that traces the lineage of resilience back to our earliest physical origins. This framework argues that the capacity to withstand life's adversities is deeply rooted in the mechanical and molecular events that build our bodies, offering a powerful new foundation for enhancing human health and resilience through the lens of developmental programming and precision medicine.

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