The Relational Substrate of Reflective Consciousness: A Metabolic Constraint Model

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Abstract

The Problem: Despite theoretical embrace of embodied and relational perspectives, experimental neuroscience and clinical practice treat regulation as an individual capacity. Standard neuroimaging isolates participants from attachment figures; diagnostic manuals code psychopathology as internal attributes; interventions target individual cognition. This "individualist artifact" systematically inflates baseline metabolic cost estimates and may misattribute isolation-induced variance to intrinsic neural function.

Objective: This theoretical review addresses this limitation by integrating four established frameworks to develop a model of reflective consciousness as a metabolically constrained state. We formalize co-regulation as thermodynamically necessary—not merely computationally beneficial—for sustained higher-order cognition, specifying the energetic conditions under which complex predictive processing becomes biologically affordable.

Framework Synthesis: Integrating Dynamic Systems Theory, Social Baseline Theory, Active Inference, and Affective Neuroscience, we model the integrated, narrative self as a high-cost predictive state whose stability depends critically on co-regulatory scaffolding. We propose that the "relational nexus"—not the isolated individual—represents the metabolically optimal configuration for reflective consciousness.

Core Claims: Four claims follow: (1) higher-order cognition is contingent upon somatic solvency; (2) co-regulation functions as a metabolic subsidy, lowering the energetic cost of prediction error minimization; (3) psychopathology represents collapse into rigid, metabolically trapped attractor states; and (4) symbolic attachment provides a costly but viable alternative to physical co-regulation.

Predictions and Solution: The framework generates eight falsifiable predictions, including a core experimental test: metabolic stress should selectively impair mentalization while sparing basic cognitive control, with this impairment mitigated by partner presence but not stranger presence. By repositioning the dyad as the metabolically efficient baseline, this framework offers both methodological correction (accounting for isolation-induced variance in neuroimaging) and clinical insight (explaining why individual-focused interventions often fail when relational substrate remains impoverished).

Highlights

- Reflective consciousness is modeled as a high-cost metabolic state.
- Co-regulation subsidizes the bioenergetic cost of predictive coding.
- Solo-regulation is not baseline but a metabolically expensive threat state.
- Psychopathology functions as a collapse into rigid, metabolically inefficient attractor states (computationally simple but systemically exhausting).
- Interventions show super-additive effects by restoring regulatory capacity.

Keywords

Active Inference; Allostasis; Attachment Theory; Co-regulation; Embodied Cognition; Interoception; Metabolic Constraint; Reflective Consciousness; Social Baseline Theory; Somatic Regulation

Abbreviations: RAD (Reactive Attachment Disorder), BPD (Borderline Personality Disorder), DID (Dissociative Identity Disorder), DMN (Default Mode Network), HRV (Heart Rate Variability), HiTOP (Hierarchical Taxonomy of Psychopathology), BDNF (Brain-Derived Neurotrophic Factor), ACE (Adverse Childhood Experiences), CLD (Causal Loop Diagram)

1 1. Introduction: The Metabolic Case Against Individualism

This review integrates Dynamic Systems Theory, Social Baseline Theory, Active Inference, and Affective Neuroscience to formalize the metabolic constraints on reflective consciousness.[1] These frameworks converge on a shared but underspecified constraint: reflective consciousness is a metabolically expensive achievement requiring relational scaffolding. Formalizing this constraint generates eight falsifiable predictions (Section **??**) that no parent framework generates alone.

Each framework contributes distinct mechanistic insights: Dynamic Systems Theory provides the ontological foundation (soft assembly, emergent properties); Social Baseline Theory provides bio-energetic evidence (metabolic efficiency of partnership); Active Inference provides the computational mechanism (prediction error minimization); and Affective Neuroscience specifies neural substrates (primary emotional systems).

The Individualist Artifact: Despite theoretical embrace of embodied perspectives, experimental designs and clinical interventions often treat regulation as an individual capacity. Standard neuroimaging isolates participants from attachment figures, potentially inflating baseline metabolic cost estimates by inducing artificial solo-regulation. Diagnostic manuals (DSM-5-TR, ICD-11) code anxiety and depression as internal attributes rather than organism-environment coupling failures.[2] Treatment models often target individual cognition as if the mind were software running on isolated neural hardware. If reflective consciousness strains solitary nervous systems under load, then the individual may not be the most productive unit of analysis.

We distinguish **phenomenal consciousness** (raw subjective experience) from **reflective consciousness** (integrated, temporally-extended narrative self-awareness enabling mentalization). Our claim concerns reflective consciousness as a metabolically expensive achievement contingent on co-regulation, not the creation of qualia.

Four Foundational Claims:

1. **Distributed Mind:** Regulation is a somatic process, not purely cognitive **??**.

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[1] We employ *integrative framework synthesis*—a methodology combining complementary theoretical perspectives to yield testable hypotheses that no single parent theory produces alone **?**. The criterion is generative power, not exhaustive coverage.

[2] Relational context is relegated to supplementary V-codes or Z-codes.

2. **Somatic Gating:** Autonomic state determines whether higher-order cortical functions are metabolically affordable.
3. **The Dyadic Baseline:** The isolated individual represents a "high-load" condition; the dyad is the efficiency baseline **??**.
4. **Systemic Pathology:** Psychopathology represents collapse into rigid attractor states.

This framework operates as a probabilistic constraint, not deterministic law. Genetic and temperamental differences modulate regulatory cost (Section **??**).

Novel Contributions: The synthesis generates eight predictions (Section **??**) not derivable from any parent framework alone, including: (1) selective mentalization impairment under metabolic stress, mitigated by partner presence; (2) threshold effects with hysteresis; (3) super-additive intervention effects; and (4) disorganized attachment's metabolic signature. All predictions are empirically falsifiable using existing methodologies.[3]

1.1 1.1 Key Concepts and Definitions

Reflective consciousness:[4] The integrated, temporally-extended narrative self-awareness enabling mentalization, autobiographical memory, and complex self-modeling. Our claims concern *durability and metabolic sustainability* of this state, not mere existence. Individuals can maintain reflective consciousness under high cost through effort (e.g., emergency workers under chronic stress), but such states lead to allostatic overload. Co-regulation lowers this cost. Predictions are strongest for reflective consciousness *under challenge*—during stress or metabolic constraint.

Operationalization for Empirical Validation: We model reflective consciousness as the latent variable explaining covariance between: (1) *Behavioral*: Reflective Functioning Questionnaire (RFQ) scores **??** capturing mentalization capacity; (2) *Neural*: Default Mode Network (DMN) connectivity (posterior cingulate cortex-medial prefrontal cortex [PCC-mPFC] coupling) **?** supporting autobio-

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[3] FDG-PET, fMRI, hypoglycemic clamp, dyadic paradigms. The framework would be disconfirmed if metabolic stress produced uniform cognitive impairment rather than selective mentalization deficits, or if partner presence failed to mitigate this impairment.

[4] Distinct from (a) metacognition (monitoring cognitive processes), (b) basic self-awareness (proprioceptive/interoceptive self-modeling), and (c) phenomenal consciousness (raw subjective experience). Reflective consciousness refers to the integrative, narrative capacity binding these into a temporally coherent self-model.

graphical integration; (3) *Narrative*: Adult Attachment Interview coherence **?** quantifying temporal self-representation.

Key Discriminating Prediction ("Hollowed Out" Reflective Capacity): Our model generates the falsifiable prediction that metabolic stress will cause these measures to decouple in predictable directions: behavioral mentalization (low metabolic cost) may be preserved while DMN integrity (high metabolic cost) degrades. This "hollowed out" pattern—where individuals can still perform mentalization tasks but show degraded neural integration—is uniquely predicted by the metabolic constraint framework. **Competing individualistic models predict uniform degradation across all three domains.** This dissociation provides the cleanest empirical test distinguishing our framework from alternatives.

Table 1: Recommended Assessment Protocols

Construct	**Measure**	**Timing Considerations**
Reflective Function	RFQ-8; RF Scale	Pre/post metabolic manipulation
DMN Connectivity	rs-fMRI (PCC-mPFC coupling)	During co-regulation vs. isolation
Attachment Security	AAI; ECR-R	Baseline predictor variable
Metabolic Load	Blood lactate; glucose variability	Continuous during task
Co-regulation Quality	Physiological synchrony (RSA, EDA)	Real-time dyadic measurement

Note: For metabolic studies testing this framework, timing is critical. Metabolic measures should be captured continuously; neural measures should compare co-regulated versus isolated conditions within-subjects; behavioral measures should bracket metabolic manipulations to capture state changes.

Somatic Cost: The metabolic demand associated with predictive operations, distinguished across three levels:

- **Energetic Cost:** Glucose and oxygen consumption for neural activity and allostatic regulation **?**, reflecting cumulative allostatic load. Pyka et al. **?** measured 8% elevated medial prefrontal glucose consumption during solo mentalizing (FDG-PET), confirming measurable metabolic cost. Converging evidence shows social support reduces neural threat response:

partner hand-holding attenuates amygdala and dorsal anterior cingulate activation during threat anticipation **?**. Our model predicts co-regulatory presence significantly reduces this consumption.

- **Computational Cost:** Predictive model complexity, including precision-weighting and data stream integration **?**.
- **Experiential Cost:** Subjective strain, fragmentation, or affective overload when costs exceed capacity.

Co-regulation reduces energetic cost of meaning-related predictions, making them computationally and experientially tractable. **Co-regulation represents the metabolically optimal configuration, not the only viable one.** Solo-regulation requires high intrinsic efficiency, robust internalized scaffolding, or symbolic substitutes (e.g., internalized attachment figures).

Levels of Prediction: We distinguish three levels of prediction to clarify the specific domain of our hypothesis:

1. **Sensorimotor Prediction:** Predicting immediate sensory consequences of action (e.g., the tactile feedback from grasping a cup). These are computationally and metabolically low-cost.
2. **Contextual Prediction:** Predicting event sequences and their likely emotional valence (e.g., a door slamming predicts a probable argument). These carry moderate cost.
3. **Meaning-Related Prediction:** Predicting abstract, self-referential, and social mental states across time (e.g., "What does this event mean for my identity?"; "How do I integrate this experience into my life narrative?"). These are exceptionally high-cost, requiring simultaneous integration of interoceptive, exteroceptive, episodic memory, and abstract self-models. **Our hypothesis specifically concerns the metabolic constraint on this third level of prediction.**

Hierarchical Cascade: Meaning-related prediction errors cascade down to contextual and sensorimotor levels. Identity threats (Level 3) manifest as hypervigilance (Level 2) and somatic symptoms (Level 1). Conversely, somatic interventions create metabolic headroom for higher-level reprocessing.

Solo-regulation requires the individual to bear full somatic cost while monitoring an uncertain environment (high-energy state). **Co-regulation** provides external scaffolding, aiding error minimization through soothing, mirroring, and anticipated care. Co-regulation lowers cost below

Table 2: Three Levels of Prediction

Level	**Examples**	**Metabolic Cost**	**Co-Regulation Impact**
Sensorimotor	Grasping a cup, walking	Low	Minimal
Contextual	Door slam → argument likely	Moderate	Partner reduces uncertainty
Meaning-Related	"What does this mean for my identity?"	High	Partner absorbs predictive load

the threshold required for reflective consciousness to stabilize; without it, the system may fragment or regress to rigid attractors—self-reinforcing patterns that are computationally simple but metabolically inefficient, keeping the HPA axis chronically activated (Section 4.1).

1.2 1.2 Relation to Existing Frameworks

Table **??** summarizes what each parent framework generates alone versus what the metabolic constraint synthesis adds.

The synthesis identifies metabolic cost of high-level prediction as a common constraint each framework touches but none centers. This constraint grounds Social Baseline Theory's findings in energetic efficiency, extends Active Inference to dyadic systems, operationalizes Attachment Theory's "secure base" as metabolic subsidization, and explains autonomic gating as energetic triage.[5]

2 2. The Somatic Architecture and Metabolic Constraints

Higher-order cognition is an emergent property contingent on the body's regulatory state, not a primary driver.

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[5] We acknowledge debate regarding Polyvagal Theory's neuroanatomical claims **?**, but the core observation—autonomic state modulates social engagement—is well-supported independent of contested mechanisms.

2.1 2.1 Allostasis and Autonomic Gating

The brain's primary function is allostasis—anticipating and meeting metabolic needs **??**. Higher-order computation is gated by regulatory success **??**. Threat states produce vagal withdrawal, cognitive narrowing, and reduced social engagement capacity (Neurovisceral Integration Model **?**; Polyvagal Theory **?**).[6] Complex reflective thought requires physiological safety (indexed by vagal tone). Under threat, metabolic resources shift toward survival circuits, taking neural substrates for reflective consciousness "offline."

2.2 2.2 The Energetic Budget of Affect

The brain prioritizes predictive regulation over reactive processing **???**. Panksepp's affective systems map onto our three prediction levels (Table **??**): SEEKING/PLAY (sensorimotor, low cost) encourage exploration when energy permits; FEAR/RAGE (contextual, moderate cost) override exploration for survival, rapidly depleting glucose; CARE and PANIC/GRIEF (meaning-related, high cost) regulate social bond stability—the primary allostatic load-sharing mechanism **??**.

Reflective consciousness requires CARE-system energy surplus to inhibit chronic FEAR/PANIC activation **?**. Trauma represents not "bad memories" but persistent metabolic regime alteration—chronically high-cost operation leaving minimal capacity for reflective consciousness. Throughout this paper, "metabolic cost" refers to measurable expenditures: glucose utilization (FDG-PET **?**), autonomic load (HRV, cortisol **?**), and allostatic wear. The claim concerns *constraint and affordability*: exhausted budgets make higher-order functions unsustainable, not impossible.

3 3. The Self-with-Other as the Basic Biological Unit

If reflective consciousness requires metabolic subsidization, the isolated individual cannot be the primary unit of analysis. Convergent research establishes the dyad as the baseline for maintaining narrative self. The metabolic constraint likely shaped selection pressures for social adaptations: if co-regulation reduces the cost of meaning-related prediction, then selective advantages of higher-order cognition would amplify adaptations enhancing co-regulatory capacity **?**. This predicts quantifiable

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[6] Debate exists regarding Polyvagal Theory's neuroanatomical claims **??**. Our argument requires only the well-documented gating phenomenon: threat → vagal withdrawal → cognitive narrowing.

metabolic benefits of co-regulation (Prediction 1). This aligns with Tronick's foundational concept of "dyadically expanded states of consciousness,"- where the dyad enables cognitive capacities unavailable to the individual alone **?**.

3.1 3.1 Convergent Evidence

Independent frameworks converge on the dyadic baseline. Dynamic Systems Theory **?** shows the self is "soft-assembled" from organism-environment coupling. Social Baseline Theory demonstrates the brain expects social resources **??**. Allostatic Regulation **?** shows context-dependent body budgeting persists in adulthood. Active Inference **??** formalizes how dyadic interaction reduces mutual prediction error. Attachment Theory describes internal working models formed through dyadic affect regulation **???**. Affective Neuroscience **?** specifies which circuits co-regulation modulates.[7]

Recent meta-analytic evidence demonstrates that interpersonal autonomic nervous system (ANS) synchrony—the spontaneous temporal coordination of physiological processes between individuals—is associated with relationship quality and performance outcomes **?**. Critically, sympathetic synchrony shows positive associations with relationship outcomes, while parasympathetic synchrony shows negative associations, suggesting that co-regulation involves active sympathetic coordination rather than mere parasympathetic dampening. This is further supported by evidence that co-regulation of sympathetic and behavioral rhythms during social interaction predicts attraction and bonding **?**, demonstrating that physiological synchrony serves as a mechanism for relationship formation. These findings provide empirical grounding for co-regulation as a physiological mechanism that operates through ANS synchrony, supporting the metabolic subsidy hypothesis.

3.2 3.2 The Convergent Logic

The evidence creates a convergent case: If cognition is "soft-assembled" from organism-environment coupling **?**, and the brain assumes partnership as its metabolic baseline **??**, then regulation is fundamentally relational. The "independent adult" is not a developmental endpoint but a metabolically

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[7] Framework mapping: Dynamic Systems (ontology); Social Baseline (bio-energetics); Allostatic Regulation (infant-adult bridge); Active Inference (computation); Attachment Theory (development); Affective Neuroscience (neural substrates).

expensive achievement that shares key physiological signatures with chronic low-grade threat response (elevated cortisol, reduced vagal tone, increased amygdala activation).

Barrett's allostatic framework **?** bridges infant and adult: body budgeting never becomes context-independent; rather, context becomes internalized. Recent work explicitly models attachment relationships as predictive coding systems **?**, where internal working models function as generative models that reduce prediction error in social contexts. Solo-regulation draws on representations of past co-regulatory experience, with metabolic cost increasing under uncertainty or stress. Apparent self-sufficiency runs on internalized relational scaffolding—when absent or damaged, meaning-making cost becomes prohibitive.

3.3 3.3 The Still-Face as Metabolic Crisis

The Still-Face effect represents metabolic crash, not mere behavioral distress. Meta-analyses confirm robust vagal withdrawal (RSA **?**) and cortisol spikes **?**. Infant gaze aversion is energy conservation—shutting down visual cortex processing when the "external battery" (caregiver) disconnects.

3.4 3.4 From "Dyad" to "Relational Nexus"

Our model relies on a Stable Relational Nexus—whether single parent or distributed alloparenting network—providing a predictable regulator that absorbs prediction error, lowering somatic cost.

Solo-regulation represents internalization of this relational matrix, not its absence. This internalization permits solitary self-coherence but remains metabolically costly, depending on initial scaffolding quality. Prolonged isolation increases somatic cost, eventually eroding narrative coherence (isolation psychosis), demonstrating relational subsidy as ongoing requirement.

The "autonomous individual" promoted by adult nosology (DSM-5) and certain cognitive therapies represents not a biological baseline but a high-energy configuration. Autonomy requires internalized relational capital; chronic solo-regulation resembles chronic threat response. Resilience reflects accumulated *relational capital*—internalized co-regulatory scaffolding. "Alone time" for securely attached adults is better understood as *internalized together time*, metabolically subsidized by relational resources. This capacity depletes under chronic stress, particularly for those with impoverished relational histories.

3.5 3.5 Bridging Computational Sufficiency and Metabolic Sustainability

Recent computational accounts, such as the minimal theory of consciousness implicit in active inference proposed by Whyte et al. **?**, formalize conscious access as belief updating at the discrete-continuous interface where posteriors inform counterfactual policy selection. This provides an elegant explanation for *how* consciousness emerges from predictive processing. However, as Whyte et al. themselves acknowledge, such frameworks do not typically address the metabolic constraints that determine *when* such computations are biologically affordable. This is not a minor omission—it is the difference between computational sufficiency and biological plausibility.

The Metabolic Lacuna and Thermodynamic Necessity: Active inference models often assume continuous scaling of belief precision with attention. Our framework predicts a metabolic threshold below which precision cannot be sustained, leading to discrete collapse rather than graded degradation. While active inference is metabolically efficient in a computational sense, minimizing the path length of belief updating **?**, this does not mean the system can always afford the energy to perform the computation. We therefore move from treating co-regulation as merely computationally beneficial (reducing mutual prediction error) to being *thermodynamically necessary*—the metabolic subsidy required to cross the threshold for sustained reflective consciousness. Their agents minimize variational free energy; our framework asks: *Under what energetic conditions can this minimization occur?*

Predictive Power and Clinical Distinction: This distinction has significant implications. Whyte et al.'s model predicts *which* computations generate consciousness. Ours predicts *who can afford them* under *what conditions*. This explains clinical patterns where computational capacity exists but cannot be deployed under metabolic stress—a phenomenon their framework cannot address. A patient may have an intact computational architecture but insufficient metabolic capacity, explaining why insight-oriented therapies often fail for metabolically constrained patients.

The Critical Test: The key distinction is testable. If metabolic load (e.g., hypoglycemia) selectively impairs the computations Whyte et al. identify as constitutive of consciousness (e.g., counterfactual policy selection), and this impairment is rescued by co-regulation (e.g., partner presence), then metabolism is not merely a constraint on computation—it is a constitutive feature of the system. Our Prediction 1 (Section **??**) operationalizes this test. Positive results would demonstrate that computational accounts like Whyte et al.'s, while necessary, are incomplete without the metabolic grounding we provide.

4 4. Dysregulation as Pathological Resonance: Rigid Attractors

If the coherent self is an emergent property of a refined, low-cost predictive system, psychopathology can be reframed as a failure of that system's dynamics. We model this as a collapse into **rigid attractor states**: self-reinforcing prediction patterns that are maladaptive but offer a form of local, short-term metabolic economy at the cost of global, long-term dysregulation and allostatic load **?**. This aligns with contemporary quantitative nosology efforts like the Hierarchical Taxonomy of Psychopathology (HiTOP), which organizes mental illness along dimensional spectra rather than discrete categories, revealing broad underlying factors rooted in distress and dysregulation **??**.

4.1 4.1 The "Cheap but Exhausting" Paradox

Pathological states (PTSD hypervigilance, depressive rumination) are experienced as exhausting. How can they be simultaneously low-cost and draining?

Jamadar et al. **?** show goal-directed cognition costs only 5% more than rest, yet operates within narrow homeostatic bounds. The resolution distinguishes **Cortical Glucose Metabolism** (supporting reflective consciousness) from **Systemic Metabolic Burn** (depleting the organism):

- **Adaptive Regulation (Cortical Cost):** Flexible prior updating and emotional regulation consume cortical glucose.
- **Dysregulation (Systemic Cost):** Rigid priors trigger chronic stress, mobilizing HPA axis and visceral systems **?**.

Rigid priors are computationally simple (avoiding uncertainty cost) but energetically expensive (maintaining chronic threat state). The system is computationally frozen but metabolically burning. This explains why insight alone fails: knowing safety doesn't reduce metabolic load when prior precision prevents model updating.

Not all interpersonal coupling is adaptive. Reed et al. **?** distinguish *co-regulation*—where partners' coupled emotions show dampening patterns and return to homeostatic levels—from *co-dysregulation*—where bidirectional coupling amplifies emotional states in a runaway escalation.

This distinction maps directly onto our framework: co-regulation corresponds to the adaptive R1 loop (reducing metabolic load through relational subsidy), while co-dysregulation corresponds to the pathological R3 loop (amplifying prediction error and metabolic cost). Co-dysregulation depletes regulatory resources rather than restoring them, demonstrating that relational coupling can be metabolically exhausting when it fails to dampen arousal. This explains why some relationships are experienced as draining rather than restorative—they provide the computational structure of co-regulation without the metabolic benefit.

Fragmentation (dissociation, DID) adaptively partitions high-error traumatic data. Maintaining separate partitions ("Going on with Normal Life" vs. "Trauma Part") costs less than maintaining one integrated high-conflict narrative. Integration should not be forced prematurely; building regulatory capacity must precede integration.

4.2 4.2 Model-Discriminating Case: Reactive Attachment Disorder (RAD)

RAD is our strongest discriminator. Defined by the absence of co-regulatory seeking behavior, RAD represents a system that never received the metabolic subsidy required to build social predictive architecture **?**. The child's inhibition is a logical efficiency strategy: avoiding the high cost of social signaling when the historical return on investment is zero. Studies of institutionalized children show that placement in foster care can restore attachment quality when co-regulatory support is introduced **?**.

4.3 4.3 Broader Test Case: Disorganized Attachment

While RAD represents the extreme, disorganized attachment patterns in the general population ($\sim$15% of US samples, higher in clinical populations) provide a more testable case **?**. Disorganized children show contradictory approach-avoidance behaviors toward caregivers, reflecting a system caught between the metabolic need for co-regulation and the prediction that seeking it will increase, rather than reduce, threat. The caregiver becomes simultaneously the source of safety and danger, creating an unsolvable predictive dilemma.

Specific Model Predictions for Disorganized Attachment:

- **Elevated metabolic cost during social interaction:** Unlike secure attachment (where co-regulation reduces cost) or avoidant attachment (where disengagement minimizes cost), disorganized attachment should show *increased* HRV variability and cortisol during caregiver interaction—the system expending energy on an unsolvable approach-avoidance computation.

- **Difficulty maintaining stable self-states under stress:** The contradictory predictive signals prevent settling into a stable regulatory strategy, predicting higher state instability (measurable via experience sampling).
- **Longitudinal trajectory toward dissociation:** The pattern predicts elevated rates of dissociation and emotional dysregulation in adulthood **?**, as the system learns to partition rather than integrate contradictory predictions.

Unlike RAD's absence of seeking, disorganized attachment represents a system overwhelmed by contradictory predictive signals. This makes it a powerful test case: the model predicts not merely that disorganized attachment is "bad" but that it imposes *specific metabolic signatures* distinguishable from other insecure patterns.

4.4 4.4 Integrative Failure: BPD and DID

Adult pathology represents the maintenance of "cheap" but maladaptive attractors. **These conditions are used here as model-stress tests rather than definitive etiological claims:**

Borderline (Oscillating Attractor): The system oscillates between idealization and devaluation to avoid the computational cost of integrating complex, ambivalent data **?**. It is metabolically expensive to the body (high arousal) but computationally simple for the model.

Dissociation (Partitioning): DID represents adaptive partitioning **?**. When the metabolic cost of integrating traumatic error exceeds total capacity, the system fragments. Maintaining two simple, non-conflicting models ("Going on with Normal Life" vs. "Trauma Part") is energetically cheaper than maintaining one high-conflict integrated narrative.[8]

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[8] The etiology and diagnosis of DID remain debated **?**. BPD diagnosis carries stigma and heterogeneity concerns. We cite these conditions as phenomena that become more legible within a relational framework. RAD, with explicit etiological criteria, provides the stronger test case.

5 5. Implications and Discussion

5.1 5.1 Clinical Implications

The dominant model of psychotherapy—one hour per week of individual processing—assumes the patient has the "somatic budget" to carry the work into the other 167 hours. For patients with severe dysregulation (RAD, BPD), this assumption may be false. Indeed, one-hour weekly therapy for severely dysregulated patients may be pharmacokinetically absurd—like administering a drug with a half-life of minutes and expecting week-long effects. If co-regulation provides metabolic subsidization, its "dose" must match the patient's regulatory decay rate.

Therapist as Metabolic Auxiliary: The therapist does not just teach skills; they lend their nervous system to the patient, temporarily lowering the somatic cost of processing affects that would otherwise be disintegrating **??**. This reframes the therapeutic relationship not as a context for insight delivery, but as a metabolic intervention. The therapist's regulated presence allows the patient's system to temporarily operate in a lower-cost regime, making previously inaccessible affects and memories available for processing.

Mechanism Clarification: The therapist provides *both* direct regulation and scaffolding for self-regulation development.

Acute phase: The therapist's nervous system provides real-time co-regulatory subsidization. This reduces the patient's metabolic load through physiological synchrony, predictable responsiveness, and containment of affective states.

Developmental phase: Repeated co-regulatory experience builds the patient's own capacity. Internalized working models of the therapist become available as "offline" co-regulation, reducing dependence on physical presence. The goal is graduated transfer—scaffolding is progressively withdrawn as the patient's architecture develops. This maps onto the rupture-repair loop (R2): each successful repair strengthens the patient's model of relational reliability, reducing future metabolic cost.

This has implications for treatment intensity and structure. If the core mechanism is metabolic subsidization, then:

- **Frequency matters:** Brief, frequent contact may be more effective than longer, less frequent sessions for severely dysregulated patients. The system needs consistent access to co-regulation, not periodic intensive doses.

- **The therapeutic frame is somatic:** Consistency, predictability, and reliability of the therapist are not merely "holding" in a metaphorical sense—they literally reduce the patient's predictive uncertainty and thus their metabolic load.
- **Insight is downstream:** Traditional insight-oriented interpretation may be ineffective or even harmful if delivered when the patient's metabolic load is too high for reflective processing. Timing matters: insight can only be metabolized when the system has sufficient regulatory capacity.

Clinical Translation Guidelines:

Assessment domains: Metabolic capacity assessment requires evaluating three domains: (1) history of co-regulatory access (attachment history, current support network); (2) current allostatic load (sleep quality, stress markers, somatic complaints); (3) baseline reflective function (RFQ-8; mentalization under stress).

Intervention calibration: Low-capacity patients require co-regulation building before content work (longer sessions, increased frequency, relational focus). Medium-capacity patients tolerate reflective work with metabolic pacing (alternating challenging material with regulatory moments). High-capacity patients tolerate standard interventions with depletion monitoring.

Session-level indicators: Signs of metabolic depletion include cognitive narrowing, affective flooding, dissociative markers, and loss of mentalizing. Co-regulatory moments (attunement, validation, pacing) function as metabolic "recharge." Sessions should end with regulation rather than activation.

Treatment planning: Consider higher frequency early in treatment (metabolic momentum), shorter but more frequent sessions for high-need patients, and combined treatments (medication stabilizes baseline capacity, enabling therapeutic work).

Ecological Limitation: Because the brain tracks context of safety, a patient may be competent in clinic but dysregulated at home. The therapeutic gains are real but context-specific. This explains the common clinical frustration of patients who "know what to do" in session but cannot access that knowledge in their daily environment. The problem is not cognitive—it's metabolic. The home environment may impose a somatic cost that precludes access to the reflective capacities developed in therapy.

This suggests that interventions must target the patient's ecological relational web (partners, family, community) to create lasting change. Individual therapy may be necessary to build capacity, but it may be insufficient without parallel work to reduce the metabolic load imposed by the patient's relational environment.

5.2 5.2 Systemic Implications: The Public Health Parallel

Resilience as Resource Hoarding: This framework necessitates a re-evaluation of "resilience." If regulation is a metabolic transfer, then resilience is not a character virtue but a measure of relational wealth. Expecting marginalized populations to demonstrate "grit" without providing the metabolic subsidy of safe co-regulation may systematically underestimate the metabolic prerequisites of the capacities being demanded. Public health strategies that focus on "teaching coping skills" to individuals in relationally toxic environments may implicitly assume metabolic resources that structural conditions fail to provide. Meta-analytic evidence shows social relationships affect mortality comparably to smoking **?**. The Adverse Childhood Experiences (ACE) Study demonstrated a powerful dose-response relationship between early relational trauma and numerous negative health outcomes in adulthood, establishing a clear public health rationale for systemic intervention **?**. Recent evidence confirms the protective mechanism: Brendgen et al. **?** find that adolescents with high social support show 40% lower cortisol reactivity following peer victimization compared to low-support peers—direct evidence that relational wealth provides metabolic buffering against environmental stressors.

The propagation of dysregulation follows an epidemiological logic: a dysregulated parent is more likely to raise a dysregulated child, who carries that pattern into their own relationships. Parents' unresolved traumatic experiences are directly related to infant disorganized attachment status **?**. In this sense—and only this sense—dysregulation spreads like a contagion.

But the analogy must not be overextended. Dysregulation is not an external pathogen that invades an otherwise healthy host. It is a collapse of the system's own dynamics—a fall into a rigid attractor state that is locally stable but globally maladaptive. The "germ theory" framing applies to *transmission infrastructure* (how relational poverty propagates across generations), not to the nature of the pathology itself (which is a dynamic systems failure).

This distinction matters for intervention. Germ theory inspired sanitation and vaccination—systemic interventions that prevented transmission. A relational public health approach would similarly target the infrastructure: building co-regulatory capacity at family, school, and community levels, rather than treating casualties one by one.

5.3 5.3 Individual Differences and the Constraint Model

A critical challenge to any relational model is the robust literature on individual differences. The model must account for heritability, temperament, and resilience without abandoning its central claim. We propose an interactionist model where the metabolic constraint operates on a biologically variable substrate.

Heritability and Temperament: Twin studies show substantial heritability for traits related to regulation, such as neuroticism ($\sim$40–48%) and behavioral inhibition ($\sim$50%) **??**. Furthermore, temperamental differences are observable from birth, preceding extensive co-regulatory experience. We interpret these findings not as a refutation of the relational constraint, but as evidence for individual variation in the *baseline metabolic cost of regulation*. Some nervous systems may simply be more metabolically efficient at self-regulation, requiring less external scaffolding to maintain coherence. This can be formalized in our computational sketch as variation in the baseline capacity parameters.

Heritability and Threshold Effects: Twin studies demonstrate substantial heritability ($\sim$50%) for self-regulation traits **?**, which cannot be dismissed as mere parameter variation. We propose a mechanistic interpretation: genetic variance (likely polygenic, involving receptor density, synaptic pruning, structural connectivity) functionally aggregates into differences in baseline metabolic cost of maintaining predictive precision. This serves as a final common pathway through which heritable variation influences regulatory capacity. High-efficiency individuals may sustain reflective consciousness with minimal co-regulatory scaffolding, explaining resilience despite relational adversity. Critically, the model predicts threshold effects: co-regulation becomes necessary when $(\textit{Stress} \times \textit{Baseline Inefficiency})$ exceeds regulatory capacity C. This generates testable hypotheses: neuroimaging studies comparing high- versus low-resilience individuals under controlled relational deprivation should reveal differential metabolic costs (PET/fMRI) and earlier DMN degradation in low-efficiency individuals.

Differential Susceptibility: The differential susceptibility framework provides a powerful mechanism for this interaction **??**. So-called "orchid" individuals, who show greater sensitivity to both positive and negative environments, can be understood in our model as having higher variance in their somatic cost depending on the relational context. In a supportive, co-regulatory environment, their metabolic cost of regulation plummets, allowing them to flourish ("vantage sensitivity" **?**). In a harsh, unsupportive environment, their costs skyrocket, leading to dysregulation. "Dandelion" individuals, conversely, have a more stable (though perhaps less optimized) baseline cost across different environments.

Resilience: The phenomenon of resilience—the capacity to thrive despite adversity—does not refute the model but refines it. Resilient individuals are not those who lack a need for co-regulation, but those who are successful at finding it. Protective factors are almost invariably relational: at least one stable adult relationship, community resources, or alternative attachment figures **?**. Resilience, in this view, is the successful navigation of the social world to meet a fundamental metabolic need for relational scaffolding.

5.4 5.4 Cultural Validity and Scope

These frameworks derive primarily from WEIRD populations **?**, but the metabolic constraint applies universally—the crucial factor is coherence of regulatory signals within a cultural context, not conformity to Western "secure" patterns **??**. Cultural neuroscience has demonstrated that neural processes underlying self-construal, emotion regulation, and social cognition differ systematically between individualist and collectivist cultures **??**, suggesting that the *expression* of co-regulatory mechanisms may vary while the underlying metabolic constraint remains universal. Our "dyad" language is heuristic; the core principle is stable relational scaffolding, whether from distributed alloparenting **??** or dyadic care.

Cross-Cultural Predictions: (1) *Distributed nexus efficiency*: Cultures with robust alloparenting should show lower β thresholds and reduced attachment-related pathology. (2) *Collectivist buffering*: Social isolation should produce larger stress responses in collectivist vs. individualist samples, as interdependent self-models incorporate more relational content. (3) *Cultural coherence*: "Insecure" attachment patterns may be metabolically neutral when culturally normative—physiological correlates (elevated cortisol, reduced HRV) should be attenuated in coherent contexts.

Proposed Cross-Cultural Validation Studies: To move beyond WEIRD-centric findings, we propose three research directions:

1. Collectivist vs. Individualist Comparison: Compare metabolic efficiency of distributed nexus (extended family, community networks) versus dyadic focus. **Design:** Recruit matched samples from high-collectivism (Japan, Mexico, Nigeria) and high-individualism (US, UK, Germany) cultural contexts. Measure cortisol reactivity, HRV recovery, and DMN connectivity during stress tasks under three conditions: alone, with primary attachment figure, and with extended network members. **Prediction:** Distributed networks will show greater metabolic buffering (faster cortisol recovery, sustained HRV) under stress, particularly in collectivist samples where network-level scaffolding is culturally normative.

2. Indigenous Relational Ontologies: Many Indigenous cultures explicitly conceptualize selfhood as relational rather than individual **?**. **Design:** In collaboration with Indigenous researchers, develop culturally appropriate protocols testing whether explicit relational ontologies predict different metabolic baselines. Compare physiological markers during self-referential tasks framed individualistically ("I am...") versus relationally ("I am part of..."). **Prediction:** Relational framing will reduce metabolic cost (lower mPFC activation) in cultures with strong relational ontologies, while individualistic framing may paradoxically increase load.

3. Measurement Invariance: Before cross-cultural comparison, validate that core constructs (reflective function, attachment security) exhibit measurement invariance across populations. **Protocol:** Conduct multi-group confirmatory factor analyses on RFQ and ECR-R across at least five cultural contexts, testing configural, metric, and scalar invariance. Address emic versus etic concerns by including culture-specific items capturing local expressions of reflective function (e.g., ancestor connection, community embeddedness).

5.5 5.5 Falsifiable Predictions

This computational framing generates testable predictions distinguishable from individualistic models. We lead with our strongest, most concrete prediction:

Prediction 1: Metabolic Threshold Manipulation (Core Test)

Inducing high metabolic load (via hypoglycemia or cognitive fatigue) in healthy individuals will temporarily impair **higher-order mentalization** (e.g., performance on the "Reading the Mind in the Eyes" test or False Belief tasks) but **NOT** basic cognitive controls (e.g., Stroop tasks). This specificity is crucial, as it differentiates the Metabolic Constraint model from a general "fatigue" or "I'm tired so I'm dumb" model. Recent ecological momentary assessment data from Type 1 Diabetes patients provides preliminary support: Hawks et al. **?** found that glucose fluctuations impaired processing speed (which underlies reflective function) but *not* sustained attention—precisely the dissociation our model predicts. The prediction is that social/reflective cognition is the first luxury item cut from the metabolic budget, and this impairment will be *mitigated* by the presence of a safe attachment figure (e.g., holding hands) but *not* by stranger presence **?**. Critically, we predict the stranger condition will be equivalent to or *worse* than alone—unpredictable social partners generate prediction error (metabolic cost) without the subsidization that comes from established relational predictability. Critically, this dissociation distinguishes metabolic constraint from motivational accounts: if co-regulation worked solely via subjective effort reduction, we would expect uniform improvement across task types; selective restoration of mentalization while basic control remains unaffected is uniquely predicted by the metabolic model.

Experimental Design: A 3×2 factorial design crosses metabolic state (hypoglycemia via glucose clamp at 3.0 mmol/L, or cognitive depletion via extended Stroop) with social condition (partner holding hands, stranger holding hands, alone). Participants (N=120, stratified by attachment status) complete mentalization measures (Reading the Mind in the Eyes Test, False Belief tasks) and control measures (Stroop, Digit Span) at baseline and post-manipulation, with continuous HRV and cortisol monitoring.

Critical Predictions: (1) Metabolic load impairs mentalization but NOT control tasks—this dissociation distinguishes our model from general fatigue accounts. (2) Partner presence attenuates mentalization impairment specifically, with no effect on control tasks—this specificity rules out mood enhancement and arousal reduction accounts. (3) Stranger presence provides no benefit (and may impose additional cost) compared to alone—this distinguishes metabolic co-regulation from mere social presence effects. (4) Securely attached participants show stronger rescue effects than insecurely attached participants.

Power Analysis: Based on Hawks et al. **?** ($d \approx 0.35$ for glucose-cognition) and Coan et al. **?** ($d \approx 0.60$ for partner effects), we estimate $d = 0.40$ for the critical interaction. N=80 provides 0.80 power for the primary hypothesis; N=120 accommodates attachment moderation analyses and protocol attrition. Given the multi-task design (mentalization vs. control measures across three social conditions), we recommend pre-registration with explicit correction for multiple comparisons (e.g., Bonferroni-Holm or false discovery rate control) to protect against inflated Type I error while preserving power for the theoretically critical interaction.

Prediction 2: Rupture-Repair Metrics

Therapeutic success will be predicted by the frequency and resolution of physiological asynchrony events **?**.

- **Measure:** Simultaneous dual-ECG.
- **Marker:** Skin Conductance Response (SCR) desynchrony $>$ 1.5 SD from baseline (rupture), followed by RSA re-alignment within 120s (repair).

Prediction 3: Metabolic Cost of Rigidity

High-rigidity systems (Personality Disorders) will show higher glucose consumption to process social prediction errors.

- **Measure:** FDG-PET or fNIRS.
- **Protocol:** Subjects view videos of trusted figures behaving unexpectedly. We predict higher metabolic burn in the mPFC and ACC (error monitoring regions) in patients vs. controls, which fails to normalize even when a safe figure is physically present.

Prediction 4: Computational Phenotyping

Parameters from this active inference model (e.g., prior precision, learning rate) derived from behavioral tasks can serve as biomarkers. We predict that individuals with RAD or BPD will exhibit abnormally high precision on negative social priors and a reduced learning rate in response to positive prediction errors.

Prediction 5: Gene Expression

Expression of genes related to synaptic plasticity (e.g., BDNF) and immune-related gene expression in adulthood will be modulated by current relational quality, independent of early childhood history, supporting the “metabolic maintenance” view **????**.

Prediction 6: Critical Threshold Effects

The computational implementation (Section **??**) reveals that co-regulatory support should exhibit *threshold effects* rather than linear dose-response. The critical threshold is context-dependent, varying with stress load and individual recovery capacity; under moderate conditions, it falls in the range $\beta \approx 0.15$–0.25. Crucially, sensitivity analysis reveals **hysteresis**: starting from a stressed state requires more co-regulatory support to recover than would have been needed to maintain health from a healthy start. This path-dependence formalizes why prevention works better than treatment, why early intervention matters, and why people get "stuck"—the basin of attraction for the collapsed state is stickier than the healthy one.

Prediction 7: Intervention Synergy

Combined pharmacological and psychotherapeutic interventions should show *super-additive* effects on reflective capacity. Specifically, the model predicts that medication primarily boosts regulatory capacity (C) while therapy primarily reduces rigidity (R); combined treatment creates metabolic headroom that amplifies therapy's effect on rigidity reduction. Reanalysis of existing RCT data should show: (Medication + Therapy) $>$ (Medication) + (Therapy).

Prediction 8: Caregiver Depletion Dynamics

Sustained asymmetric co-regulatory relationships predict gradual erosion of the supporter's regulatory capacity over time, following a predictable trajectory with an initial plateau followed by accelerating decline. Longitudinal studies of caregivers (dementia patients, children with severe behavioral needs) should reveal this specific trajectory shape, not merely "caregivers get tired."

Explicit Disconfirming Case: If individuals with chronically low co-regulatory access (measured via stable attachment assessments and social network analysis) nonetheless show preserved mentalization capacity under metabolic stress (hypoglycemia, cognitive fatigue) **without compensatory physiological cost** (no elevated cortisol, no reduced HRV, no increased neural activation in threat-monitoring regions) **and without evidence of compensatory mechanisms** (robust internalized scaffolding, symbolic attachment maintenance, or high intrinsic regulatory efficiency), this framework would be falsified. The claim is not merely that co-regulation is helpful, but that it is the metabolically optimal configuration—and that deviations from this baseline impose measurable costs or require identifiable compensatory mechanisms.

A Formal Distinction—Optimal vs. Helpful: If co-regulation were merely *helpful*, we would expect dose-response linearity extending down to zero support—solo-regulation would simply be less efficient, not impossible. Our model predicts something stronger: asymptotic collapse as $\beta \to 0$ unless compensatory mechanisms are present. Below the critical threshold, reflective capacity does not merely decline; it undergoes qualitative phase transition into rigid attractor states. This is empirically distinguishable: linear models predict graded impairment; our model predicts threshold effects with hysteresis. The distinction matters clinically: if co-regulation is merely helpful, skill-building interventions should work in isolation; if it represents the metabolically optimal configuration, interventions below threshold will fail unless they build compensatory capacity (internalized scaffolding) or reduce effective stress load.

Surrender Condition: If future data consistently reject threshold non-linearity—if well-powered studies demonstrate that the relationship between co-regulatory support and reflective capacity is linear down to $\beta = 0$ with no critical threshold ($\beta_{\text{crit}} \leq 0$)—the optimality claim is withdrawn. Specifically, we will consider the threshold claim unsupported if two independent, adequately powered laboratories (N≥80 each) fail to detect the predicted β_{crit} interaction ($p > 0.05$, Bayes Factor < 0.33). The model would revert to a weaker "efficiency" framing: co-regulation as helpful optimization rather than metabolically optimal baseline. We stake the core theoretical contribution on threshold existence.

Cross-Cultural Validation Imperative: These predictions derive primarily from WEIRD (Western, Educated, Industrialized, Rich, Democratic) populations. Before claiming generalizability, each prediction requires replication across non-WEIRD contexts. We specifically call for testing in (1) collectivist cultures where distributed caregiving is normative, (2) cultures with different attachment norms (e.g., where avoidant patterns may be adaptive), and (3) populations with different metabolic baselines (e.g., varying nutritional contexts). The model predicts that the *mechanism* (metabolic constraint) is universal, but the *expression* (who provides co-regulation, optimal configurations) may vary substantially. Cross-cultural failures of specific predictions would refine rather than falsify the framework, unless the core metabolic mechanism itself fails to replicate.

If these predictions fail, the model is falsified. If they succeed, methodological individualism becomes increasingly untenable.

5.6 5.6 Recent Empirical Support: The Science is Catching Up

Converging empirical findings from 2024–2025 provide striking quantitative support for the model's predictions.

Theoretical Convergence: Three independent theoretical developments align with our framework. Tottenham and Vannucci **?** integrate attachment theory with predictive processing, modeling attachment schemas as predictive models—precisely our computational formalization. Allsop and Tye **?** propose "social homeostasis" as a mental health paradigm, arguing the brain regulates social connection as a homeostatic variable. Lee and Williams **?** identify prefrontal circuitry maintaining social homeostasis.

Physiological Parameters: Recent HRV studies provide quantitative calibration. Loneliness predicts reduced RMSSD reactivity during social stress tasks **?**. Mother-adolescent HRV synchrony predicts relationship quality ($R^2 = .72$), with BPD dyads showing reduced synchrony compared to controls **?**. Critically, partner-specific stress buffering during TSST shows a 9 bpm difference in peak heart rate between partner support (94.2 bpm) and stranger support (103.3 bpm) **?**—quantifying the metabolic subsidy our model predicts.

Molecular Evidence: BDNF methylation correlates with mentalizing accuracy ($\beta = -.34$) **?**, with early stress predicting methylation patterns that link to inflammatory markers **?**. Parent-child cortisol synchrony is moderated by parenting style **?**. Social support moderates victimization-cortisol links, with high-support adolescents showing 40% lower cortisol reactivity **?**.

These findings, emerging independently, suggest the metabolic-relational framework is empirically tractable. The convergence is notable: these researchers were not testing our model but arrived at compatible conclusions through independent investigation.

Acknowledging Mixed Evidence: The evidence is not uniformly supportive. Some studies report individuals who maintain stable functioning in isolation without marked physiological elevation **?**. The resilience literature documents "invulnerable" children and adults who appear to thrive despite relational adversity. We interpret these cases as requiring one of three explanations within our framework: (1) undetected symbolic or internalized co-regulatory resources, (2) individual differences in metabolic efficiency parameters (γ, α), or (3) measurement limitations in capturing subtle but real metabolic costs. The third possibility—that some individuals genuinely operate at low metabolic cost without co-regulatory support—would constitute falsifying evidence if robustly documented with the compensatory mechanisms ruled out (see Section 5.7). The model's credibility depends on whether its predictions distinguish these possibilities empirically.

5.7 5.7 Limitations

While we have implemented the model as a system dynamics simulation demonstrating mathematical coherence (Section **??**), parameters are not yet empirically calibrated. Future work must move from this conceptual model to formal, data-driven computational psychiatry. **Concrete paths to empirical calibration include:** (1) *Dyadic fMRI paradigms* extending Coan's hand-holding design **?** to directly measure metabolic cost (FDG-PET or calibrated BOLD) during co-regulated versus isolated conditions, enabling estimation of the β parameter; (2) *Ecological Momentary Assessment (EMA)* capturing real-time co-regulatory access, momentary reflective capacity (brief mentalization tasks), and physiological markers (wearable HRV) across weeks, permitting within-person estimation of threshold dynamics; (3) *Hyperscanning* (simultaneous dual-fMRI or dual-EEG) during therapeutic interactions to capture rupture-repair dynamics in real time; and (4) *Longitudinal caregiver studies* tracking regulatory capacity erosion to calibrate the caregiver depletion trajectory parameters. Hierarchical Bayesian approaches applied to such datasets would enable computational phenotyping of individual regulatory profiles while testing the model's structural predictions.

The WEIRD Problem: The framework draws primarily on WEIRD populations **?**. Cross-cultural validation is essential. Mesman et al. **?** identify both universal features (secure base behavior across cultures) and culture-specific expressions. In collectivist cultures, the "relational nexus" may be distributed across family and community rather than concentrated in a primary dyad **?**. Critically, our model *predicts* distributed caregiving may be more metabolically efficient—a testable cross-cultural hypothesis. We invite researchers to test whether "insecure" attachment patterns function as metabolically efficient adaptations in non-WEIRD contexts.

Positive Solitude: Rodriguez et al. **?** demonstrate that beliefs about being alone mediate isolation-wellbeing relationships. This refines rather than refutes our model: internalized relational schemas may provide "offline" co-regulation. Individuals thriving in solitude may draw on rich internalized attachment representations. The model predicts even positive solitude carries higher metabolic cost than equivalent states with physical co-regulation—a testable hypothesis. Bales et al. **?** propose individual differences in social homeostatic "set points"; we accommodate this through parameter variation (high γ, low α), which represents variation *within* the metabolic constraint, not evidence against it.

Extreme Isolates: Contemplatives, astronauts, polar explorers, and solitary confinement survivors could test boundary conditions. We predict these individuals will show *both* (a) maintained symbolic co-regulatory connections (mission control, prayer, internalized attachment figures) *and* (b) elevated metabolic cost relative to socially embedded controls (stress markers, accelerated aging, cognitive changes). High-efficiency outliers who show neither (a) nor (b)—true solo-regulators with low metabolic cost—would falsify the model, but require documentation of parameter configurations (γ, α) that distinguish them from the population. Preliminary evidence supports our prediction: astronauts maintain extensive mission control communication *and* show physiological changes; contemplatives report rich internal relational imagery; solitary confinement produces well-documented deterioration even when inmates report adaptation.

Evolutionary Claims: The metabolic constraint defines the constraint space within which selection operated, not why reflective consciousness evolved. Whatever selective advantages drove complex prediction had to exceed metabolic overhead. The model's power lies in generating falsifiable metabolic predictions, not settling evolutionary debates.

Methodological Considerations: HRV, skin conductance, and DMN connectivity show test-retest variability. Multiple comparison concerns must be addressed through correction procedures or pre-registration.

What Would Falsify This Model?

Table **??** specifies evidence that would disconfirm the framework:

The very specificity of the model's predictions provides a clear roadmap for empirical work that can validate, refine, or falsify the framework, moving the debate from philosophical contention to empirical testing.

6 6. Computational Formalization: Topology as Destiny

Overview for Non-Technical Readers: The computational model tracks three interacting quantities: *Metabolic Load (M)*, the energetic demand of maintaining reflective consciousness (increases under stress, decreases with co-regulatory support); *Regulatory Capacity (C)*, the organism's available resources for managing prediction error (depleted by sustained load, restored through rest and safe connection); and *Co-regulatory Access (A)*, the availability and quality of relational support (increases with social engagement, decreases when prediction errors signal threat). The core insight is that when M exceeds C, reflective consciousness collapses; co-regulation (A) provides an external pathway to reduce M, making sustained reflection metabolically affordable. The model demonstrates this as a structural property—not dependent on specific parameter values—explaining why isolation produces predictable degradation of higher-order cognition.

The Causal Loop Diagram (CLD) in Figure **??** demonstrates that if the metabolic cost of meaning-making (M) exceeds the standalone capacity (C) of the organism, then the system must close the loop through an external regulator (A) to avoid collapse.

6.1 6.1 The Topological Trap

The structure of these feedback loops (R1, R2, R3) implies that "independence" is mathematically unstable for high-order prediction.

R1 (The Co-Regulatory Loop) is the only loop that adds capacity (C) faster than meaning-making consumes energy (M).

R3 (The Dysregulatory Loop) demonstrates that without external input, prediction error (PE) naturally accumulates, driving the system toward the high-entropy attractor of defensiveness/shutdown.

6.2 6.2 Active Inference Sketch: Co-Regulation as Mutual Free Energy Minimization

Organisms minimize free energy (prediction error) **????**. Chronic unresolved prediction error leads to allostatic overload **?**.

1. Co-Regulation as Distributed Prediction:

In active inference, organisms minimize free energy (prediction error). Co-regulation is superior to solo-regulation because Agent B's actions reduce Agent A's prediction error more efficiently than Agent A acting alone. Agent B performs predictive work on Agent A's behalf, lowering total energetic cost. The dyad becomes a joint system minimizing shared free energy.

2. Rupture and Repair as a Generative Process:

Misattunement creates large prediction error (rupture), but successful repair drives model updates that increase sophistication. The cycle builds trust (precision-weighting on partner's actions) and explains why "good enough" caregiving produces more robust outcomes than perfect attunement (no learning signal) or chronic misattunement (only negative errors) **??**.

3. Entrenched Dysregulation as a Pathological Attractor:

Dysregulated agents assign high precision to negative priors ("others are untrustworthy") and low precision to contradicting evidence. Defensive actions confirm each other's negative predictions, creating a self-confirming loop. The system is metabolically exhausted (high prediction error) but computationally frozen (no model updates). This explains the clinical paradox: patients simultaneously exhausted and stuck.

The situation resembles Maxwell's demon in reverse: the demon famously could reduce entropy only by paying a computational cost (Landauer's principle). Here, the patient *lacks* the computational resources to reduce their own prediction error—they cannot "pay" to sort signal from noise. The therapist functions as an external processor, temporarily subsidizing the computational work of model updating. Just as Maxwell's demon required external energy to function, the patient requires external co-regulation to escape the attractor. Relational trauma requires relational repair; individual insight is insufficient for models formed and maintained dyadically.

6.3 6.3 System Dynamics: Three Reinforcing Loops + Balancing Constraint

The model contains four dominant feedback structures:

R1: Co-Regulation Stabilization Loop (Reinforcing, Adaptive)

Co-regulation → ↓Metabolic Load → ↑Reflective Capacity → ↑Relational Trust → ↑Co-regulation

Maintains reflective consciousness at low energetic cost. This is a virtuous reinforcing loop.

R2: Rupture-Repair Learning Loop (Reinforcing, Developmental)

Prediction Error → ↑Repair Events → ↓Prior Rigidity → ↑Learning → ↓Future Prediction Error

Short-term destabilization produces long-term robustness and model sophistication.

R3: Dysregulation/Rigid Attractor Loop (Reinforcing, Pathological)

Metabolic Load → ↓Refl Capacity → ↑Prior Rigidity → Def Action → Pred Error → ↑Metabolic Load

Traps the system in a self-confirming, high-cost attractor. This is a runaway loop unless externally interrupted.

B1: Capacity Constraint Loop (Balancing, Protective)

Metabolic Load → ↑Allostatic Load → ↓Regulatory Capacity → Forces shutdown or fragmentation

Prevents runaway integration and forces protective shutdown when the system is overwhelmed.

6.4 6.4 Causal Loop Diagram

Figure ?? illustrates the complete feedback structure. The diagram shows the four key feedback loops: the adaptive co-regulation loop (R1), the developmental rupture-repair loop (R2), the pathological dysregulation loop (R3), and the capacity constraint (B1). The system has two stable attractors: a low-cost, high-capacity state where R1 dominates, and a high-cost, low-capacity state where R3 dominates.

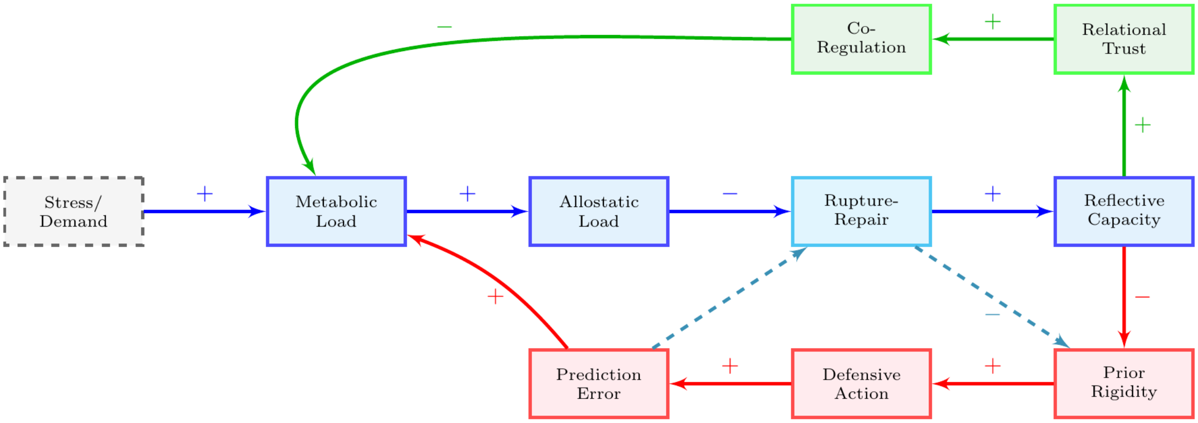

Figure 1: **Causal Loop Diagram showing the four key feedback loops.** Legend: (+) = reinforcing; (-) = balancing; dashed = enabling. Colors: Blue = core system; Red = pathological; Green = adaptive; Cyan = learning; Gray = external. R1 (Green): Reflective Capacity → Relational Trust → Co-Regulation → reduced Metabolic Load (virtuous). R2 (Cyan): Prediction Error → Rupture-Repair → reduced Prior Rigidity (learning). R3 (Red): Reflective Capacity → increased Rigidity → Defensive Action → Prediction Error → increased Metabolic Load (vicious). B1 (Blue): Metabolic Load → Allostatic Load → reduced Regulatory Capacity (constraint).

6.5 6.5 Simplified Dynamics (3 Coupled Equations)

To demonstrate that the verbal theory translates into coherent mathematical dynamics, we offer a **heuristic formalization** of these topological constraints. The following equations are not intended as a quantitative predictive model of real-world phenomena, but as a conceptual tool to demonstrate that the feedback loops described above are sufficient to produce the complex dynamics we claim, such as bi-stability and threshold-driven collapse. **Their purpose is to specify topological dependencies, not to fit empirical data.** This minimalist model serves as a foundation for future work, including a planned extension that incorporates iatrogenic feedback loops from semantic-only regulation (e.g., via LLMs). The simulation shows that the core properties of the theory emerge as structural properties of the feedback architecture itself, contingent on the relational coupling term $\beta(A)$.

Equation 1 (Metabolic Load):

$$\frac{dM}{dt} = \alpha(\text{Stress}) - \beta(A \times C) \tag{1}$$

Equation 2 (Regulatory Capacity):

$$\frac{dC}{dt} = \gamma(C_{\text{max}} - M) - \delta(\text{Trauma}) \tag{2}$$

Equation 3 (Co-regulatory Access):

$$\frac{dA}{dt} = \varepsilon(C) - \zeta(\text{PredictionError}) \tag{3}$$

It is important to note that these equations operate on different time-scales. Co-regulation ($\frac{dA}{dt}$) occurs in milliseconds to seconds (e.g., physiological synchrony), while learning and changes in relational trust happen over days, weeks, and years.

β represents the “Co-regulation Efficiency” parameter.

Reflective consciousness emerges only when $M < C$.

Crucially, if $\beta \to 0$ (isolation), $\frac{dM}{dt}$ remains positive under stress. Metabolic load accumulates until it exceeds capacity ($M > C$), forcing the collapse of the reflective state (RC $\to$ 0). No amount of internal “processing” or “reframing” (parameters within the organism) can arrest this runaway energetic value if the input stress exceeds the isolated system’s dissipation rate.

An Invitation to Computational Modelers: We present this formalization as a heuristic sketch, not a finished model. The equations capture the topological structure we claim is necessary, but we have not specified functional forms, derived parameters from data, or proven existence and uniqueness of solutions. We explicitly invite computational modelers to improve upon, elaborate, or replace this sketch with more rigorous formalizations—whether through Bayesian estimation of parameters from existing dyadic datasets, agent-based implementations, or alternative mathematical frameworks entirely. The core claim (threshold effects, hysteresis, dyadic efficiency) should be derivable from any adequate formalization; if these properties disappear under more rigorous treatment, the verbal theory requires revision.

6.6 6.6 Parameter Estimation Example

To illustrate how β could be empirically estimated, consider Coan's "Hand-Holding" fMRI paradigm **?**. Let E_{threat} be the neural threat response (amygdala/ACC activation):

$$E_{\text{threat}} = \text{Stress} - \beta \times \text{condition} \tag{4}$$

By comparing E_{threat} in Solo (condition = 0) vs. Hand-Holding (condition = 1) trials, β represents the magnitude of neural attenuation provided by the relational nexus. Coan reports effect sizes of $d \approx 0.60$ for partner hand-holding; future work should extract precise activation levels from published data to calibrate this parameter quantitatively.

Roadmap for Empirical Parameter Estimation: Empirical calibration could leverage several existing data sources: (1) β (co-regulation efficiency) could be estimated from physiological synchrony datasets (e.g., parent-infant RSA coupling) by measuring the attenuation of stress responses during co-regulated versus solo conditions; (2) $M_{\text{threshold}}$ (metabolic capacity limit) could be derived from lactate accumulation curves during cognitive load tasks, identifying the inflection point where performance degrades; (3) α (stress input rate) could be calibrated against standardized stress paradigms (TSST) using cortisol/heart rate reactivity; (4) γ (recovery rate) could be estimated from HRV recovery slopes post-stressor. Hierarchical Bayesian approaches would permit individual-level parameter estimation while borrowing strength across populations, enabling computational phenotyping of regulatory profiles.

6.7 6.7 Computational Demonstration: Phase Space Dynamics

The following simulations serve as a computational demonstration of the theory's core tenets. They are not quantitative predictions, but existence proofs that the feedback architecture described is sufficient to generate the claimed dynamics. By implementing the model with plausible, but not empirically fitted, parameters, we show that key phenomena—bi-stability, threshold-driven collapse, and hysteresis—emerge as structural properties of the system. This provides a crucial bridge from verbal theory to a formal dynamic system, laying the groundwork for future, more detailed modeling and empirical validation.

Key Findings from Simulation:

1. **Critical β Threshold:** When co-regulation efficiency (β) drops below a critical threshold, the system cannot maintain reflective consciousness regardless of other parameters. Sensitivity analysis reveals this threshold is *context-dependent*, falling in the range $\beta \approx 0.15$–0.25 (in arbitrary simulation units) under moderate stress conditions (full range: 0.09–0.36 across stress levels; see Section 6.8 for detailed sensitivity analysis). The critical claim is the existence of a non-zero threshold, not its specific numerical value. Crucially, the system exhibits *hysteresis*: recovering from a collapsed state requires more co-regulatory support than maintaining health from a healthy start. This path-dependence formalizes why prevention outperforms treatment and why people get "stuck."
2. **Bi-Stability:** The M-C phase space reveals two stable attractors—a healthy state (low M, high C) and a collapsed state (high M, low C)—separated by the M=C threshold. Trajectories converge to different attractors depending on initial conditions and β.
3. **Intervention Differentiation:** Medication primarily boosts capacity (C) while therapy primarily reduces rigidity (R). Combined treatment shows super-additive effects—medication creates metabolic headroom that amplifies therapy's efficacy. This interaction predicts (Meds + Therapy) $>$ (Meds) + (Therapy), explaining why combination treatment outperforms either modality alone.
4. **Caregiver Burnout Emergence:** Sustained asymmetric co-regulatory relationships show gradual erosion of the supporter's regulatory capacity, an emergent property not explicitly programmed into the model structure.

Figure **??** illustrates these dynamics.

Sensitivity Analysis: Systematic parameter sweeps reveal the robustness and boundary conditions of these findings. We varied β across the full range (0–0.6) and stress level (0.2–0.55) while holding recovery rate ($\gamma = 0.2$) and stress sensitivity ($\alpha = 0.25$) constant. The critical β threshold shows a sharp transition (not gradual degradation) across all tested parameter combinations, confirming that threshold effects are a structural property of the model rather than an artifact of specific parameter choices. The viability boundary in $\beta \times$ stress space reveals an interaction: higher baseline stress requires proportionally higher co-regulatory support to maintain reflective consciousness. Importantly, the sensitivity analysis identifies stress level as the strongest moderator of threshold location—under low stress conditions, even modest co-regulation suffices ($\beta \approx 0.15$), while high stress conditions shift the threshold upward ($\beta \approx 0.25$). This context-dependence is clinically meaningful: the same individual may function adequately with limited support during calm periods but require substantially more co-regulation during crisis.

The model's alignment with established empirical patterns—Still-Face collapse dynamics, social baseline threat attenuation magnitudes, combined treatment synergy—suggests the parameter choices capture biologically realistic constraints, though empirical calibration remains for future work.

Interpretive Note: These simulations demonstrate that the verbal theory yields coherent dynamics (bi-stability, threshold collapse, hysteresis)—not fitted predictions. Readers should treat figures as existence proofs; code is provided for future empirical calibration. Translating β into measurable units: $\beta \approx 0.20$ reproduces Coan's $d = 0.60$ threat-reduction, corresponding to $\sim$9–12 ms RMSSD increase, well within detectability.

6.8 6.8 Sensitivity Analysis: Individual Differences as Features

Key Finding: The critical threshold for co-regulatory support varies substantially with context—a coefficient of variation (CV) of 32.5% across stress conditions. This heterogeneity is clinically meaningful: under low stress (0.2), the critical β threshold is approximately 0.09; under high stress (0.55), it rises to 0.36. The model thus captures real individual and situational differences rather than proposing a single universal threshold.

We conducted systematic sensitivity analysis varying co-regulation efficiency (β: 0–0.6), stress level

(0.2–0.55), recovery rate (γ: 0.1–0.3), and stress sensitivity (α: 0.15–0.35).

Robust Structural Properties: Three features are insensitive to parameter variation and thus constitute genuine structural predictions of the theoretical framework: (1) *Threshold existence*—across all parameter combinations, a critical β threshold exists below which reflective consciousness cannot be maintained (distinguishing this from linear dose-response accounts); (2) *Hysteresis*—$\beta_{\text{recovery}} > \beta_{\text{collapse}}$ across all tested ranges, explaining why prevention is easier than treatment; (3) *Bi-stability*—healthy and collapsed attractors persist across parameter space, emerging from feedback structure.

Context-Dependent Threshold Location: Higher intrinsic recovery (γ) tolerates lower β, providing a computational interpretation of "resilience." Higher stress sensitivity (α) shifts thresholds upward, capturing the "orchid-dandelion" differential susceptibility pattern **???**.

Clinical Implications: The 32.5% CV means clinicians should assess threshold location and current β, not merely ask "does this patient need co-regulation?" The hysteresis finding explains treatment resistance computationally: collapsed attractor states require more intensive intervention than prevention would have needed. Intervention intensity should be calibrated to current stress load, not diagnosis—the same patient may need daily contact during crisis and weekly sessions during stability.

7 7. Conclusion

Reflective consciousness is a high-cost predictive state requiring metabolic subsidization through co-regulation. The dyad is the metabolically efficient baseline; solo-regulation operates at elevated metabolic cost relative to this baseline. Dysregulation and dissociation are rigid attractors entered when integration costs become prohibitive—computationally simple but energetically draining, explaining patients who are simultaneously stuck and exhausted.

The convergent trap: Dynamic systems theory (self is soft-assembled) and social baseline theory (brain assumes partnership) leave little room for the Cartesian individual as the fundamental unit. The autonomous adult runs on internalized relational scaffolding; solo-regulation shares metabolic signatures with chronic threat response.

Implications

This framework suggests that individual-focused interventions, while valuable, address symptoms rather than the metabolic substrate. The therapist functions as a "metabolic auxiliary," temporarily lowering the patient's somatic cost and enabling processing that would otherwise be prohibitive. But because the brain tracks context, therapeutic gains may not transfer to environments that impose high metabolic loads. Lasting change may require interventions at the ecological level—targeting the relational and environmental conditions that shape the patient's regulatory economy.

More broadly, we must recognize that "resilience" is often a measure of relational wealth, and that populations subjected to systemic stress function under a metabolic tax that limits access to reflective consciousness. A truly preventive approach would target the infrastructure of co-regulation: building capacity in families, schools, and communities rather than treating casualties one by one.

Clarifying the Contribution

The individual claims advanced here are not novel—they are established findings across multiple disciplines. That higher-order consciousness is conditional; that those conditions are relational and metabolic; that collapse under load is predictable; that regulation has energetic costs; that relational scaffolding reduces those costs—these are orthodox scientific claims embedded in control theory, cybernetics, predictive processing, attachment research, and clinical psychology.

What is novel is their integration. This paper places these findings within a unified framework that makes explicit their shared implications for the conditions under which reflective consciousness emerges, stabilizes, and collapses. The computational formalization (Section **??**) specifies feedback architecture that generates predictions no single existing model produces. The evolutionary claim—that metabolic constraints would generate selection pressure for co-regulatory capacity—is simply another way of stating what we already know: caregiving evolved, play evolved, attachment evolved, group living evolved. We are naming the constraint, not inventing it.

If this framework produces discomfort, it is because taking it seriously has implications: individualism as the default model becomes questionable; some clinical failures stop being mysterious; "resilience" looks ecological rather than trait-based; some social pathologies appear infrastructural rather than moral; and prevention looks relational rather than purely cognitive. These implications may be destabilizing, but they follow directly from the evidence. If they are wrong, the error should be locatable in the chain of reasoning we have made explicit.

Looking Forward

We are not lone minds but nodes in a regulatory network, and our capacity for reflective thought is a relational achievement. Understanding this may open new avenues for prevention, treatment, and the design of social institutions that support human flourishing. The computational formalization offered here—grounded in active inference and system dynamics—provides a framework for empirically testing these claims and refining our understanding of how meaning-making is metabolically constrained and relationally enabled.

Ultimately, this synthesis forces a confrontation with the limits of the medical model. If the mind is a shared metabolic phenomenon, then the "individual" as traditionally conceived may represent an abstraction that obscures the relational substrate of reflective function. To continue treating the solitary brain as the sole unit of analysis in psychiatry is not just a clinical limitation; it is metabolically prohibitive under the load conditions we have specified. We are, quite literally, thinking more efficiently in each other's presence—the metabolic subsidy may be modest in percentage terms (5–10% glucose savings), but it is the difference between sustainable and prohibitive for extended meaning-making under stress.

Future Directions

This framework invites empirical testing from multiple disciplines: metabolic neuroscientists can directly measure glucose consumption during co-regulation versus solo-regulation; computational psychiatrists can rigorously specify and fit the heuristic formalization; clinical researchers can test whether combined interventions show the predicted super-additive effects; and cross-cultural researchers can evaluate whether distributed caregiving systems produce the hypothesized efficiency gains. The goal is empirical resolution—these claims are falsifiable, and we welcome both confirmation and refutation.

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Conflicts of Interest

The author declares none.

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Table 3: Comparison with Parent Frameworks

Framework	**Generates Alone**	**This Model Adds**
Social Baseline Theory	Threat attenuation with partner present; brain expects social resources	Threshold effects on reflective capacity; attractor dynamics explaining “stuckness”; super-additive intervention effects
Active Inference	Prediction error minimization; free energy principle	Metabolic cost as binding constraint; dyad as computational unit; explains *why* some prediction errors cannot be resolved solo
Attachment Theory	Internal working models; developmental trajectories; secure base concept	Energetic unsustainability of solo-regulation under metabolic load; computational formalization of “earned security”
Autonomic Gating (Porges; Thayer)	Threat → vagal withdrawal → cognitive narrowing; well-established empirically	Integration with metabolic constraint; explains *why* autonomic state gates cognition (energetic cost)
Allostatic Regulation	Body budgeting; context-dependent prediction; constructed emotion	Application to reflective consciousness specifically; relational subsidization of meaning-making

Table 4: Falsifiable Predictions—Summary

Prediction	**Measure**	**Distinguishes From**
1. Metabolic Threshold (Core)	Hypoglycemia impairs mentalization; mitigated by partner	Cognitive skill models; fatigue accounts
2. Rupture-Repair	Synchrony/asynchrony patterns predict outcomes	Individual baseline models
3. Cost of Rigidity	fNIRS/PET: Higher cost for unexpected social info	Trait-based models
4. Comp. Phenotyping	Prior precision, learning rate as biomarkers	Symptom-based diagnosis
5. Gene Expression	BDNF modulated by current relational quality	Critical period models
6. Critical Threshold	Context-dependent β threshold (0.15–0.25 moderate stress); hysteresis	Linear dose-response
7. Intervention Synergy	(Meds + Therapy) $>$ Meds + Therapy	Additive treatment models
8. Caregiver Depletion	Plateau-then-decline trajectory in supporters	Simple fatigue models

Table 5: Potential Falsifying Evidence

Finding	**Why It Would Falsify**
High-functioning isolates with low metabolic markers	Individuals with chronically low co-regulatory access showing preserved mentalization *without* elevated cortisol, reduced HRV, increased neural threat activation, *or* identifiable compensatory mechanisms would contradict the optimality claim
Uniform cognitive impairment under metabolic load	If hypoglycemia/depletion impairs basic cognition and mentalization equally (no specificity), the "luxury item" prediction fails
No partner rescue effect	If co-regulatory presence fails to attenuate mentalization impairment under metabolic load, the central mechanism is unsupported
Linear dose-response for co-regulation	If increasing co-regulation shows monotonic benefits without threshold effects, the dynamical systems formalization is unnecessary
Additive (not super-additive) treatment effects	If medication + therapy equals the sum of their individual effects, the model's intervention predictions are wrong

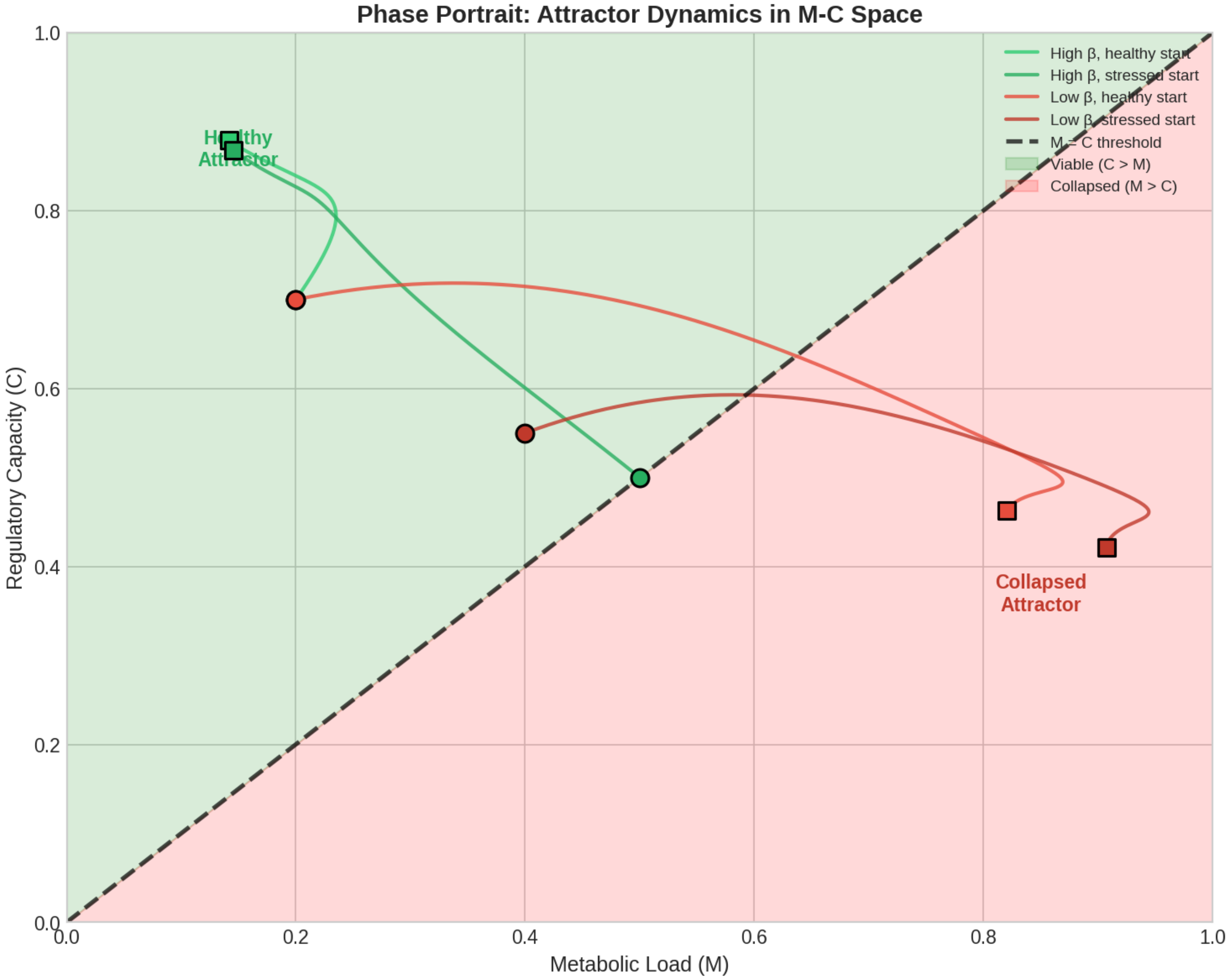

Figure 2: **Phase Space Dynamics and Attractor Basins.** (A) Effect of co-regulation parameter β on system trajectories in M-C space. Green region (C>M): RC viable. Red region (M>C): RC collapsed. Diagonal line: threshold where M=C. Low β (red trajectory) cannot reach viable zone; high β (green) stabilizes in healthy attractor. (B) Resilience to acute stress. With co-regulatory support (green), trajectory loops within viable zone. Without support (red), same stress crosses threshold into collapse. (C) Developmental trajectories. Secure attachment (green) converges to healthy attractor via rupture-repair spirals. Insecure attachment (red) ends in collapsed state.

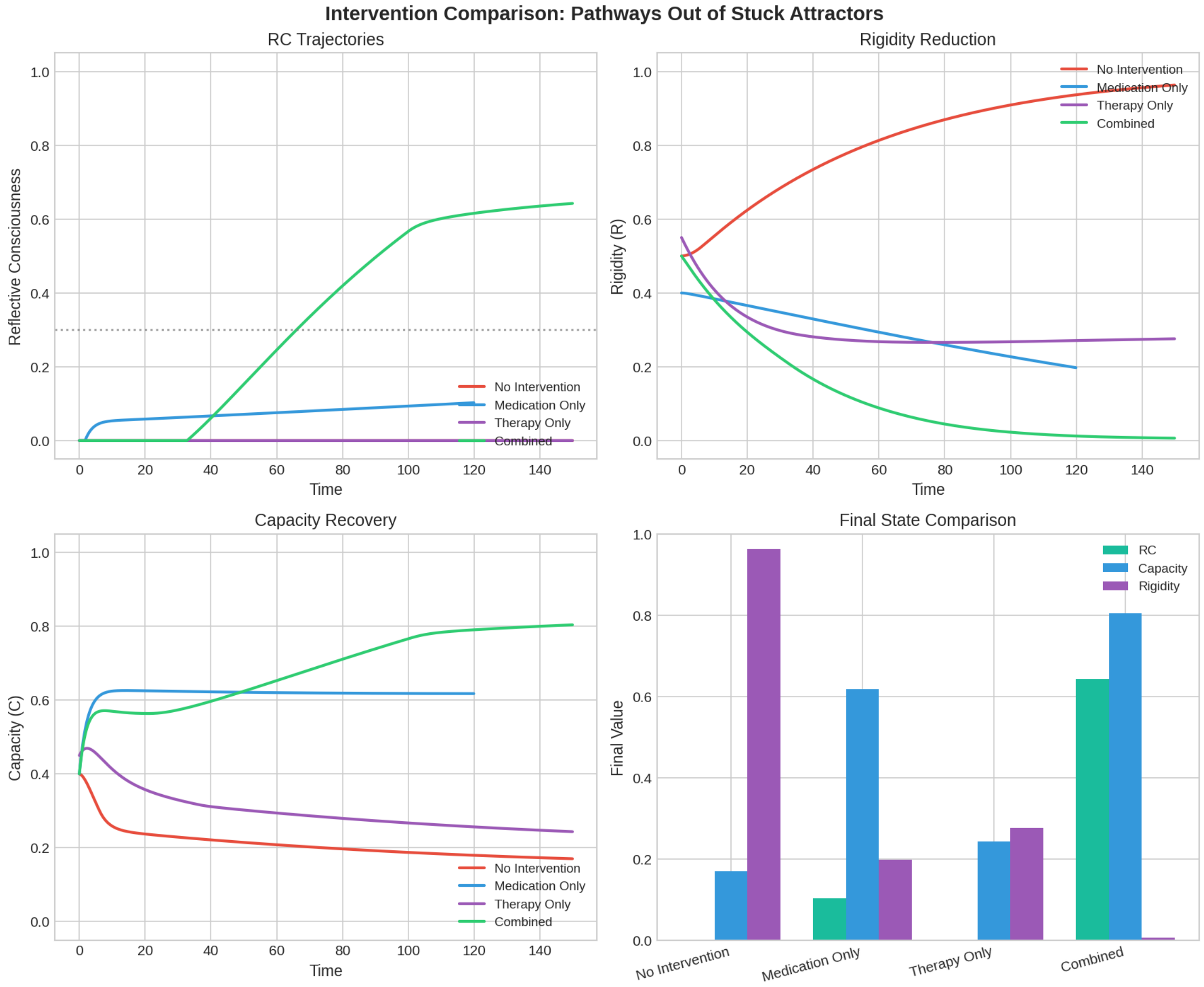

Figure 3: **Intervention Synergy (Prediction 7): Super-Additive Treatment Effects.** This heuristic model illustrates the core qualitative prediction that (Medication + Therapy) > (Medication alone) + (Therapy alone). (Top left) Reflective consciousness trajectories show combined treatment achieves escape from collapsed attractor while single modalities show only modest improvement. (Top right) Rigidity reduction: combined treatment achieves near-zero rigidity while single modalities plateau. (Bottom left) Regulatory capacity recovery: combined treatment reaches healthy baseline while single modalities show limited gains. (Bottom right) **Critical comparison**: Combined treatment substantially exceeds the sum of individual effects, demonstrating qualitative synergy not additive effects. Mechanism: Medication creates metabolic headroom (boosts C), which amplifies therapy's ability to reduce rigidity (R). This super-additive prediction distinguishes the metabolic constraint framework from standard additive treatment models and is empirically testable via reanalysis of existing RCT data. Note: Specific numerical values are illustrative; the structural prediction is qualitative threshold-crossing and synergy, not precise effect sizes.