The Regulation-Gap Model:

A Load-Capacity Framework for Habit Formation, Portfolio Dynamics, and Condition-Dependent Dissolution

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Abstract

Current models of habit formation emphasize automaticity, reinforcement, and context-dependence but lack a unified framework explaining why certain habits resist dissolution, why removing one habit often produces compensatory behaviors, and why habit change systematically fails under chronic stress. This paper introduces the Regulation-Gap Model (RGM), a load-capacity framework proposing that persistent, costly habits function as regulatory solutions to a measurable deficit: the gap between total organismic load and available regulatory capacity.

The model formalizes this through a ten-layer regulation map spanning condition fields through dissolution pathways, identifies six functional regulation channels (shutdown, stimulation, absorption, discharge, closure-simulation, and dissociation/avoidance), and predicts that habit dissolution occurs primarily through capacity restoration and load reduction rather than through motivational or willpower-based interventions. The framework integrates converging evidence from allostatic load research, neurovisceral integration theory, transdiagnostic models of compulsivity, the habit discontinuity hypothesis, and cross-addiction literature.

Four testable predictions with explicit kill criteria are specified, each operationalized through existing physiological measures including heart rate variability, cortisol, actigraphy-derived sleep metrics, and validated behavioral instruments. The model applies most strongly to persistent, costly, self-reinforcing habits and acknowledges clear boundary conditions: it does not claim to explain all habitual behavior, it treats its six channels as functional roles rather than rigid taxonomy, and it positions the regulation gap as a conceptual tool requiring further operationalization. Epistemic status declarations classify each claim by evidentiary tier, separating empirically grounded foundations from literature-consistent theoretical extensions and framework-inferred hypotheses requiring novel empirical testing.

Keywords: habit formation, emotion regulation, allostatic load, self-regulation, habit dissolution, regulatory capacity, condition-dependent behavior, transdiagnostic, portfolio dynamics

1. Introduction: The Habit That Won't Listen to You

A person resolves to stop checking their phone before bed. They understand the consequences, report high motivation, and can articulate the health costs of blue light exposure and fragmented sleep. Within seventy-two hours, the behavior has returned. The standard interpretation attributes this failure to insufficient willpower, inadequate planning, or weak habit-replacement strategies. This paper proposes an alternative: the phone-checking persists because it is performing a regulatory function the person has no other means to fulfill, and no amount of motivation can override a load-bearing behavior until the load it carries has somewhere else to go.

1.1 Contemporary Habit Models: A Critical Review

The prevailing landscape of habit science provides essential but incomplete tools.

Reinforcement Learning Frameworks. RL models emphasize dopaminergic prediction error signals and reward-based learning mechanisms (Schultz, Dayan, & Montague, 1997). While successful in explaining automatic behavior acquisition, they struggle to account for habit discontinuity following environmental change and spontaneous remission from addiction without continued extinction exposure.

Cue-Routine-Reward Models. Wendy Wood's influential body of work established that habits are context-response associations activated automatically by environmental cues, operating independently of current goals and intentions (Wood & Rünger, 2016; Wood, Mazar, & Neal, 2022; Wood, 2024). This framework explains habit persistence through automaticity rather than deliberation: once formed, context directly activates the habitual response in mind without requiring motivational input (Mazar & Wood, 2022). However, this framework cannot explain why habits sometimes form rapidly under acute stress without extensive training, or why deeply entrenched habits may dissolve spontaneously when life circumstances change.

Identity-Based Models. These approaches emphasize self-concept and personal values in driving behavioral persistence (Oyserman, Destin, & Novin, 2012). Yet they

face substantial challenges in accounting for natural recovery from addiction without deliberate identity transformation.

Self-Control and Ego Depletion. The ego depletion framework once promised a resource-based account of self-control failure but has largely collapsed. Two large-scale multilab replications found effects indistinguishable from zero: Hagger et al. (2016) reported d = 0.04 across 23 laboratories (N = 2,141), and Vohs et al. (2021) reported d = 0.06 across 36 laboratories (N = 3,531). The process model of self-control proposed by Inzlicht and Schmeichel (2012) reframed apparent depletion as motivational reallocation rather than resource exhaustion. More recently, Hofmann (2024) argued that self-control research has been excessively individualistic, proposing multilevel analysis incorporating structural and environmental factors. Chater and Loewenstein (2023) formalized this critique, arguing that the "i-frame" (individual-level behavioral interventions) systematically diverts attention from the "s-frame" (structural solutions).

1.2 Four Persistent Anomalies in Habit Research

What existing models do not adequately explain is a set of observations that clinicians and individuals encounter daily:

Habit Discontinuity. Rapid behavioral shifts following environmental transitions — relocation, job change, relationship change — that exceed what cue disruption alone would predict. The habit discontinuity hypothesis demonstrated that disrupting contextual cues during life transitions creates windows for behavior change (Verplanken & Roy, 2016; Thomas, Poortinga, & Sautkina, 2016).

Natural Recovery. Spontaneous remission from addiction without formal treatment. Approximately 70% of persons with AUD improve without formal interventions (Tucker, Chandler, & Witkiewitz, 2020). This is difficult to reconcile with models emphasizing the necessity of structured behavioral intervention.

Behavior Clustering. The simultaneous emergence of multiple compensatory behaviors under stress. Prochaska, Spring, and Nigg (2008) noted that 92% of smokers exhibit at least one additional risk behavior. Health-risk behaviors cluster in ways that isolated habit models cannot explain.

Rapid Dissolution. Sudden loss of craving or urge without prolonged suppression efforts, often reported as the behavior becoming "irrelevant" rather than "resisted."

1.3 The Regulation-Gap Model

The Regulation-Gap Model (RGM) proposed here occupies the space these critiques have opened. Rather than treating habit as primarily a behavior to be replaced, a neural circuit to be retrained, or a willpower challenge to be overcome, the RGM treats persistent costly habits as routing decisions made by a load-bearing organism inside a particular condition field. The model's central claim is that dissolution occurs when the regulation gap closes — when available capacity meets or exceeds total load — not when motivation increases. This reframes the clinical and practical question from "How do I make myself stop?" to "What is this behavior regulating, and how can I close the gap it is filling?"

> *Epistemic status: The preceding reframing is a Tier 2 theoretical extension. The critiques of ego depletion, the limitations of motivation-based models, and the evidence for structural approaches are empirically grounded (Tier 1). The specific claim that the regulation gap is the primary determinant of persistent habit dissolution is a framework-inferred hypothesis (Tier 3) requiring the novel empirical tests specified in Section 6.*

2. Converging Evidence for a Load-Capacity Architecture

The RGM draws on four empirically grounded research programs that independently point toward a load-capacity architecture underlying persistent habitual behavior. None of these programs has previously been integrated into a single formal framework.

2.1 Allostatic Load as Cumulative Regulatory Burden

Bruce McEwen's allostatic load (AL) model provides the physiological foundation for the "load" component of the RGM. Allostatic load represents cumulative physiological wear resulting from chronic stress and the repeated cycling of adaptive systems (McEwen & Stellar, 1993). As documented in a systematic review by Guidi, Lucente, Sonino, and Fava (2021), AL is measured through composite biomarker indices spanning neuroendocrine (cortisol, DHEA-S, catecholamines), cardiovascular (blood pressure, resting heart rate), metabolic (waist-hip ratio, cholesterol, glycosylated hemoglobin), and immune (C-reactive protein, IL-6) systems.

Critically for the present model, Juster and Misiak (2023) noted that the AL framework has expanded to account for how chronic stress is "bi-directionally synergized by health-related behaviors." Behavioral patterns both contribute to and result from allostatic load, creating feedback loops the RGM seeks to formalize.

Bobba-Alves, Juster, and Picard (2022) proposed reframing allostatic load in energetic terms: mitochondrial energy production capacity modulates behavioral and psychological states. Under energy constraints, organisms shift toward less effortful behavioral patterns — a prediction directly compatible with the RGM's claim that high load favors low-friction regulatory channels.

2.2 Heart Rate Variability as an Index of Regulatory Capacity

The "capacity" side of the equation is indexed most directly by vagally mediated heart rate variability (HRV). The neurovisceral integration model (Thayer & Lane, 2000; Smith, Thayer, Khalsa, & Lane, 2017) proposes that a central autonomic network

integrating prefrontal cortex, anterior cingulate, insula, amygdala, and brainstem regulates both autonomic function and cognitive-emotional processing. HRV indexes the functional output of this network and thus serves as a biomarker of self-regulatory capacity.

The meta-analytic evidence supports this link quantitatively. Magnon et al. (2022) conducted a systematic review and meta-analysis of 13 correlational studies (52 effect sizes) and found that vagally mediated HRV predicted executive functioning at $r = 0.19$ (95% CI [0.15, 0.23], $p < .0001$), with stronger associations for cognitive inhibition and cognitive flexibility — precisely the capacities required to override habitual responding.

2.3 Stress Promotes Habitual Over Goal-Directed Behavior

The mechanism linking load to habit is the stress-induced shift from goal-directed to habitual behavioral control. Wirz, Bogdanov, and Schwabe (2018) reviewed evidence that stress promotes a general shift from reflective memory systems (hippocampus, prefrontal cortex) to reflexive habit systems (amygdala, dorsolateral striatum), operating through non-genomic corticosteroid action at mineralocorticoid receptors in interaction with noradrenergic arousal.

An important boundary condition: Hartogsveld et al. (2023) failed to replicate the acute stress-to-habit effect in two preregistered studies, suggesting laboratory demonstrations may not be robust in all settings. However, chronic stress effects remain well-supported: Dias-Ferreira et al. (2009) showed that three weeks of chronic unpredictable stress in rats produced habitual responding correlated with prefrontal/dorsomedial striatal atrophy and dorsolateral striatal hypertrophy.

2.4 Emotion Regulation as Transdiagnostic Habit Driver

The most robust finding linking these streams is that emotion regulation — specifically, the relief of aversive internal states through negative reinforcement — operates as a transdiagnostic mechanism across diverse habit types. González-Roz et al. (2024) conducted five separate meta-analyses across 189 studies ($N = 78,733$) and found emotional dysregulation significantly related to all addictive behaviors

examined, with effect sizes ranging from r = .245 for gambling to r = .372 for cannabis problems.

Baker, Piper, McCarthy, Majeskie, and Fiore (2004) formalized this as the negative reinforcement model of addiction: escape and avoidance of negative affect is the prepotent motive for addictive drug use. Koob (2021) extended this through the hyperkatifeia framework, describing how potentiated negative emotional states during withdrawal drive the shift from positive to negative reinforcement as addiction progresses.

These findings converge on a single principle: persistent costly habits are maintained not primarily by the reward they deliver but by the aversive state they relieve.

> *Epistemic status: All four evidence streams summarized above are Tier 1 (empirically grounded). The integration of these streams into a single load-capacity architecture is Tier 2 (literature-consistent theoretical extension).*

3. Neural Substrates of Habit Formation

Understanding the RGM requires grounding in the neural circuitry that mediates the transition from goal-directed to habitual behavioral control.

3.1 Basal Ganglia Circuits and the Dorsolateral Striatum

The basal ganglia, and particularly the dorsolateral striatum (DLS), constitute the core neural substrate of habitual behavioral control. DLS lesions prevent the transition from goal-directed to habitual responding that normally occurs with extended training (Yin & Knowlton, 2006). Conversely, DLS inactivation in well-trained animals restores sensitivity to outcome devaluation, suggesting that habitual control requires ongoing DLS function.

The DLS receives convergent input from sensory and motor cortices, enabling the integration of contextual cues with learned action patterns. Cortico-striatal synaptic plasticity, mediated by dopamine signaling, encodes the stimulus-response associations that constitute habitual memories.

3.2 Cortico-Striatal-Thalamo-Cortical Loops

CSTC loops create recurrent processing circuits linking specific cortical regions with corresponding striatal territories (Alexander, DeLong, & Strick, 1986). The closed-loop structure enables self-sustaining activity patterns that maintain behavioral execution with minimal external input. Recent evidence suggests that the transfer from goal-directed to automatic control involves competitive rather than purely cooperative dynamics between dorsomedial and dorsolateral striatal systems (Turner et al., 2022).

3.3 Dopamine Pathways and Reinforcement

Two dopamine pathways are particularly relevant. The mesolimbic pathway (ventral tegmental area to nucleus accumbens) supports incentive motivation and reward anticipation. The nigrostriatal pathway (substantia nigra to dorsal striatum) is directly implicated in habit learning and execution. Dopaminergic prediction error

signals (Schultz, Dayan, & Montague, 1997) drive initial learning, but as behavior becomes habitual, dopamine's role shifts from encoding reward prediction errors to maintaining stimulus-response associations.

3.4 The Stress-Induced Shift

Acute stress produces a systematic shift from goal-directed to habitual behavioral control, rendering behavior insensitive to outcome devaluation — the hallmark of habitual control (Schwabe, Dickinson, & Wolf, 2011). Chronic stress produces more durable changes: Dias-Ferreira et al. (2009) demonstrated that three weeks of chronic unpredictable stress produced prefrontal/dorsomedial striatal atrophy alongside dorsolateral striatal hypertrophy. This structural remodeling favors habitual responding long after the stressor has resolved, providing a neural mechanism for how chronic load can produce persistent habit formation.

Critical gap in standard models: Standard prediction error models struggle to explain compulsive checking and cleaning behaviors that produce minimal reward yet persist and intensify under stress. This suggests behaviors may be maintained not by reward prediction but by temporary reduction of aversive states — precisely the function proposed by the RGM's closure-simulation channel.

> *Epistemic status: The neural substrates described in this section are Tier 1 (empirically grounded). The specific claim that these circuits implement the RGM's channel selection process is Tier 2 (literature-consistent theoretical extension).*

4. The Regulation-Gap Model: Formal Framework

4.1 The Regulation Gap Equation

The core of the RGM is expressed as a conceptual inequality:

Regulation Gap (RG) = Total Load (L) − Available Capacity (C)

When RG > 0, the system enters a state of regulatory deficit and activates what the model terms habit search behavior — the organism's implicit scanning for available means to reduce the experienced gap. The channel selected follows a weighted heuristic function inspired by decision theory:

$$\textit{Channel Selected} = \textit{argmax}\,[\textit{Relief}(i) \times \textit{Availability}(i) \times \textit{Speed}(i) \times (1/\textit{Friction}(i))]$$

Critical caveat and epistemic honesty: This equation is a conceptual tool, not a measurable formula in its current form. It should be interpreted as a directional inequality (RG > 0 triggers habit search; RG ≤ 0 permits regulatory stability) rather than as a scalar calculation. The equation's value lies in generating directional predictions rather than in point estimation.

4.2 Threshold Dynamics and Gap Emergence

Gap effects are not linear but show threshold dynamics: small gaps may be tolerated without behavioral compensation through residual capacity or passive regulatory mechanisms; moderate gaps trigger single-channel recruitment; large gaps produce multi-channel portfolio activation. This non-linearity creates conditions where apparently small changes in load or capacity produce disproportionate behavioral effects — explaining the commonly observed phenomenon of sudden habit formation during stress periods and sudden dissolution during life transitions.

4.3 Feedback Loops

Regulatory habits generate two classes of feedback:

Negative feedback occurs when channel engagement successfully reduces the regulation gap. The behavior provides genuine, if temporary, relief. This is the standard maintenance mechanism for most regulatory habits.

Positive feedback operates when channel engagement paradoxically increases load or depletes capacity. Alcohol initially reduces the gap through GABAergic sedation but subsequently increases physiological load through HPA axis disruption, sleep fragmentation, and metabolic cost. This positive feedback dynamic characterizes the transition from habit to addiction described in the RGM's Layer I (Collapse Conditions).

4.4 Attractor States and Behavioral Stability

From a dynamical systems perspective, habit portfolios represent attractor states — stable configurations toward which the system converges from diverse initial conditions. The strength of attraction (habit stability) depends on basin depth and width: deeply entrenched habits (long-established, multiply reinforced, emotionally intense formation) resist perturbation and rapidly recover when disturbed; shallow attractors (recently formed, single-channel, low emotional intensity) permit easier transition.

The Regulation-Gap Model adds critical insight: attractor strength is dynamically modulated by load-capacity balance. Under high load, habit attractors deepen — behavior becomes more rigid and resistant to change. Under enhanced capacity, attractors shallow — behavior becomes more flexible and responsive to new information.

4.5 The Ten-Layer Human Regulation Map

The RGM organizes the process from environmental conditions through habit dissolution into ten functionally distinct layers:

Layer A — Condition Field

The total environment in which the organism operates: light exposure, food environment, work demands, digital landscape, relational ecology, sleep

architecture, and movement opportunity. This is the ground from which load is generated and within which capacity is either supported or degraded.

Layer B — Load Generation

Five load types are distinguished: cognitive (decision density, information processing demands), emotional (unresolved affect, grief, anxiety, relational rupture), relational (co-regulation deficits, attachment strain, social conflict), environmental (noise, instability, financial precarity), and physiological (sleep debt, circadian disruption, inflammation, pain). The bandwidth tax of poverty documented by Mani, Mullainathan, Shafir, and Zhao (2013) — which reduces effective cognitive capacity by approximately 13 IQ points — illustrates how environmental load can consume regulatory resources independent of individual factors.

Layer C — Regulatory Capacity

The organism's available resources for managing load: sleep quality and architecture, nutritional status, movement and physical activity, social safety and co-regulation availability, closure and completion of pending demands. The meta-analytic finding that within-person sleep quality predicts self-control at $r = 0.35$ (Guarana et al., 2021) provides direct evidence that capacity fluctuates meaningfully on a daily basis.

Layer D — Regulation Gap

The computed difference between Layers B and C. When the gap is positive, the organism experiences regulatory deficit. This is the model's key causal variable.

Layer E — Channel Selection

The implicit search process that identifies the highest-relief, lowest-friction, fastest-onset, most contextually available regulatory behavior. This process operates largely outside conscious awareness.

Layer F — Habit Formation

The standard trigger → channel → relief → reinforcement cycle, consistent with established habit formation research. Relief from regulatory deficit functions as the reinforcing outcome.

Layer G — Portfolio Stabilization

Over time, organisms develop not a single regulatory habit but a portfolio of multiple channels serving different aspects of the regulation gap. This is the RGM's explanation for the robust finding that health-risk behaviors cluster.

Layer H — Portfolio Rebalancing

When one channel is removed or blocked, the load it was carrying redistributes across remaining channels. This layer generates the model's most distinctive prediction about compensatory behavior.

Layer I — Collapse Conditions

When load vastly and persistently exceeds capacity, the system enters states recognizable as clinical addiction, behavioral fragmentation, or burnout. Koob's (2021) three-stage addiction cycle describes the dynamics of collapse.

Layer J — Dissolution Pathway

The central therapeutic implication: habit dissolution occurs primarily through capacity increases combined with load decreases, which together close the regulation gap and render the regulatory habit functionally irrelevant.

> *Epistemic status: The ten-layer structure is a Tier 2 theoretical organization of empirically grounded phenomena (Tier 1). No claim is made that these layers represent discrete neural systems or that the ordering is invariant.*

Figure 1

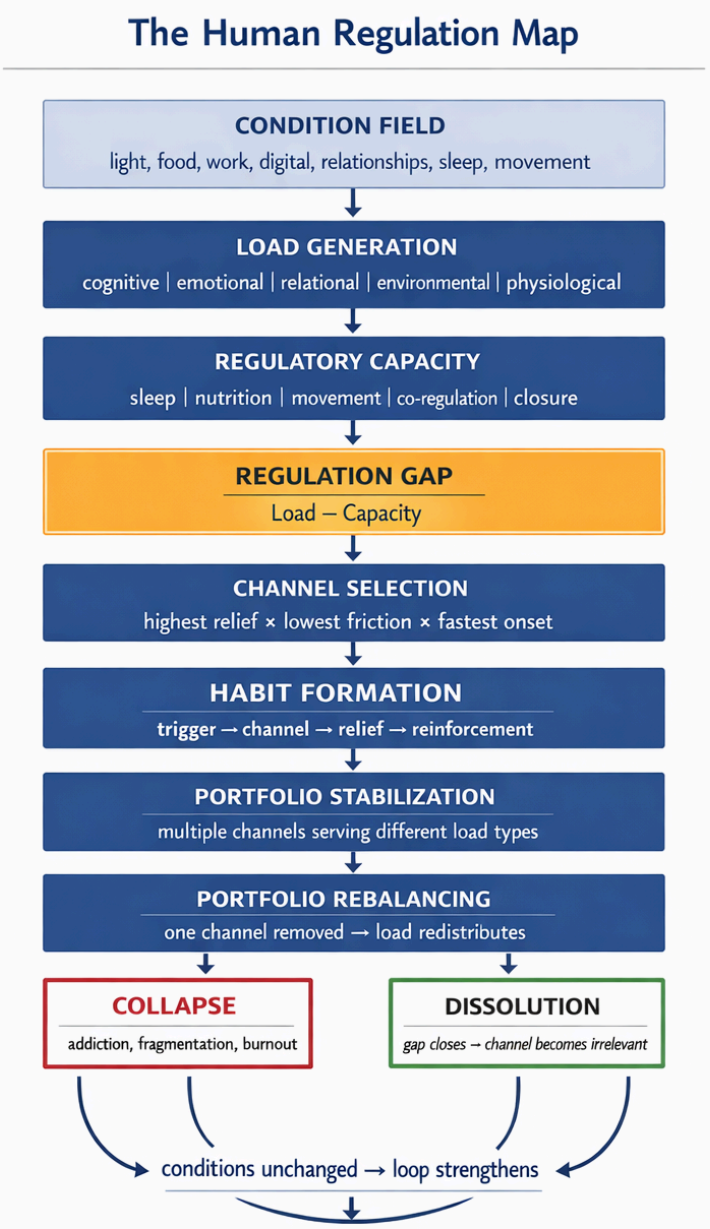

Figure 1. The Human Regulation Map: a ten-layer process model from environmental conditions through habit dissolution. The regulation gap (Layer D) is

the model's key causal variable. When load exceeds capacity, the system activates channel selection behavior. Dissolution occurs when the gap closes through load reduction, capacity enhancement, or both. Collapse occurs when the gap persistently exceeds the system's compensatory range.

5. The Six Regulation Channels

The RGM classifies persistent regulatory habits by their functional mechanism rather than by the specific behavior or substance involved. These are functional roles, not rigid taxonomy — a single behavior can serve multiple channels simultaneously, and individuals may use the same channel through different behaviors.

5.1 Shutdown

Reduces signal intensity through inhibitory neurochemistry. Alcohol achieves this through allosteric modulation at GABA-A receptors, producing anxiolytic and sedative effects and elevating GABAergic neurosteroids (Morrow et al., 2006; Stephens & Wand, 2012). HPA axis cortisol modulation occurs simultaneously: mild intoxication can actually inhibit HPA axis response to stress, while higher doses elevate ACTH, cortisol, and norepinephrine. Other shutdown behaviors include cannabis use, overeating, and sedative medication.

5.2 Stimulation

Increases activation to override affective flatness or to generate engagement that displaces aversive internal states. Caffeine, social media scrolling, notification-checking, and novelty-seeking behaviors serve this function. Variable-ratio reinforcement schedules in digital environments create "dual activation" where both conditioned and unconditioned stimuli trigger dopamine release, enabling perpetual escalation of incentive salience (Clark & Zack, 2023).

5.3 Absorption

Narrows all attentional bandwidth to a single track, eliminating peripheral awareness of load. Natasha Dow Schüll's (2012) ethnographic documentation of the "machine zone" in gambling has been extended empirically to social media. Baughan et al. (2022) found that 42% of participants reported dissociative experiences during social media use. Work immersion, research spirals, and binge-watching serve equivalent absorptive functions.

5.4 Discharge

Releases stored physiological tension through motor activity. Body-focused repetitive behaviors (BFRBs) — skin picking, hair pulling, nail biting, jaw clenching — are the paradigmatic examples. Moritz et al. (2024) found that 84.7% of individuals with BFRBs reported stress release as their primary motive. Houghton et al. (2018) documented subclinical BFRBs in 59.55% of a large college sample, suggesting discharge behaviors are near-universal regulatory mechanisms.

5.5 Closure-Simulation

Generates proxy completion experiences that temporarily reduce the aversive load of unfinished tasks and open loops. Cleaning the kitchen instead of making a difficult decision, checking email to feel "caught up," or reorganizing a workspace during a period of professional uncertainty all serve this function. The Zeigarnik effect — intrusive cognition about incomplete tasks — generates load that closure-simulation behaviors temporarily relieve.

5.6 Dissociation/Avoidance

Delays transitions and suppresses awareness of pending demands. Staying in the car after arriving home, numbing, postponing decisions, and experiential avoidance behaviors fall in this channel. Den Ouden et al. (2020) demonstrated that experiential avoidance predicts transdiagnostic compulsive behavior through the mediating pathway of psychological distress (N = 469).

5.7 Channel Selection Dynamics

Channel selection is determined by three interacting factors:

Load characteristics. Acute versus chronic load favors different channels. High-intensity load favors high-intensity channels. Domain match matters: cognitive overload may favor absorption, while emotional overload may favor shutdown.

Individual history. Past reinforcement shapes channel availability. Cultural norms influence social acceptability. Biological sensitivity (genetic factors, neurodevelopmental conditions) affects response magnitude.

Contextual affordances. Channels must be contextually accessible. Social setting determines which channels are available. Time pressure affects which onset speeds are feasible.

> *Epistemic status: The six-channel taxonomy is Tier 2 (literature-consistent theoretical extension). The functional categories are supported by converging evidence from emotion regulation, addiction neuroscience, and BFRB research, but the specific taxonomy is proposed by this model and has not been independently validated.*

6. Testable Predictions with Explicit Kill Criteria

The following predictions are designed to be falsifiable through feasible experimental designs using existing instruments. Each includes a specific kill criterion — a result pattern that, if obtained, would require abandoning or substantially revising the corresponding component of the model.

Prediction 1: Habit Intensity Tracks the Regulation Gap Physiologically

Claim: Within individuals, habit engagement intensity correlates with a composite physiological load-capacity index operationalized as the ratio of salivary cortisol to resting HRV (RMSSD).

Design: Within-subjects ecological momentary assessment over 21 days ($n \geq 30$). Participants provide twice-daily salivary cortisol samples and continuous HRV via wearable device. Multilevel modeling tests whether within-person fluctuations in cortisol/HRV ratio predict same-day and next-day habit engagement intensity.

Kill criterion: If habit frequency shows no within-person correlation ($r < 0.15$) with physiological load markers across the study period, the regulation-gap model fails at the physiological measurement level.

Prediction 2: Channel Blocking Produces Measurable Portfolio Rebalancing

Claim: When environmental friction for one regulatory channel increases, compensatory engagement in alternative regulatory channels increases within the same experimental session.

Design: Within-subjects crossover design ($n \geq 40$). Participants undergo both a channel-blocked condition (primary self-identified regulatory habit physically prevented for 4 hours) and a control condition in counterbalanced order.

Kill criterion: If blocking one channel produces no measurable compensatory increase in alternative channel engagement (< 10% increase) in ≥ 85% of subjects, portfolio rebalancing is not occurring at observable timescales.

Prediction 3: Capacity Change Outpredicts Motivation for Habit Dissolution

Claim: In a prospective design following individuals attempting habit change, physiological capacity markers predict six-month cessation success more strongly than self-reported motivation, intention strength, and willpower confidence.

Design: Prospective cohort ($n \geq 120$) of individuals initiating voluntary habit change. Baseline and monthly assessments over six months. Hierarchical regression testing incremental prediction.

Kill criterion: If motivation/intention scores predict cessation success better than physiological capacity markers (motivation adds $\Delta R^2 > 0.05$ while capacity adds < 0.02), the condition-change model loses explanatory priority. This is the prediction most likely to kill the model.

Prediction 4: Life Transitions Disrupt Habits Through Regulation-Gap Mechanisms

Claim: The well-documented habit disruption during life transitions is partially mediated by changes in the regulation gap — specifically, transitions that increase capacity produce greater habit disruption than transitions that merely change context.

Design: Longitudinal within-subjects design ($n \geq 80$) assessing individuals before, during, and after verified life transitions. Path analysis tests whether regulation gap change mediates the transition-disruption relationship.

Kill criterion: If regulation gap change shows no independent mediation effect (indirect effect 95% CI includes zero) beyond context change, the RGM adds nothing to the existing habit discontinuity hypothesis and should be considered redundant.

Epistemic status: All four predictions are Tier 3 (framework-inferred hypotheses). The individual components cited within each prediction are Tier 1. The specific composite measures and their proposed relationships have not been tested.

7. The Dissolution Pathway and Why Capacity Change Predicts Recovery

The RGM's most clinically significant claim is that persistent costly habits dissolve primarily through condition change rather than through motivational intensification. Three lines of evidence converge.

Natural recovery is the statistical norm for alcohol use disorder. Tucker, Chandler, and Witkiewitz (2020) reported that approximately 70% of persons with AUD improve without formal interventions, with fewer than 25% utilizing alcohol-focused services. This finding is predicted by the RGM: as life conditions change, the regulation gap narrows and the habit becomes progressively less necessary.

The habit discontinuity hypothesis demonstrates context-dependent dissolution. Verplanken and Roy (2016) showed that interventions promoting sustainable behaviors were more effective among recently relocated participants. Bouton (2021, 2024) provided mechanistic clarification: context change does not erase habit memories but suppresses their expression by restoring goal-directed control.

Physiological capacity markers predict self-regulation more robustly than motivational variables. The within-person effect of sleep quality on self-control ($r = 0.35$; Guarana et al., 2021) exceeds most effect sizes reported for motivational interventions.

The RGM does not dismiss willpower entirely but relativizes its role. Willpower can interrupt a habit execution, bridge a gap during a transition period, or initiate a condition-change process. What it cannot do, the model predicts, is sustain dissolution in the absence of capacity change.

7.1 Four Dissolution Outcomes

The RGM specifies four possible outcomes when habit-change is attempted:

Suppression. The person fights the channel while leaving the regulation gap intact. Desire remains, behavior is blocked through effortful control. This is costly, unstable, and often produces portfolio rebalancing.

Substitution. The person swaps one channel for another with lower cost profile. Some stabilization occurs, but the system remains channel-dependent.

Dissolution. The conditions that generated the regulation gap change sufficiently that the channel is no longer selected. The behavior becomes irrelevant rather than resisted. This is the RGM's predicted optimal outcome.

Residual vulnerability. The regulation gap has closed and the behavior has become functionally irrelevant, but the stimulus-response association remains latent and cue-reactivable (Bouton, 2021, 2024). This explains relapse upon return to the original habit context even after extended dissolution.

8. Habit Portfolio Dynamics

8.1 Portfolio Emergence Under Sustained Load

Under sustained regulatory deficit, organisms rarely adopt a single regulatory strategy. Instead, they develop habit portfolios — coordinated sets of regulatory channels that collectively manage different aspects of the regulation gap across the day:

Morning: stimulation (caffeine to counter sleep debt); Workday: absorption (work immersion to manage cognitive complexity); Evening: shutdown (alcohol to reduce accumulated emotional and cognitive load); Night: discharge (nail biting or skin picking to release somatic tension before sleep).

This portfolio structure explains the well-documented clustering of health-risk behaviors: smoking, alcohol use, physical inactivity, and poor diet do not merely co-occur — they serve complementary regulatory functions within a single load-management system.

8.2 Substitution Dynamics

When individuals attempt to eliminate one regulatory behavior without addressing the underlying regulation gap, the system recruits replacement channels. Post-bariatric surgery data showing elevated alcohol use disorder rates (11-20%) following gastric bypass may reflect surgical closure of the eating channel without concurrent gap reduction. Weight gain following smoking cessation — occurring in 77-86% of cases — exemplifies portfolio rebalancing in the consumption domain.

8.3 Alternative Explanations for Apparent Rebalancing

Portfolio rebalancing should not be presented as established law. Alternative explanations include: visibility effect, recovery-phase distortion, shared trait confound, and opportunity effect. Sinclair et al.'s (2021) finding that only 17.65% of studies supported addiction substitution while 52.94% found concurrent recovery indicates that portfolio rebalancing is not the dominant pattern. The RGM predicts it

should occur specifically when the regulation gap remains positive after one channel is removed.

8.4 Distinguishing Predictions from Competing Models

9. Somatic Regulation and the Body as Regulatory Reservoir

9.1 Vagal Regulation and the Polyvagal Framework

Stephen Porges's polyvagal theory offers an evolutionary-phylogenetic framework for understanding vagal regulation. The theory distinguishes three hierarchical neural circuits: the ancient unmyelinated vagal system supporting immobilization defenses; the sympathetic nervous system supporting mobilization (fight-flight); and the mammalian myelinated vagal system supporting social engagement and communication (Porges, 2011).

The social engagement system enables co-regulation through social interaction that can substantially expand individual regulatory capacity. This provides neurobiological grounding for the RGM's inclusion of co-regulation as a capacity resource: secure social engagement literally increases the denominator in the regulation gap equation.

9.2 Interoception and Body-Based Regulation

Interoceptive awareness — the capacity to detect and interpret internal physiological signals — represents both a capacity resource and a regulation mechanism. Slow, diaphragmatic breathing enhances vagal tone and HRV, directly increasing regulatory capacity. Body awareness and movement practices enhance regulation through somatic integration.

9.3 Somatic Regulation and Connective Tissue Constraints

While the Regulation-Gap Model is primarily framed in behavioral and physiological terms, an additional substrate of regulatory load may exist within the body's connective tissue system. Fascia is a continuous connective tissue network that surrounds and interpenetrates muscles, organs, nerves, and vascular structures. Rather than functioning solely as passive packaging, fascia is now understood as a dynamic sensory and regulatory organ with dense mechanoreceptive innervation (Schleip, Jäger, & Klingler, 2012).

From the perspective of the Regulation-Gap Model, fascial tension patterns can be understood as somatic load storage — a structural adaptation that emerges when regulatory demand exceeds the system's capacity to resolve stress dynamically.

Under repeated mechanical or emotional stress, fascia demonstrates viscoelastic remodeling. Fibroblasts within fascial tissue respond to sustained strain by reorganizing collagen fibers and altering tissue stiffness. Clinically, these structural adaptations manifest as persistent jaw tension, restricted diaphragm movement, thoracic compression, hip or sacral stiffness — patterns that correspond closely to zones where individuals report chronic stress holding.

The diaphragm functions not only as the primary respiratory muscle but as a central mechanical interface between thoracic and abdominal cavities. Under chronic stress, reduced diaphragmatic motion can limit vagal tone and decrease heart rate variability, directly reducing regulatory capacity. The result is a feedback loop: chronic stress → fascial restriction → reduced vagal tone → reduced capacity → larger regulation gap → increased habit dependence.

> *Epistemic status: The somatic load distribution hypothesis is Tier 4 (analogical/cross-domain). The individual components — fascial mechanoreception, viscoelastic remodeling under stress, diaphragmatic influence on vagal tone — are Tier 1. The specific claim that fascial tension patterns constitute a measurable substrate of the regulation gap is a novel hypothesis requiring empirical testing.*

10. Epistemic Status Registry

The RGM employs a tiered epistemic status system to distinguish empirically grounded claims from theoretical extensions:

Tier 1 — Empirically Grounded

Emotion dysregulation as transdiagnostic driver; habits as context-dependent automatic associations; life transitions disrupting habits; stress promoting habitual behavior; automaticity transfer from prefrontal to striatal circuits; friction-based and structural interventions affecting behavior; HRV indexing regulatory capacity ($r = 0.19$); sleep quality predicting self-control ($r = 0.35$); natural recovery from AUD (~70%); dorsolateral striatum mediating habitual control; BFRBs serving emotion regulation functions; fascial mechanoreception and viscoelastic remodeling.

Tier 2 — Literature-Consistent Theoretical Extension

The regulation gap as a formal model integrating load and capacity constructs; the six-channel functional taxonomy of regulatory habits; the ten-layer regulation map as a process model; willpower as interrupt/bridge function; threshold dynamics in gap emergence; attractor state formalization of habit portfolios; feedback classification of regulatory habit dynamics.

Tier 3 — Framework-Inferred Hypotheses

Portfolio rebalancing as a systematic, measurable phenomenon; the complete dissolution pathway; channel selection following the specified weighted heuristic; cortisol/HRV ratio as an operationally valid composite index of the regulation gap; capacity markers outpredicting motivation for dissolution outcomes; residual vulnerability as a fourth dissolution outcome.

Tier 4 — Analogical and Cross-Domain

Structural parallels between fascial tissue adaptation and behavioral habit patterning; somatic load distribution as a measurable substrate of the regulation gap.

This paper makes no Tier 4 claims in its formal predictions. All testable predictions are Tier 3, grounded in Tier 1 evidence through Tier 2 theoretical integration.

11. Limitations, Boundary Conditions, and Honest Uncertainties

11.1 The Model's Scope Is Narrower Than Habits in General

The RGM applies most strongly to what might be termed regulatory habits — persistent, costly, self-reinforcing behaviors maintained primarily through negative reinforcement. It does not claim to explain all habitual behavior. A useful three-way distinction separates: (a) automatic routines that are habitual but not regulatory, (b) regulatory habits (the RGM's primary domain), and (c) compulsive habits where regulatory function has been overtaken by neuroadaptive processes.

11.2 The Six Channels Are Functional Roles, Not Rigid Taxonomy

Behaviors serve multiple channels simultaneously. Alcohol can function as shutdown, absorption, and dissociation. The taxonomy's value lies in asking "what function is this behavior serving right now" rather than in classifying behaviors into fixed categories.

11.3 The Equation Is Not Yet Independently Measurable

Total Load and Available Capacity are multidimensional constructs that cannot be reduced to single scalar values with existing instruments. Formalizing these variables into independently measurable quantities remains a priority for future operationalization.

11.4 Willpower Is Relativized, Not Dismissed

Executive inhibitory control can interrupt a habit, bridge a period of high gap, and initiate environmental restructuring. What it cannot do is sustain dissolution in the absence of capacity change — a specific and testable claim (Prediction 3) that could be falsified.

11.5 Individual Differences Are Underspecified

The current model does not adequately account for individual differences in neurodevelopmental conditions (ADHD, autism spectrum), personality traits, genetic vulnerability, or learning history.

11.6 Meaning and Identity Functions of Habits

Some persistent habits serve meaning-making and identity functions that the RGM underweights. Identity functions may themselves serve regulatory purposes (belonging reduces relational load; purpose reduces existential load), but this integration requires further development.

11.7 Poverty and Constrained Choice

The model's emphasis on condition change may be inapplicable when conditions are structurally constrained. The RGM's most important implication for constrained populations may be negative: stop blaming individuals for regulatory habits that are rational responses to conditions they cannot change.

11.8 Cultural Variability

Cultural norms and values significantly influence which regulatory channels are available, acceptable, and effective. Channel selection is not culturally neutral.

12. Toward Clinical Translation

While this paper reports no clinical data, the RGM generates specific implications for intervention design that differ from standard approaches.

Assessment priority shifts from behavior to function. Rather than asking "How often do you drink?" the clinically relevant question becomes "What is the drinking regulating?" The six-channel taxonomy provides a structured assessment framework.

Intervention targets the condition field, not just the behavior. Capacity-building interventions — sleep hygiene, circadian realignment, social reconnection, physical movement — directly reduce the regulation gap and should be initiated before or simultaneously with habit-targeted interventions.

Friction-based approaches are preferred under high load. Structural and friction-based interventions bypass depleted executive resources. Decision-structure nudges outperform decision-information nudges in the food domain by a factor of 2.5× (Mertens et al., 2022).

Portfolio awareness prevents channel migration without gap closure. When clinicians or individuals successfully eliminate one regulatory behavior, monitoring for compensatory channel engagement is essential unless the regulation gap itself has been addressed.

13. Conclusion: What Changes If the Regulation Gap Is Real

The Regulation-Gap Model proposes a simple but consequential reframing: a persistent costly habit is not fundamentally a behavior problem, a motivation deficit, or a neural circuit malfunction. It is a routing decision by a load-bearing organism that has found the lowest-friction available path to regulatory relief.

If the model survives empirical testing, several implications follow. The blame traditionally directed at individuals for failing to change their habits becomes recognizable as a category error: demanding that someone stop a load-bearing behavior without changing the load is not a reasonable expectation but a misunderstanding of the system's architecture. The clinical focus shifts from "How do I make you stop?" to "What is this behavior carrying, and how do we build alternative support?" The population-level implication aligns with Hofmann's (2024) call for multilevel self-control research and Chater and Loewenstein's (2023) s-frame: conditions that generate chronic regulatory deficits are not merely correlated with persistent costly habits but are, under the RGM, their primary cause.

If the model fails empirical testing — particularly if Prediction 3 shows motivation clearly outpredicting capacity — this would indicate that persistent habits are more volitional and less condition-dependent than the RGM assumes. To be clear: this model is intended to explain persistent, costly, self-reinforcing habits — not all routine automatized behavior. The kill criteria specified for each prediction ensure that the model can die cleanly, and the epistemic status registry ensures that the reader knows which claims carry empirical weight and which are theoretical proposals awaiting evidence. No theoretical framework deserves to survive contact with data it cannot accommodate. This one is designed to be tested.

Memetic Core

> *A persistent habit is not primarily a failure of discipline. It is a routing decision made by a biological system attempting to stabilize itself under load. When total regulatory demand exceeds the organism's available capacity, a regulation gap emerges. Behaviors that reduce this gap — by lowering signal intensity, increasing stimulation, narrowing attention, discharging tension, simulating closure, or delaying contact with stressors — are reinforced because they restore temporary stability. These behaviors persist not because they are pleasurable, but because they are load-bearing. Attempts to eliminate them without reducing the underlying load or increasing regulatory capacity force the system to recruit alternative regulatory channels. Sustainable habit change occurs not when motivation increases but when the conditions generating the regulation gap change. When load decreases or capacity increases sufficiently, the behavior that once stabilized the system becomes unnecessary and often dissolves without force.*

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Conflicts of Interest

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