Appendix D

This appendix is an independent analysis based on the framework presented in:
A Dynamical Model of Subjectivity: Integrating Affective Gain, Cognitive Bias, and Self-Regulation
doi:10.31234/osf.io/8dbft_v2

Reconstructing ADHD through the G–μ–S Dynamic Model:

A Framework Integrating Theory, Evidence, and Clinical Practice

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June 12, 2025

Disclaimer

This appendix is intended solely to explore potential applications of the underlying article and does not constitute medical or psychological advice. The ADHD G–μ–S dynamic model presented herein is a hypothetical framework; its clinical validity has not yet been established. When reusing or citing this material, you must reproduce this disclaimer in full, clearly indicate any modifications made, and comply with the repository's license. The author accepts no liability for any loss or damage arising from use of this content. If you do not agree to these terms, please refrain from using this material.

Abstract

This paper refines the G–μ–S dynamic model, integrates recent computational and neuroscientific findings, and delineates clinical implications for Attention-Deficit /Hyperactivity Disorder (ADHD). By conceptualising ADHD as a dysregulation of a higher-order self-system that learns to modulate affective gain (G) and cognitive bias (μ), the framework unifies diverse symptoms, individual differences, and treatment mechanisms within a coherent, testable architecture.

0 Objective and Overview

We enhance the original draft along three axes:

1. Theoretical refinement of the G–μ–S framework
2. Synthesis of cutting-edge evidence from computation and neuroscience
3. Concrete mapping of clinical and interventional implications

The paper is organised as follows:

1. Re-organisation of the G–μ–S framework
2. Parameter-space interpretation of ADHD
3. Neural and computational substrates of G, μ, and the Self-System (S)
4. Visualisation of dynamic instability and simulation insights
5. Individual differences and comorbidities via the SGBD extension
6. Intervention mapping and research predictions

1 Key Features of the G–μ–S Framework

- **Mind Topography Map** The G–μ plane comprises stable, bistable, and unstable regions actively navigated by the Self-System (S).
- **Ideal Dynamic Equilibrium (IDE)** At $(G = 1, \mu = 0)$ the linear term disappears, yielding a state that is both stable and highly responsive to noise.
- **Gain-dependent Effective Bias ($G\mu$)** Because bias is multiplied by gain, changes in G amplify or attenuate μ dynamically, distinguishing this model from prior approaches.

2 Interpreting ADHD in Parameter Space

Key Point. ADHD primarily reflects a meta-learning deficit in S—either a reduced learning rate for its hyper-parameters ($\boldsymbol{\theta}_{\mathrm{S}}$) or optimisation of a short-sighted objective $\mathcal{L}$. Empirical evidence corroborates reward delay discounting and impaired neural gain.

3 Neural and Computational Mechanisms

3.1 Affective Gain (G)

Dopamine and noradrenaline modulate the signal-to-noise ratio (SNR). Stimulants such as methylphenidate enhance prefrontal–striatal SNR, effectively nudging G toward 1. The neural-gain impairment hypothesis attributes behavioural variability to malfunctioning local gain regulation.

Table 1: Interpretation of ADHD core symptoms within the G–μ–S parameter space.

Core Symptom	**Affective Gain (G)**	**Cognitive Bias (μ)**	**State of S & Dynamic Consequence**
Inattention	Low G weakens error-driven updating and stimulus selectivity.	μ fluctuates and fails to stabilise.	Context-adaptive retuning by S is delayed; even in a monostable zone convergence is slow, impairing sustained attention.
Hyperactivity / Impulsivity	High G triggers over-reactions to minor errors.	μ rapidly collapses toward impulsive attractors.	Gain control by S cannot keep pace; trajectories leap into high-G regions, diverging or plunging toward a bias.
Emotional Dysregulation	High G combined with strong negative μ.		S's objective $\mathcal{L}$ is shortened via delay discounting. IDE's restorative pull weakens, yielding chaotic transitions or bistable switching.

3.2 Cognitive Bias (μ)

A latent vector shaped by development, memory, and belief. In ADHD, μ oscillates between a positive component favouring immediate reward and a negative component driven by the anticipation of failure.

3.3 Self-System (S)

Equivalent to Barkley's self-regulation construct: executive-function deficits signify weak control by S. Hierarchical Bayesian learning in S corresponds to the frontopolar–anterior-cingulate–basal-ganglia loop; in ADHD, a lowered learning rate (η_θ) and mis-estimated confidence slow convergence.

4 Dynamic Instability and Clinical Observations

The bistable zone ($G < 0, |\mu| > 0$) reproduces abrupt switches between hyper-focus and distractibility reported by patients. Amplified noise (ϵ) manifests as elevated reaction-time variability, which reliably normalises following treatment.

5 Modelling Individual Differences and Comorbidities with SGBD

Structural matrices $\mathbf{M_G}$ and $\mathbf{M}_\mu$ capture variations in emotional-cognitive wiring. Persistent negative μ in a specific dimension can simultaneously model comorbid anxiety or

depression, e.g. a $\mu < 0$ on the social-evaluation axis in social-anxiety comorbidity, while G remains typical.

6 Therapeutic Intervention Mapping

Table 2: Mapping of therapeutic interventions to the G–μ–S model parameters.

Intervention	**Model Target**	**Theoretical Effect**	**Expected Parameter Trajectory**
Stimulants (MPH/AMP)	G, ϵ	Elevate SNR $\rightarrow$ normalise G toward 1, dampen noise.	Converge toward IDE from both high- and low-G extremes.
CBT / Parent Training	S ($\boldsymbol{\theta}_S$, $\mathcal{L}$)	Support meta-learning; re-appraise reward.	$\eta_\theta \uparrow$; $\mathcal{L}$ re-aligned to long-term goals.
Metacognitive Training / Mindfulness	G, S	Detect and voluntarily modulate gain.	Reduced G variability; increased IDE dwell time.
Digital Neurofeedback	μ, G	Real-time bias visualisation.	$\mu \rightarrow 0$, G into optimal range.

Key Point. This mapping illustrates the framework's utility in mechanistically unifying diverse interventions. Pharmacological treatments can be seen as directly tuning low-level parameters (G, ϵ), while psychosocial therapies aim to reshape the higher-order Self-System (S), representing complementary approaches to regulating the same dynamic system.

Future Research Directions

- Pupil-diameter variability as a biomarker of G and treatment responsiveness.
- Task-based estimation of η_θ in hierarchical reinforcement-learning paradigms to quantify plasticity of S.
- Longitudinal neuro-model coupling to chart how interventions redirect Mind-Map trajectories.

Conclusion

Reconceptualising ADHD as a dysregulation of the meta-learning Self-System (S) that governs affective gain (G) and cognitive bias (μ) coherently integrates computational, neuroscientific, and clinical findings. This framework:

1. Explains heterogeneous symptoms, individual differences, and comorbidities within a single dynamic model.
2. Treats pharmacological and psychosocial interventions as parameter-tuning processes.
3. Generates testable biomarkers and task predictions grounded in model dynamics.