Appendix D

This appendix is an independent analysis based on the framework presented in:
A dynamic model of subjectivity integrating emotional gain, internal bias, and self-regulation
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Reconstructing ADHD through the G–μ–S Dynamic Model: A Framework Integrating Theory, Evidence, and Clinical Practice

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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 emotional gain (G) and internal 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

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

Core Symptom	**Emotional Gain (G)**	**Internal 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 overreactions 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.

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 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.

3.2 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 emotional gain (G) and internal 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.