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
https://doi.org/10.1101/XXXXXXX

Reconstructing ADHD through the G–μ–S Dynamic Model: A Framework Integrating Theory, Evidence, and Clinical Practice

Takeo Imaizumi
Independent Researcher, Kyoto City, Japan

June 8, 2025

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.