# Appendix D

# 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 12, 2025

# Abstract

This paper refines the G–$\mu$–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 ($\mu$), 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–$\mu$–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–$\mu$–S framework
2. Parameter-space interpretation of ADHD
3. Neural and computational substrates of $G$, $\mu$, 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–$\mu$–S Framework

- **Mind Topography Map**   The $G$–$\mu$ 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 $\mu$ 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 ($\boldsymbol{\theta}_S$) 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–$\mu$–S parameter space.

| Core Symptom | Affective Gain ($G$) | Cognitive Bias ($\mu$) | State of $S$ & Dynamic Consequence |
|---|---|---|---|
| **Inattention** | Low $G$ weakens error-driven updating and stimulus selectivity. | $\mu$ 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. | $\mu$ 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 Dys-regulation** | High $G$ combined with strong negative $\mu$. | | $S$'s objective $\mathcal{L}$ is shortened via delay discounting. IDE's restorative pull weakens, yielding chaotic transitions or bistable switching. |

## 3.2 Cognitive Bias ($\mu$)

A latent vector shaped by development, memory, and belief. In ADHD, $\mu$ 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 ($\eta_\theta$) 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 ($\epsilon$) 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 $\mu$ 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

**Key Point.** This mapping illustrates the framework's utility in mechanistically unifying diverse interventions. Pharmacological treatments can be seen as directly tuning low-

Table 2: Mapping of therapeutic interventions to the G–$\mu$–S model parameters.

| Intervention | Model Target | Theoretical Effect | Expected Parameter Trajectory |
|---|---|---|---|
| **Stimulants (MPH/AMP)** | $G$, $\epsilon$ | Elevate SNR $\rightarrow$ normalise $G$ toward 1, dampen noise. | Converge toward IDE from both high- and low-$G$ extremes. |
| **CBT / Parent Training** | $S$ ($\boldsymbol{\theta}_{\mathrm{S}}$, $\mathcal{L}$) | 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** | $\mu$, $G$ | Real-time bias visualisation. | $\mu \rightarrow 0$, $G$ into optimal range. |

level parameters ($G$, $\epsilon$), 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 $\eta_\theta$ 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 ($\mu$) 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.