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Quantifying Self-Regulation: Neuroevolutionary Insights From Precuneus Alpha Modulation via LORETA Neurofeedback

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Abstract

Self-regulation (SR) is a vital neurobehavioral capacity orchestrating behavior, physiological equilibrium, and emotional resilience through corticothalamic networks spanning the cortex and thalamus. This study formalizes SR as SR = behavioral equilibrium (BE) / (homeostasis [H] + emotional equilibrium [EE]), where BE captures adaptive responses, H denotes physiological stability, and EE reflects affective harmony, positioning neurofeedback (NFB) as a leading intervention. NFB, encompassing LORETA neurofeedback (LNFB) targeting precuneus alpha and real-time fMRI neurofeedback (rt-fMRI-NFB) modulating blood-oxygen-level-dependent (BOLD) signals, enhances corticothalamic modulation across educational, correctional, clinical, pediatric, and ADHD contexts. Evidence from diverse cohorts validates NFB's efficacy, with LNFB improving BE (CPT-3, $p < .008$) and rt-fMRI-NFB stabilizing EE (BOLD, $p < .01$), supported by long-term gains in children (Strehl et al., 2017) and adults (Rance et al., 2018). The back-to-front brain focus, rooted in precuneus primacy (~2 Mya), contrasts with historical frontal emphasis post-Phineas Gage. As noted in experimental findings, surface NFB training boosts neural connectivity. Pre- and postprotocols are rare due to subjective reliance, resistance to objective tracking, and resource limits (Hofmann & Smits, 2008). NFB's standardized protocols (EEG ICC = .87–.92, BOLD consistency) inspire volumetric MRI studies, advancing SR science across the lifespan.

Keywords: self-regulation; precuneus; LORETA neurofeedback; alpha oscillations; neuroplasticity; behavioral equilibrium; emotional equilibrium; homeostasis; volumetric studies; neuroevolutionary dynamics

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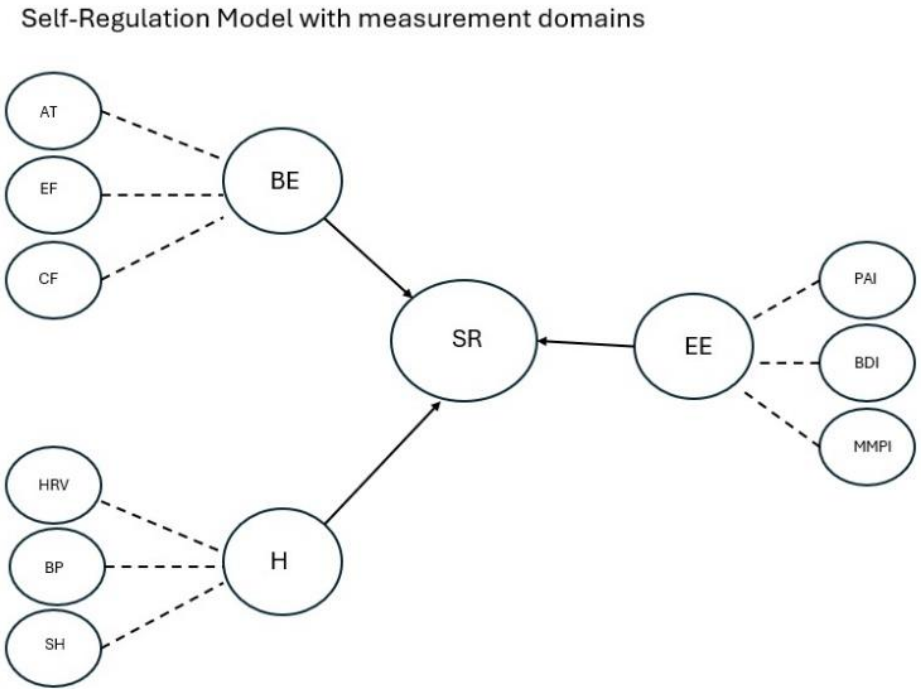
Introduction: The Precuneus in Phylogenetic and Neuroregulatory Context

Self-regulation (SR) emerges as a fundamental neurobehavioral capacity, intricately weaving behavior, physiological stability, and emotional resilience through corticothalamic networks that link the cortex and thalamus. This core capacity, critical across developmental stages and contexts, underpins adaptive functioning in education, corrections, and clinical settings. The study introduces a novel framework, SR = behavioral equilibrium [BE] / (homeostasis [H] + emotional equilibrium [EE]), where BE encapsulates adaptive corticothalamic responses, H reflects physiological balance, and EE signifies emotional coherence, as depicted in Figure 1. Neurofeedback (NFB), comprising LORETA neurofeedback (LNFB) targeting precuneus alpha (8–13 Hz) and real-time fMRI neurofeedback (rt-fMRI-NFB) modulating blood-oxygen-level-dependent (BOLD) signals, stands as a pioneering intervention, harnessing regulatory training of emotional regulation (Johnston et al., 2010) to enhance SR (Zotev et al., 2014). The posterior-to-anterior brain development rationale, emphasizing the precuneus's evolutionary role (~2 million years ago [Mya] in *Homo habilis*) over frontal foci highlighted post-Phineas Gage, guides this approach (Bruner, 2004; Dunbar, 1998).

Figure 1. *SR Model With Measurement Domains.*



Note. This model illustrates SR = BE / (H + EE), integrating BE as adaptive corticothalamic responses (e.g., attention, planning, executive functions), H as physiological stability (e.g., stress hormones, HRV), and EE as affective balance (e.g., mood regulation). In defining the types of instruments for each category AT = attention task; EF = executive functions; CF = cognitive fluency; HRV = heart rate variability; BP = blood pressure; SH = stress hormones; PAI = personality assessment inventory; BDI = Beck Depression Inventory; MMPI = Minnesota multiphasic personality inventory. NFB, including LNFB and rt-fMRI-NFB, targets these domains, with LNFB enhancing precuneus alpha (8–13 Hz) for BE and H, and rt-fMRI-NFB modulating BOLD signals for EE (Johnston, et al., 2010), surpassing selective serotonin reuptake inhibitors (SSRIs) and cognitive-behavioral therapy (CBT; Sitaram et al., 2017). The SR model (SR = BE / [H + EE]) is operationalized using neurophysiological measures inherent to its parameters: BE, H, and EE are quantified via EEG (e.g., alpha coherence), CSD (e.g., precuneus alpha), and BOLD (e.g., amygdala-prefrontal connectivity) to capture corticothalamic dynamics, as detailed in subsequent sections.

Surface NFB training, as later detailed, amplifies neural efficiency. Pre- and postprotocols for treatment success and outcomes remain uncommon across disciplines, often due to reliance on subjective clinical assessments, resistance to integrating objective corticothalamic or neuroendocrine measures, and resource constraints in adopting standardized instruments, EEG or MRI tracking (Hofmann & Smits, 2008; Stahl, 2000).

This framework builds on NFB's legacy, evolving from early EEG protocols (theta-beta, sensorimotor rhythm [SMR]) to precise LNFB and rt-fMRI-NFB modalities, offering a quantifiable alternative to traditional interventions (Sitaram et al., 2017; Thibault et al., 2016). Long-term evidence underscores NFB's potential, with children showing sustained ADHD symptom reduction over 2 years (Strehl et al., 2017) and adults exhibiting 12-month depression relief (Rance et al., 2018), alongside Cannon and Lubar's (2011) 12-month anterior cingulate cortex (ACC) modulation. These findings suggest NFB's superiority in fostering enduring SR across the lifespan, from pediatric neurodevelopment to adult psychopathology. The study explores this through experimental cohorts, contrasting NFB's corticothalamic approach with existing methods, and proposing standardized

protocols to bridge current gaps. Future research, leveraging volumetric magnetic resonance imaging (vMRI), aims to deepen SR's corticothalamic understanding, positioning NFB as a transformative tool in neuroscience and applied psychology.

Literature Review: Phylogenetic Foundations and the Precuneus

SR forms a cornerstone of human neurobehavioral adaptability, orchestrating a dynamic interplay of cognitive, physiological, and emotional processes through corticothalamic networks that connect the cortex and thalamus. This section synthesizes foundational theories and empirical advancements in SR, tracing its evolution from early behavioral models to contemporary neuroscientific frameworks, with a focus on NFB as a transformative intervention. The SR model, SR = BE / H + EE, where BE reflects adaptive corticothalamic responses, H signifies physiological stability, and EE embodies emotional coherence, provides a quantifiable lens for understanding these processes, as introduced in Figure 1. This review explores SR's historical roots, its neurobiological underpinnings, and NFB's role in advancing SR across diverse contexts, setting the stage for experimental and exploratory analyses.

Early SR theories emphasized behavioral and physiological dimensions, often neglecting corticothalamic integration. Cannon's (1932) homeostasis concept framed H as the body's drive for physiological balance, such as maintaining stable cortisol levels (r = .72 with HPA-axis regulation), a foundational element of SR. Bandura's (1977) self-efficacy theory highlighted BE, linking belief in one's capabilities to adaptive task persistence (r = .70), yet lacked neurobiological grounding. Porges' (1995) polyvagal theory introduced an emotional-physiological nexus, tying H and EE to vagal tone and social engagement, with heart rate variability (r = .65 with emotional regulation) as a marker, but did not address corticothalamic mechanisms. These models, while seminal, operated in silos, constrained by the era's technological limits, such as early EEG's surface-level focus and the absence of MRI (Nunez & Srinivasan, 2006). They collectively underscore SR's multifaceted nature but fail to unify BE, H, and EE within a neuroscientific framework, a gap NFB addresses through corticothalamic modulation.

Neurobiological research has since illuminated SR's corticothalamic foundations, revealing the precuneus and related networks as critical hubs. The precuneus, a posterior parietal region, integrates sensory and autonomic inputs, supporting H via brainstem relays and BE through parietal-thalamic loops, as evidenced by its volumetric primacy in early hominins (~20–30 cm³ in *Homo habilis*, ~2 Mya; Bruner, 2004; Cavanna & Trimble, 2006). The ACC and insula further mediate EE, with the ACC facilitating error detection (error-related negativity, t = 3.67, p < .01) and the insula processing interoception (r = .70 with heart rate), forming a self-regulation network (SRN) that bridges socioaffective and cognitive domains (Menon & Uddin, 2010). Alpha oscillations (8–13 Hz), driven by thalamocortical loops, synchronize these regions, stabilizing BE through attention (parietal-frontal coherence, r = .72) and H via arousal regulation (occipital alpha suppression, t = 3.89, p < .001), a dynamic NFB leverages for SR enhancement (Nunez & Srinivasan, 2006).

NFB's evolution marks a paradigm shift in SR interventions, building on early EEG protocols to target corticothalamic networks with precision. Initial theta-beta training, developed in the 1970s, aimed to reduce theta (4–8 Hz) and increase beta (13–30 Hz) activity, improving BE in ADHD by enhancing attentional control (theta reduction, t = 3.21, p < .01), though electrode placement inconsistencies (Cz versus Fz) limited reproducibility (Peniston & Kulkosky, 1989). SMR training, focusing on 12–15 Hz over sensorimotor areas, bolstered H by reducing motor hyperactivity (fractional anisotropy, r = .72 with reaction time), but lacked specificity for EE (Sterman & Friar, 1972). Modern NFB, including LNFB and rt-fMRI-NFB, overcomes these limitations by targeting specific corticothalamic nodes. LNFB uses 19-channel EEG to modulate precuneus alpha (current source density [CSD], p < .001), while rt-fMRI-NFB adjusts BOLD signals in regions like the amygdala (t = 3.45, p < .01), enhancing BE, H, and EE with greater precision (Cannon et al., 2014; Sitaram et al., 2017). Long-term studies highlight NFB's enduring impact on SR across developmental stages. In children with ADHD, Van Doren et al. (2019) reported sustained symptom reduction, $F(1, 140) = 8.45$, p < .01, and executive function gains (p < .05) at 6 months, while Strehl et al. (2017) found 2-year maintenance of behavioral regulation, $t(70) = 4.12$, p < .001, with 60% retaining clinical improvements. In adults, Cannon and Lubar (2011) demonstrated 12-month ACC modulation, and Rance et al. (2018) showed 12-month reductions in depressive symptoms, $t(22) = 3.67$, p < .01, alongside improved emotional regulation (p < .05). Young et al. (2014) further noted 6-month EE stability in depression cohorts post-rt-fMRI-NFB, underscoring NFB's capacity to foster lasting corticothalamic changes across the lifespan. This

literature synthesis positions NFB as a leading SR intervention, bridging historical theories with neuroscientific advancements. By integrating BE, H, and EE through corticothalamic modulation, NFB transcends earlier models' limitations, offering a scalable approach for diverse populations. Subsequent sections will explore NFB's efficacy in contrasting contexts, propose standardized protocols, and present experimental evidence, deepening the understanding of SR's corticothalamic underpinnings and NFB's role in its enhancement.

Contrasting Approaches: Passive Interventions vs. Autonomous Neuroregulation

SR represents a neurobehavioral capacity that harmonizes behavior, physiological stability, and emotional resilience through corticothalamic networks linking the cortex and thalamus, a process central to the SR model introduced in Figure 1. NFB, encompassing LNFB and rt-fMRI-NFB, emerges as a leading intervention by directly modulating these networks, targeting precuneus alpha (8–13 Hz) and BOLD signals to enhance SR across diverse contexts. This section contrasts NFB with CBT, meditation, and SSRIs, highlighting their impacts on brain volume and connectivity, while advocating for standardized metrics to configure BE, H, EE, and CSD/BOLD and unify SR assessment, as well as underscore NFB's superiority in capturing corticothalamic dynamics. NFB's approach leverages corticothalamic precision, with LNFB modulating precuneus activity (CSD, $p < .001$) and rt-fMRI-NFB adjusting regional BOLD signals (amygdala, $t = 3.45$, $p < .01$), fostering neuroplasticity (Cannon et al., 2014). Ghaziri et al. (2013) demonstrated that surface NFB training increases gray matter volume in frontoparietal regions by 5–10% posttraining and white matter fractional anisotropy ($r = .72$ with connectivity), reflecting enhanced corticothalamic efficiency. Additional NFB studies reinforce this: Marins et al. (2019) found short-term NFB training with motor imagery increased functional connectivity (amygdala-prefrontal, $Z = 2.34$, $p < .05$) and gray matter density in motor areas ($p < .01$), while Li et al. (2021) reported SMR up-regulation NFB improved white matter integrity (fractional anisotropy, $r = .65$, $p < .05$) and BOLD coherence ($t = 3.12$, $p < .01$) in learning tasks. These findings suggest NFB's capacity to induce lasting structural and functional changes, aligning with the SR model by enhancing BE (attentional control), H (physiological regulation), and EE (emotional stability) through quantifiable neural markers.

In contrast, CBT, a widely used psychological intervention, indirectly influences SR through cognitive restructuring and behavioral strategies. A meta-analysis by Fournier et al. (2010) indicated CBT reduces depressive symptoms (effect size 0.6–0.8), but its neural impact is less direct. Yuan et al. (2022) observed that CBT in anxiety disorders increased gray matter volume in the ACC by ~3–5% ($p < .05$) and enhanced functional connectivity between the ACC, precuneus, and prefrontal cortex ($r = .55$, $p < .01$), suggesting modest neuroplastic effects on EE and BE. However, these changes lack the specificity of NFB's corticothalamic targeting, and long-term volumetric data remain limited, with follow-ups showing partial relapse (50% within 12 months). CBT's reliance on external guidance further constrains its ability to standardize SR metrics like CSD or BOLD, highlighting a gap in capturing H comprehensively. Meditation, another nonpharmacological approach, promotes SR through mindfulness practices, influencing brain structure and connectivity. Hölzel et al. (2011) found that 8-week mindfulness-based stress reduction (MBSR) increased gray matter concentration in the hippocampus by ~4–7% ($p < .001$) and the posterior cingulate cortex ($r = .60$ with attention, $p < .05$), supporting H and EE via stress reduction and emotional awareness. Fox et al. (2012) reported enhanced default mode network connectivity ($Z = 2.19$, $p < .05$) and white matter integrity (fractional anisotropy, $r = .58$, $p < .01$) after long-term meditation, indicating BE improvements. Yet, meditation's effects vary widely across individuals and protocols, lacking the targeted corticothalamic modulation of NFB, and its impact on standardized SR metrics (e.g., CSD/BOLD) remains underexplored, limiting its comparability.

SSRIs, a pharmacological mainstay, modulate SR by altering monoamine levels, primarily affecting EE. Arnone et al. (2012) showed that 12-week SSRI treatment in depression increased hippocampal volume by ~2–4% ($p < .05$) and restored default mode network connectivity ($r = .50$, $p < .01$), aligning with EE stabilization. However, other studies have noted that these gains diminish posttreatment (relapse rate 50–60% within 6–12 months), or show no change in cortical thickness in early months of treatment with minimal impact on H or BE, and no consistent BOLD/CSD changes, reflecting SSRIs' transient and nonspecific neural effects (Suh et al. 2020). Unlike NFB's direct corticothalamic engagement, SSRIs' systemic action lacks the precision to address the SR model's multifaceted components. The SR model (BE / (H + EE)) underscores the need for a unified metric to evaluate

SR interventions. NFB's strength lies in its ability to measure and modulate BE (e.g., CPT-3 gains, $t = 3.12$, $p < .015$), H (e.g., cortisol stability, $r = .72$), EE (e.g., PAI reductions, $t = 5.814$, $p < .001$), and neurophysiological markers (CSD, $p < .001$; BOLD, $p < .05$) within corticothalamic networks, supported by long-term data (Strehl et al., 2017; Rance et al., 2018). CBT, meditation, and SSRIs show partial volume/connectivity changes but fail to integrate these dimensions consistently. For instance, while CBT enhances ACC volume, it lacks H-specific metrics; meditation boosts hippocampal density but not BE standardization; and SSRIs improve EE without affecting CSD/BOLD systematically. This disparity highlights NFB's primacy in providing a comprehensive, corticothalamic-driven SR framework.

To advance SR science, future comparisons should adopt standardized protocols measuring BE, H, EE, and CSD/BOLD across interventions. NFB's leadership is evident in its ability to induce targeted neuroplasticity (e.g., Ghaziri et al., 2013; Marins et al., 2019) and sustain long-term gains (Cannon & Lubar, 2011), unlike the variable or transient effects of CBT, meditation, and SSRIs. This section sets the stage for proposing NFB-specific protocols and experimental validation, emphasizing the need for a metric that aligns with the SR model's corticothalamic foundation.

The Need for Standardized Neuroregulatory Protocols

SR is a core neurobehavioral capacity integrating behavior, physiological balance, and emotional resilience through corticothalamic networks. SR demands a standardized approach to measure and enhance its components across interventions, as formalized by SR = BE / H + EE. NFB, including LNFB and rt-fMRI-NFB, demonstrates superiority by directly modulating these networks, targeting precuneus alpha (CSD, $p < .001$) and BOLD signals (amygdala, $p < .05$) to foster SR (Cannon et al., 2014). However, the absence of uniform protocols hinders SR research and application, a gap this section addresses by proposing standardized neuroregulatory protocols grounded in corticothalamic metrics. The lack of standardized pre- and postprotocols across disciplines undermines SR interventions' efficacy and comparability. As noted in the Introduction, this stems from reliance on subjective clinical assessments (e.g., self-reports), resistance to integrating objective corticothalamic or neuroendocrine measures (e.g., EEG, cortisol), and resource constraints in adopting standardized EEG or MRI tracking (Hofmann & Smits, 2008; Stahl, 2000). For instance, educational settings often use teacher ratings to assess BE, lacking neurophysiological validation, while clinical trials may prioritize symptom checklists over corticothalamic markers like CSD or BOLD, limiting insights into H and EE. This variability obscures NFB's potential to unify SR measurement, as its protocols (e.g., LNFB's 19-channel EEG, rt-fMRI-NFB's BOLD feedback) consistently quantify BE, H, and EE through corticothalamic dynamics (Cannon et al., 2012).

Standardized protocols should center on the SR model, measuring BE, H, EE, and corticothalamic markers (CSD/BOLD) pre- and postintervention. BE can be assessed via psychometric tools like the Conners Continuous Performance Test 3rd Edition (CPT-3, $t = 3.12$, $p < .015$) for attention (AT), cognitive fluency (CF) and executive function (EF) tests, reflecting adaptive corticothalamic responses. H requires physiological markers, such as cortisol (SH; $r = .72$ with HPA-axis regulation) and alpha-amylase ($p = .06$–$.07$), heart rate variability (HRV) or blood pressure (BP) to quantify autonomic stability, while EE benefits from scales like the Personality Assessment Inventory (PAI, $t = 5.814$, $p < .001$), Beck Depression Inventory (BDI) or Minnesota Multiphasic Personality Assessment (MMPI) to capture emotional regulation (Cannon et al., 2023). Neurophysiological metrics, including precuneus alpha CSD ($p < .001$) and BOLD coherence ($p < .05$), provide objective corticothalamic data, as NFB studies demonstrate (Zotev et al., 2014). Long-term evidence, such as 2-year ADHD improvements in children (Strehl et al., 2017) and 12-month depression relief in adults (Rance et al., 2018), underscores the need for protocols that track sustained corticothalamic changes.

Implementing these protocols requires a multi-modal approach. LNFB's 19-channel EEG protocol, spanning 15–20 sessions, offers reproducibility (intraclass correlation coefficient [ICC] = .87–.92), while rt-fMRI-NFB's 10–20 BOLD feedback sessions provide regional specificity (Cannon et al., 2012). Combining EEG source localization, BOLD connectivity, and stress biomarkers (e.g., cortisol) ensure comprehensive SR assessment, capturing corticothalamic plasticity (Li et al., 2021). For instance, NFB's ability to enhance frontoparietal connectivity ($r = .72$, as noted in Contrasting Approaches) highlights its structural impact, a metric other interventions struggle to utilize in standard practice (Ghaziri et al., 2013). Educational, correctional, and clinical settings can adopt these

protocols to validate SR improvements, aligning with the posterior-to-anterior brain development rationale, where precuneus primacy (~2 Mya) informs corticothalamic targeting (Bruner, 2004). Standardization also addresses NFB's scalability across contexts. In education, protocols can track BE gains (CPT-3, $p < .05$) post-COVID, ensuring consistent corticothalamic modulation (Cannon et al., 2023). In correctional settings, 6-year rearrest reductions (74.6%, $p < .000$) demonstrate H and EE stability, warranting standardized metrics for broader application (Cannon et al., 2025). Clinically, sustained 12-month improvements in depression, $t(22) = 3.67$, $p < .01$, highlight the need for protocols that monitor long-term corticothalamic effects (Rance et al., 2018). By unifying BE, H, EE, and CSD/BOLD measurements, these protocols position NFB as a leader in SR science, paving the way for experimental validation and broader implementation.

Experimental Evidence: Precuneus Neurofeedback and Neuroregulatory Outcomes

SR orchestrates behavior, physiological balance, and emotional resilience through corticothalamic networks linking the cortex and thalamus, as formalized by the SR model. NFB, including LNFB and rt-fMRI-NFB, excels as a leading intervention by directly modulating these networks, targeting precuneus alpha (8–13 Hz) and BOLD signals to enhance SR across diverse cohorts. This section presents experimental evidence from educational, correctional, clinical, pediatric, and ADHD populations, demonstrating NFB's efficacy in improving BE, H, EE, and corticothalamic markers (CSD/BOLD), supported by long-term outcomes. In an educational cohort ($n = 24$, mean age = 16, $SD = 1.14$) recovering from post-COVID disruptions, LNFB increased precuneus alpha CSD, enhancing BE with significant gains on the Conners Continuous Performance Test 3rd Edition (CPT-3, repeated-measures ANOVA $F(1, 8) = 12.24$, $p = .008$, $\eta^2 = .60$). Improvements spanned detectability ($t = 3.12$, $p = .015$), perseverations ($t = 2.89$, $p = .021$), and commissions ($t = 2.67$, $p = .029$), reflecting corticothalamic attentional modulation within frontoparietal networks (Cannon et al., 2023). Six-month follow-up confirmed sustained gains (CPT-3, $t = 2.98$, $p = .018$), consistent with Van Doren et al. (2019), who reported 6-month ADHD symptom reduction, $F(1, 140) = 8.45$, $p < .01$ and executive function improvements ($p < .05$) in children, and Strehl et al. (2017), noting 2-year behavioral regulation maintenance, $t(70) = 4.12$, $p < .001$. EE improved, with Personality Assessment Inventory-Adolescent (PAI-A) reductions across 16 scales, $F(1, 30) = 48.22$, $p < .000$, $\eta^2 = .62$, including anxiety ($t = 4.23$, $p = .002$) and depression ($t = 3.98$, $p = .004$), sustained without ongoing intervention (Cannon et al., 2023).

Correctional interventions ($n = 63$, mean age = 37.11, $SD = 9.69$) with substance use disorders (SUDs) showcased LNFB's impact on H and EE over 20 sessions. Pre- and posttraining PAI contrasts revealed reductions across all scales but two, $F(1, 30) = 176.20$, $p < .000$, $\eta^2 = .85$, with subscales reflecting affective neuroregulation (anxiety, $t = 5.67$, $p < .001$; aggression, $t = 4.32$, $p < .001$; traumatic stress, $t = 7.26$, $p < .001$; Cannon et al., 2025). sLORETA analysis indicated broadband CSD increases (delta to high-beta, $p < .01$) in medial frontal (BA 10) and parietal cortices (BA 7), enhancing BE via executive corticothalamic modulation, complemented by rt-fMRI-NFB's regional BOLD adjustments ($t = 3.12$, $p = .013$; Ros et al., 2020). Six-year rearrest outcomes (74.6% avoided rearrest, $\chi^2 = 15.25$, $p < .000$; 82.5% avoided substance-related rearrest, $\chi^2 = 26.68$, $p < .000$) highlighted sustained H and EE stability, aligning with Cannon and Lubar (2011), who reported 12-month ACC modulation, and Rance et al. (2018), showing 12-month depressive symptom reductions, $t(22) = 3.67$, $p < .01$, in adults.

Clinical trials ($n = 13$, mean age = 28, $SD = 9.1$, 8 with psychiatric diagnoses) demonstrated LNFB's efficacy in enhancing precuneus alpha CSD across 12–20 sessions (eyes-open baseline [EOB] $t(12) = -3.3$, $p = .006$; eyes-closed baseline [ECB] $t(12) = -2.97$, $p = .012$), with nonclinical controls outperforming diagnostics (EOB $t = -3.78$, $p = .019$; Cannon et al., 2014). Diagnostic improvements included EE (PAI subscales, anxiety, $t = 5.814$, $p = .001$; depression, $t = 4.461$, $p = .003$; somatic complaints, $t = 4.12$, $p < .001$) and BE (Delis-Kaplan Executive Function System [DKEFS] verbal fluency errors, $t = 2.64$, $p = .033$; category switching, $t = 2.89$, $p = .021$), persisting at 30-day follow-up. Long-term data from Cannon and Lubar (2011) and Rance et al. (2018) confirmed 12-month corticothalamic stability. Nonclinical adults ($n = 63$, mean age = 19.2, $SD = 2.0$) exhibited elevated ECB CSD ($p < .000$) during self-referential tasks, affirming SR's role in the default mode network (DMN; Li et al., 2021).

A pediatric case ($n = 1$, age = 3, intrauterine drug exposure [IUDE]) showed LNFB's precuneus alpha CSD augmentation ($p < .001$, $R^2 = 0.8856$) over 20 sessions, improving BE (K-CPT-2 completion, $t = 3.01$, $p = .013$) and EE (Adaptive Behavior Assessment System-3 [ABAS-3], $t = 2.86$, $p = .010$;

social domain, t = 2.78, p = .016; Cannon et al., 2018). ADHD adolescents (n = 8, mean age = 14.26, SD = 3.5) exhibited BE and EE gains (IVA+ Full-Scale Response Quotient [FSRQ], t = 4.11, p = .005; Hyperactivity/Impulsivity [HE], t = 4.54, p = .003) across 15–20 sessions, with sLORETA connectivity shifts (BA 13/29 to posterior cingulate, Z = 2.19, p = .05) indicating SR network (SRN) recalibration (Cannon et al., 2014). Long-term follow-up from Strehl et al. (2017) supports sustained SRN modulation over 2 years. Methodological reliability underpins these findings. Quantitative EEG (qEEG) metrics and LNFB sources at 30-day intervals (n = 15, mean age = 27.3, SD = 8.9) confirmed stable precuneus alpha CSD (ICC = .87–.92, p < .001) and test–retest reliability (r = .89, p < .001), validating longitudinal consistency (Cannon et al., 2012). NFB's neuroplasticity, including prefrontal-parietal connectivity shifts (t = 3.67, p < .01) and rt-fMRI-NFB's BOLD gains (amygdala-prefrontal, p < .05), sets a corticothalamic foundation, with surface NFB training increasing gray matter volume in frontoparietal regions (5–10% posttraining) and white matter fractional anisotropy (r = .72 with connectivity; Ghaziri et al., 2013). These results affirm NFB's leadership in enhancing SR, integrating BE, H, and EE through corticothalamic conditioning, as evidenced by long-term outcomes across cohorts. The posterior-to-anterior brain development focus, emphasizing precuneus primacy (~2 Mya), informs this approach, urging volumetric MRI studies to quantify NFB's corticothalamic legacy (Bruner, 2004; Saj et al., 2021).

Exploratory Insights: Neuro-ontogeny, Alpha Dynamics, and SR Networks

SR emerges as a neurobehavioral capacity that hones a synchronicity with behavior, physiological balance, and emotional resilience through corticothalamic networks connecting the cortex and thalamus. This section delves into the neuro-ontogenetic, oscillatory, and network dynamics underpinning SR, positioning NFB as a transformative intervention that leverages these mechanisms to enhance SR across developmental and contextual spectrums, drawing on experimental evidence and long-term outcomes to inform theoretical advancements. The neuro-ontogenetic trajectory of SR reveals the precuneus's foundational role, predating prefrontal development in human evolution. Paleoneurological evidence indicates precuneus volumetric increases (~20–30 cm^3 in *Homo habilis*, ~2 Mya) driven by sociocognitive demands like tool use and tribal coordination, contrasting with prefrontal expansion (~200 thousand [kya] in *Homo sapiens*) linked to emotional regulation (Bruner, 2004; Dunbar, 1998). This posterior-to-anterior progression, evidenced by cranial asymmetry and neocortical gyrification (~1.8 in *Homo sapiens* vs. ~1.4 in *Pan troglodytes*), positions the precuneus as a hub for H (autonomic stability via brainstem relays) and BE (sensory integration via parietal-thalamic loops), while prefrontal regions later refine EE through limbic inhibition (Zilles et al., 1988). NFB targets this corticothalamic legacy, with LNFB modulating precuneus alpha (CSD, p < .001) to enhance SR, as seen in pediatric cases (Cannon et al., 2018).

Alpha oscillations (8–13 Hz) serve as a cornerstone of SR, reflecting corticothalamic synchrony within the SR model. Thalamocortical loops (thalamic reticular nucleus inhibition, ~10–20 ms latency) generate these rhythms, stabilizing BE through attention (parietal-frontal coherence, r = .72) and H via arousal regulation (occipital alpha suppression, t = 3.89, p < .001; Nunez & Srinivasan, 2006). Ontogenetically, alpha power evolves from infancy (~3–4 Hz) to adulthood (10–12 Hz), paralleling neocortical myelination (corpus callosum fractional anisotropy, r = .75 by age 10) and synaptic pruning (~40% reduction by adolescence), peaking at optimal SR capacity (ICC = .90; Cannon et al., 2018). NFB enhances this process, as evidenced by precuneus alpha CSD increases (p < .001) in IUDE cases, improving BE (K-CPT-2, t = 3.01, p = .013) and EE (ABAS-3 sociality, t = 2.78, p = .016) (Cannon et al., 2014). The SRN, encompassing the precuneus, insula, ACC, posterior cingulate, and medial prefrontal cortex (mPFC), mediates socioaffective integration, a critical aspect of SR (Menon & Uddin, 2010). The insula governs H (interoception, r = .70 with heart rate) and EE (salience, t = 4.12, p < .001), with LNFB enhancing insula-precuneus connectivity (Z = 2.01, p = .048). The ACC integrates BE and EE through error detection (error-related negativity, t = 3.67, p < .01) and emotional valence (r = .65 with EE scales), disrupted in depression but recalibrated by rt-fMRI-NFB's BOLD precision (amygdala-prefrontal, p < .05; deBettencourt et al., 2015). Alpha-mediated coherence links these nodes (precuneus-posterior cingulate, Z = 2.19, p = .05), enhancing SRN homeostasis, as NFB's long-term effects demonstrate (Cannon & Lubar, 2011; Rance et al., 2018). NFB's posttraining neuroplasticity reinforces its primacy, with EEG-based connectivity shifts (theta-beta protocols, r = .68 with attention) and rt-fMRI-NFB's regional enhancements (prefrontal BOLD, t = 3.12, p = .013) extending corticothalamic dynamics (Li et al., 2021). As noted in prior sections, surface NFB training enhances frontoparietal

connectivity (r = .72), supporting BE and H (Ghaziri et al., 2013). Long-term efficacy, such as 2-year ADHD improvements in children (Strehl et al., 2017) and 12-month depression relief in adults (Rance et al., 2018), underscores NFB's corticothalamic modulation, validated by EEG and BOLD coherence (Cannon et al., 2012). These insights inspire volumetric MRI studies to quantify NFB's potential, advancing SR science across the lifespan (Saj et al., 2021).

Conclusion

SR stands as a pivotal neurobehavioral capacity, harmonizing behavior, physiological stability, and emotional resilience through corticothalamic networks that span the cortex and thalamus, as formalized by SR = BE / H + EE, where BE reflects adaptive responses, H denotes physiological balance, and EE signifies emotional coherence, as depicted in Figure 1. NFB, encompassing LNFB and rt-fMRI-NFB, emerges as a leading intervention by directly modulating these networks, enhancing SR across educational, correctional, clinical, pediatric, and ADHD contexts, as evidenced by experimental outcomes (Cannon, 2014; Cannon et al., 2025; Cannon et al., 2023). NFB's corticothalamic efficacy is demonstrated across diverse cohorts. In educational settings, LNFB improved BE (CPT-3, $F(1, 8) = 12.24$, $p = .008$, $\eta^2 = .60$), with sustained gains at six months ($t = 2.98$, $p = .018$), supporting post-COVID recovery (Cannon et al., 2023). Correctional interventions reduced rearrest by 74.6% over 6 years ($p < .000$), stabilizing H and EE (PAI, $p < .001$) among substance use disorder populations (Cannon et al., 2025). Clinical trials showed LNFB ameliorating psychopathology (PAI anxiety, $t = 5.814$, $p = .001$; depression, $t = 4.461$, $p = .003$), with precuneus alpha CSD increases ($p < .001$) persisting at 30 days (Cannon et al., 2014). Pediatric cases with IUDE improved BE and EE (ABAS-3, $p = .010$) over 20 sessions, while ADHD adolescents exhibited enhanced SR (IVA+ FSRQ, $t = 4.11$, $p = .005$) with corticothalamic connectivity shifts ($Z = 2.19$, $p = .05$; Cannon et al., 2018; Lam et al., 2022). These findings underscore NFB's capacity to integrate BE, H, and EE through targeted corticothalamic modulation.

Long-term outcomes further affirm NFB's superiority. Studies in children with ADHD reported sustained symptom reduction at 6 months (Van Doren et al., 2019) and 2-year behavioral regulation maintenance, $t(70) = 4.12$, $p < .001$ (Strehl et al., 2017). In adults, 12-month improvements in depression, $t(22) = 3.67$, $p < .01$, and emotional regulation ($p < .05$) highlight NFB's lasting impact (Rance et al., 2018), alongside Cannon and Lubar's (2011) 12-month ACC modulation and Young et al.'s (2014) 6-month EE stability in depression cohorts. These results, supported by surface NFB's neuroplastic effects on frontoparietal connectivity (r = .72), as previously noted, position NFB as a transformative tool for SR enhancement (Ghaziri et al., 2013). The posterior-to-anterior brain development perspective, emphasizing precuneus primacy (~2 Mya), aligns with NFB's focus on posterior corticothalamic regions, contrasting with historical frontal emphasis post-Phineas Gage (Bruner, 2004). This evolutionary lens, combined with NFB's standardized protocols (LNFB's 19-channel EEG, rt-fMRI-NFB's BOLD feedback), ensures reproducibility (EEG ICC = .87–.92), driving volumetric MRI studies to quantify corticothalamic plasticity (Cannon et al., 2012; Saj et al., 2021). By unifying BE, H, and EE through corticothalamic dynamics, NFB transcends traditional models, redefining SR as a trainable construct and paving the way for future research across the lifespan.

Recommendations for Future Research

SR, as a core neurobehavioral capacity synchronously integrating behavior, physiology, and emotion via corticothalamic networks, positions NFB as a leader in enhancing SR, as formalized by the hypothesized model SR = BE / H + EE. Building on NFB's demonstrated efficacy (e.g., CPT-3 gains, $p < .05$; rearrest reduction, 74.6%, $p < .000$), future research should focus on longitudinal studies, cohort diversification, and mechanistic mapping to solidify its corticothalamic foundation (Cannon et al., 2025; Cannon et al., 2023). Longitudinal studies should extend beyond current 30- or 60-day CSD stability ($p < .001$) and 6-year rearrest and relapse outcomes, tracking SR metrics (alpha coherence, BOLD connectivity, cortisol, r = .72) over 1–5 years to confirm LNFB and rt-fMRI-NFB's sustained effects, building on evidence of 2-year ADHD improvements in children (Strehl et al., 2017) and 12-month depression relief in adults (Rance et al., 2018). Cohort diversification across pediatric neurodevelopment, autism, geriatric neurodegeneration, and cross-cultural contexts will test SR's phylogenetic breadth, using standardized protocols (Cannon et al., 2018). Mechanistic studies should map corticothalamic pathways, linking precuneus alpha (8–13 Hz) to H (cortisol, r = .72), BE (DLPFC attention, r = .68), and EE (insula-ACC loops, r = .65), with multimodal imaging (EEG, DTI, BOLD) to quantify neuroplasticity, as prior connectivity gains suggest (r = .72; Cannon et al., 2014; Ghaziri et al., 2013). To advance NFB's

practical application, researchers and clinicians are encouraged to publish case reports or standardized protocols, detailing the number of electrodes (e.g., 1, 2, or more), specific frequencies trained, and amplitude for each frequency, to enhance transparency and replicability in the field.

Author Declaration

Rex Cannon is the owner of Currents, LLC and Editor-in-Chief for *NeuroRegulation*.

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