

NeuroRegulation



The Official Journal of



Volume 13, Number 3, 2026

NeuroRegulation

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NeuroRegulation (ISSN: 2373-0587) is published quarterly by the International Society for Neuroregulation and Research (ISNR), 2146 Roswell Road, Suite 108, PMB 736, Marietta, GA 30062, USA.

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Aim and Scope

NeuroRegulation is a peer-reviewed journal providing an integrated, multidisciplinary perspective on clinically relevant research, treatment, and public policy for neurofeedback, neuroregulation, and neurotherapy. The journal reviews important findings in clinical neurotherapy, biofeedback, and electroencephalography for use in assessing baselines and outcomes of various procedures. The journal draws from expertise inside and outside of the International Society for Neuroregulation and Research to deliver material which integrates the diverse aspects of the field. Instructions for submissions and Author Guidelines can be found on the journal website (<http://www.neuroregulation.org>).

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The Effects of Alpha-Theta EEG Biofeedback Training on Anxiety and Stress: Novel Use of Hair Cortisol Concentration as a Biomarker

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Abstract

Electroencephalographic (EEG) biofeedback is a widely used nonpharmaceutical intervention for enhancing human functioning. This pilot study examined whether 24 sessions of alpha-theta (AT) training would reduce stress, as measured by hair cortisol concentration (HCC) and subjective anxiety scales, in 11 adults with generalized anxiety disorder (GAD). Pre- and postintervention measures included the State-Trait Anxiety Inventory (STAI), the Perceived Stress Scale (PSS), and HCC from hair samples. A novel 5-point Likert scale assessed five areas of functionality. Wilcoxon signed-rank tests revealed significant decreases in STAI and PSS scores ($p < .01$), with large effect sizes ($d = 1.174$; $d = 1.655$). Likert responses were positive, with most improvement in resilience and emotional status. Eight participants showed decreased postintervention HCC, though overall changes for $N = 10$ were not statistically significant ($p > .01$), as they included participants who did not respond to the training. The magnitude of HCC change indicated a moderate effect size ($d = 0.62$). In responders, HCC reduction was significant ($p = .006$) and linked to higher crossover percentages, suggesting deeper AT learning may lower cortisol. This is the first study to show the potential of AT training as a nonmedication approach to anxiety and stress reduction, with HCC being a novel objective biomarker.

Keywords: neurofeedback; alpha-theta training; hair cortisol concentration; stress biomarker; anxiety

Citation: Zlatkova, K., Kerson, C., Sherman, R., & Bendinskas, K. (2026). The effects of alpha-theta EEG biofeedback training on anxiety and stress: Novel use of hair cortisol concentration as a biomarker. *NeuroRegulation*, 13(3), 220–238. <https://doi.org/10.15540/nr.13.3.220>

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Stress is essential for a healthy life because it promotes goal-directed behavior and motivation. However, when stress exceeds the optimal levels, it can lead to feelings of overwhelm and conditions such as generalized anxiety disorder (GAD). The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; APA, 2013) defines GAD as excessive worry occurring more days than not for at least 6 months and interfering with daily activities. It affects an estimated 7 million adults every year; women are two times more likely to be affected by anxiety than men (Anxiety and Depression Association of America, 2022). People with GAD suffer from prolonged and uncontrollable worry, uneasiness, tension, insomnia, distractibility, and irritability, which can be detrimental to everyday

activities, social life, and academics (Abdian et al., 2021). Anxiety and stress are commonly assessed via the State-Trait Anxiety Inventory (STAI) and the Perceived Stress Scale (PSS), both shown to be valid and reliable psychometric instruments (Thomas & Cassady, 2021).

The conventional approach to treating anxiety includes psychotherapy or pharmacotherapy, or both. Among nonpharmaceutical interventions, cognitive behavioral therapy (CBT) has demonstrated efficacy. In a 2005 study by Linden et al. ($N = 72$), 25 CBT sessions led to 14.6% and 11.6% reductions in STAI scores in two groups. It seems valuable to examine how other

nonpharmaceutical interventions, such as EEG biofeedback, compare in effectiveness.

Physiological and behavioral effects of stress are well studied. McEwen (2007) explains that responses to stress promote allostasis, an adaptive process which, through chemical messengers of the endocrine system, allows the body to return to a state of homeostasis. However, as the body strives to maintain homeostasis, prolonged stress can lead to allostatic load (“wear and tear”) and dysregulation within multiple organs (McEwen, 1998). Among the many things chronic stress has been linked to are decreased immunity, inflammation in the body, neural excitotoxicity, adipose fat accumulation, compromised arterial function, alteration of the gut microbiome, and telomere shortening (Chang et al., 2024; Shonkoff et al., 2012).

Cortisol

One way to examine the impact of stress and anxiety on the organism is to assess the function of the hypothalamic-pituitary-adrenal (HPA) axis. During stressful times, neurons of the hypothalamic paraventricular nucleus activate corticotropin and arginine vasopressin (AVP) secretion. These hormones influence the frontal lobe of the pituitary gland, leading to the release of glucocorticoid (cortisol) from the adrenal glands (Bozovic et al., 2013). Typically, cortisol downregulates the HPA activity, and the system returns to its homeostasis. When cortisol levels in the blood remain elevated over time, it indicates the body is unable to disengage from the stress response, suggesting a state of chronic stress or dysregulation. While acute stress responses may have an adaptive role, prolonged cortisol production may be harmful and lead to illness (Chrousos, 2009). Overactivation of the HPA axis can lead to prolonged elevation of cortisol levels, which in turn can exacerbate anxiety symptoms (Lenze et al., 2011).

Thus, cortisol is considered a reliable stress response biomarker and can be measured via biological samples such as saliva, blood, urine, and hair strands (Noushad et al., 2021). Hair cortisol concentration (HCC) analysis has become the preferred method for long-term examination of cortisol changes over several months (Abell et al., 2016). HCC is considered a reliable and valid biomarker of long-term stress response, with strong test–retest reliability and low intraindividual variability over time (Sauvé et al., 2007; Stalder et al., 2017). Hair strand collection can be conveniently performed in an outpatient setting without the need for special preparations. HCC levels in healthy individuals with

low levels of stress typically range between 1–5 pg/mg, while in stressed individuals, they can range between 5–40 pg/mg (Stalder et al., 2017).

Electroencephalography (EEG)

Electroencephalography (EEG) is a method of measuring and recording local field potentials from the scalp and visually presenting the tracings either digitally or on paper. The measured EEG comprises the summation of excitatory postsynaptic potentials (EPSP) and inhibitory postsynaptic potentials (IPSP) from a pool of neurons that fire simultaneously (Smith, 2005). Modern technology, such as the fast Fourier transform (FFT), decomposes the raw EEG into frequency bands for closer investigation. These EEG frequencies, organized by the number of oscillations per second, or hertz (Hz), are generally classified as delta (1–4 Hz), theta (4–8 Hz), alpha (8–11 Hz), beta (12–35 Hz), and gamma (35+ Hz).

EEG Biofeedback

EEG biofeedback, also known as neurofeedback, is a self-regulating technique based on operant conditioning learning (Sherlin et al., 2011). It involves measuring cortical potentials from one’s scalp and feeding the brainwave information back to the subject via specialized equipment. Visual or auditory signals provide positive feedback to increase the likelihood of the desired EEG activity happening again. Hence, neurofeedback teaches self-regulation of brain functioning by measuring EEG (Marzbani et al., 2016; Sherlin et al., 2011).

Alpha EEG

Alpha brainwave (8–12 Hz) was first named in the late 1920s by Hans Berger (Berger, 1929). Produced by the thalamus and predominantly seen in the posterior-occipital areas of the cortex, alpha is commonly found during relaxed and effortless alertness (Zani et al., 2020). In healthy adults, posterior alpha amplitude is around 15–50 μ V (Nunez, 2017). Often referred to as the brain’s idling or resting rhythm, alpha waves are associated with states of flow, meditative calm, disengagement from the external world, and physical relaxation (Kerson, 2017, p. 132).

Theta EEG

Neural oscillatory activity in the theta band is reported as 4–8 Hz. The hippocampus is a key structure involved in the generation and organization of theta activity, as part of a broader medial septal-hippocampal network (Nuñez & Buño, 2021). Further, it is theorized that hippocampal theta is implicated in memory encoding and retrieval (Kropotov, 2009). Considered a normal variant, theta

is noted during wakefulness (Marcuse et al., 2016), drowsy states and is associated with deep relaxation, meditation, and hypnagogic states (Vaitl et al., 2005). Theta is also related to introspection, intuition, and creativity (Kerson, 2017, p. 131).

Alpha-Theta Neurofeedback Training

Alpha-theta (AT) neurofeedback training is a noninvasive therapeutic technique designed to reinforce the production of alpha (8–12 Hz) and theta (4–8 Hz) brainwaves, typically linked to calm and meditative states. The first attempts to train alpha for relaxation occurred in the late 1960s, when Kamiya taught subjects to increase their alpha rhythms through operant conditioning (Kamiya, 1971). Subjects learned to assess their mental state when alpha amplitude rose. Furthermore, they associated these states with relaxation, “letting go of control,” and feeling pleasant (Kamiya, 1971). Theta waves are often explored as a medium for maintaining states of reverie and hypnagogia (Gruzelier, 2009). The concept of AT training was reported in the late 1980s by Peniston and Kulkosky (1989), who incorporated it into their behavioral intervention protocol for addiction. AT training has been widely investigated in both clinical and nonclinical contexts, including the treatment of addiction disorders, posttraumatic stress disorder (PTSD; Peniston & Kulkosky, 1989), chronic alcoholism, and depression (Saxby & Peniston, 1995); enhancement of music and artistic performance (Gruzelier, 2009); and improvement of personality, mood (Raymond et al., 2005), and well-being (Boynton, 2001).

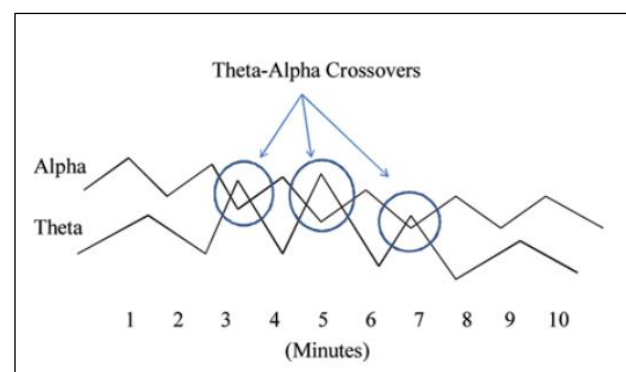
Alpha-Theta Crossover

AT training teaches subjects to enhance alpha and theta amplitudes, typically with eyes closed. Upon eye closure, the visual cortex ceases to receive visual input, synchronizing alpha oscillations and increasing alpha amplitude. As the trainee relaxes, alpha drops, allowing theta to surpass it in amplitude (J. P. Moore et al., 2000). This event is known as the *alpha-theta crossover* (see Figure 1).

The alpha dropout is consistent with the onset of stage 1 sleep and signifies a drop in vigilance (Niedermeyer, 1999). The neurophysiology of the AT training involves “the ascending mesencephalic-cortical arousal system and limbic systems” (Gruzelier, 2009). The crossover results from a complex interplay between the reticular nucleus of the thalamus (nucleus reticularis thalami, nRT), the cortex, the brainstem, and ascending acetylcholine pathways. The nRT is a group of GABAergic cells that surround the dorsal part of the thalamus

(Halassa & Acsády, 2016). Clemente-Perez et al. (2017) describe the nRT as the “the gateway guardian” of the thalamus for its role modulating the thalamocortical interactions that govern functions such as attention, sensation, sleep, and consciousness (Crick, 1984). The nRT is responsible for blocking sensory information from reaching the cortex, facilitating a reduction in external input and allowing theta activity to increase.

Figure 1. *Theta-Alpha Crossovers.*



Note. “The Therapeutic Crossover in A/T Training Neurofeedback: Temporal and Spectral Components of Access to Levels of Consciousness” by M. Johnson & E. Bodenhamer-Davis, in A. Martins-Mourao & C. Kerson (Eds.), *Alpha Theta Neurofeedback Training in the 21st Century: A Handbook for Clinicians and Researchers* (2nd ed., pp. 141–172), 2017, A FNNR Publication. Reprinted with permission by FNNR.

Peniston associated crossover events with hypnagogic imagery, visualization, and states of high suggestibility, attributing therapeutic efficacy to these experiences (Peniston & Kulkosky, 1989). In a different study, Johnson et al. (2013) analyzed 182 crossover events, identifying 69 therapeutic crossovers (associated with the emergence of meaningful visualizations) and 62 nontherapeutic crossovers (lacking any personally relevant material or imagery). However, J. P. Moore et al. (2000) postulated the alpha-theta crossover was controversial and not an essential component for the effectiveness of the intervention. Crossover frequency has been used as an indirect indicator of neurophysiological marker or depth of training during AT sessions in previous studies (Gruzelier, 2009; Peniston & Kulkosky, 1991); however, the reliability and validity of crossover as a measure have not been formally established and standard criteria for defining and quantifying crossovers remain inconsistent.

In the present study, the crossover is considered a significant point in the AT training, reflecting the moment of “letting go of control,” reduced tension, and drop in vigilance. Achieving this state can be particularly challenging for individuals with GAD. Therefore, we view the occurrence of crossovers as an important indicator of successful training.

Alpha-Theta Training for GAD, Stress, and Emotional Regulation

Empirical observations demonstrate that AT training leads to favorable outcomes in stress reduction. Dadashi et al. (2015) employed alpha and theta uptrain protocol in 28 GAD patients. The results from their study showed an increase in alpha and theta amplitudes, which were inversely correlated with symptoms of anxiety.

This was supported by another study by Vanathy et al. (as cited by Dadashi et al., 2015) which trained 15 GAD patients to increase their alpha and theta amplitudes over 20 sessions. The postintervention analysis revealed significant decreases ($p < .05$) in ranked anxiety scores and improved emotional regulation.

Gruzelier (2014) investigated whether AT training and heart rate variability (HRV) training could improve dance performance, cognitive creativity, and self-reported anxiety and depression in 64 contemporary dancers. Participants were randomly placed into one of the four groups: a control group, a choreology group, an AT group, or an HRV group. Compared to the two nonintervention groups, the HRV group and the AT group demonstrated lower anxiety scores.

Bennett et al. (2018) employed AT training as their experimental intervention where 60 participants with traumatic brain injury were randomly assigned to either the AT neurofeedback group or to a treatment-as-usual (TAU) group. Pre- and postsymptom ratings were analyzed, perceived stress was measured via the PSS scale, and cortisol levels were obtained via blood samples. The results demonstrated significant reductions in perceived stress and cortisol levels in the AT group compared to the control group, supporting the efficacy of AT training for mitigating perceived stress in individuals with traumatic brain injury.

Imperator et al. (2017) used exact low-resolution electromagnetic tomography (eLORETA) to examine EEG changes after 10 AT training sessions. They found improved emotional awareness and affect regulation, along with increased alpha and beta

connectivity between key brain regions, including the anterior cingulate, hippocampi, temporal lobes, and posterior cingulate. These changes suggest enhanced introspective mental processing following AT training (Imperator et al., 2017).

Lastly, N. C. Moore's (2005) meta-analysis looked at studies of GAD, phobias, obsessive-compulsive disorder (OCD), and PTSD. He evaluated the efficacy of neurotherapy for these disorders and concluded that enhanced alpha and theta brainwaves can help with mitigating anxiety beyond the placebo effect. Prior research has indicated that AT training shows promise in reducing stress, anxiety, and arousal (Edwards, 2013) while also enhancing subjective mood as measured by the Profile of Mood States (Raymond et al., 2005). Given this context, the present study employed a novel 5-point Likert functionality scale (see Appendix A) to evaluate the effects of AT training across several categories: emotional status, sleep quality, attention span, autonomic nervous system function, and resilience. As this was an exploratory measure, formal reliability and validity testing have not yet been conducted.

EEG Biofeedback and Cortisol Measures

Previous studies examined the effects of EEG biofeedback on salivary cortisol (Costa et al., 2016; Gadea et al., 2020), showing that cortisol levels and anxiety symptoms decreased as the neurofeedback training advanced. Overall, studies have examined the application of AT training in clinical and nonclinical settings, emphasizing its potential role in stress-related disorders. This research has pointed to the positive effects of this intervention in individuals who suffer from anxiety and a lack of emotional well-being (Dadashi et al., 2015). However, the limited focus on the specific impact of AT training on cortisol modulation among individuals with GAD underscores the need for further empirical exploration in this domain. Given the high prevalence of stress-related health issues among individuals with anxiety, it is crucial to examine the potential benefits of AT neurofeedback training on HCC. Although AT neurofeedback has been studied in individuals with GAD, existing research has primarily focused on psychological outcomes or short-term cortisol measures such as saliva. It appears this is the first study to evaluate changes in HCC following alpha-theta training, as it has not been addressed in published studies and there is a noticeable gap in the literature. As a chronic biomarker, HCC allows for the assessment of long-term physiological stress response, offering a novel and more stable measure of treatment response. In

line with the objectives of this pilot study, the following hypotheses were tested:

- H₁: Participants who complete 24 AT neurofeedback training sessions will exhibit a reduction of at least 20% in hair cortisol levels as compared to baseline.
- H₂: After undergoing 24 AT neurofeedback sessions, the participants will demonstrate superior improvements of at least 15% in their subjective assessments of anxiety, stress levels, and perceived well-being, relative to their preintervention evaluations (STAI and PSS).

Material and Methods

This research project was a pilot study with a within-subjects pre–post design. A power analysis based on Glantz (2012) was conducted. Assuming a medium effect size (Cohen's $d = 0.6$), an alpha level of .01, and 80% power, the estimated minimum sample size required was $N = 8$.

Participants

Twelve qualifying participants were recruited from a private neurofeedback and psychotherapy practice

in Des Plaines, IL. Participants were eligible if they were adults (18–65) with a GAD diagnosis and seeking treatment for anxiety and stress management. Individuals were excluded if they had breathing issues, traumatic brain injury, or PTSD; were using psychotropic or recreational drugs, or glucocorticoids; or were undergoing other treatments for anxiety (e.g., other forms of biofeedback or behavioral interventions). One individual withdrew without a specific reason from the study, leaving a final sample of 11 participants, ages 19–52 (see Table 1). The study was IRB approved (1336) and all participants provided informed consent. All participants who completed the study were given a \$50 Amazon gift card.

Study Design

Baseline Measurements. Participants who met the inclusion criteria and were not disqualified by any exclusion criteria completed the informed consent process. Following this, they scheduled AT training sessions at a convenient time for them. At their first session, the participants underwent baseline measurements, which included providing a hair sample and completing the STAI and the PSS. The study procedure is outlined in Figure 2.

Table 1

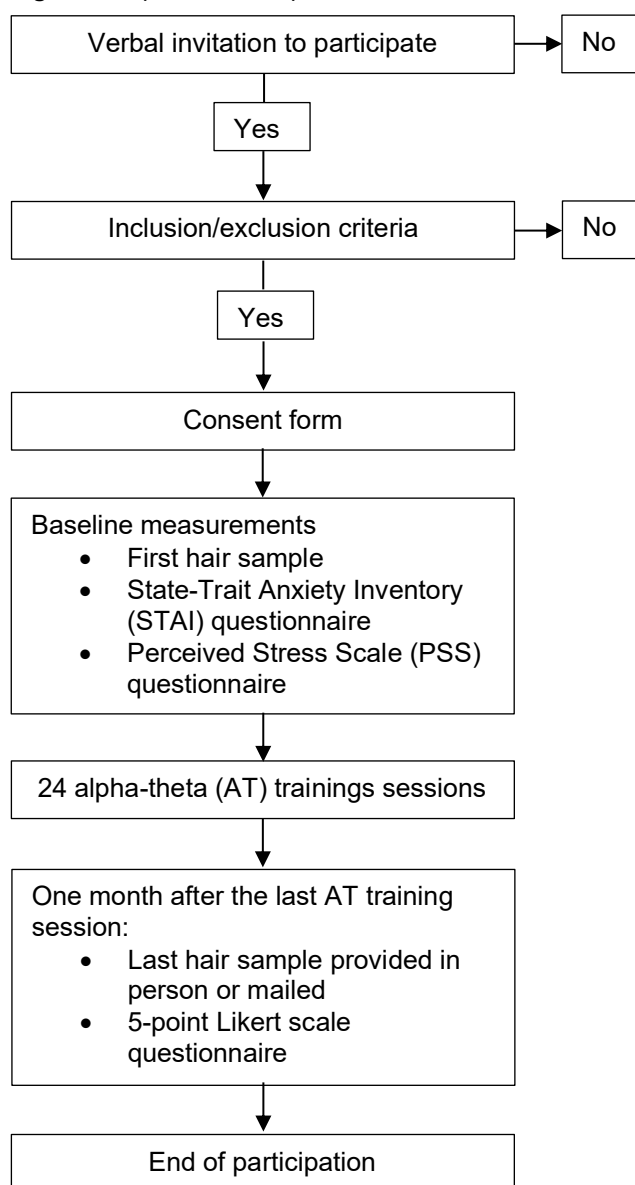
Participants' Demographics (N = 11)

Variable	Age	Gender	Completion Status	Training Length (Weeks)	Session with Crossovers ^a	% of Sessions with Crossovers
AT001	37	F	Y	12	2	8.33
AT002	30	F	Y	13.5	20	83.30
AT003	36	F	Y	12	4	16.70
AT004 ^b	19	F	N	5	–	–
AT005	37	F	Y	13	1	4.20
AT006	29	F	Y	12	21	87.50
AT007	39	F	Y	12	4	16.70
AT008	44	M	Y	12	20	83.30
AT009	43	F	Y	12	19	79.00
AT010	52	F	Y	12	2	8.33
AT011	35	F	Y	12	12	50.00
AT012	44	M	Y	12	15	62.50

Note. Total female participants = 9; total male participants = 2.

^a Total sessions with crossovers = 120.

^b Participant dropped out of study, so $N = 11$.

Figure 2. Important Participation Timeline Points.

Biological Measures. HCC is considered a reliable stress response proxy of long-term HPA axis activity, with evidence supporting good test–retest reliability and low intraindividual variability under stable conditions (Stalder et al., 2017). The first hair sample was collected before the first AT training session along with the other baseline measurements. Hair samples were obtained from the posterior vertex, where hair growth is most consistent and exhibits low variability, in accordance with current guidelines (Favretto et al., 2023; Sauvé et al., 2007). A lock of hair, at least 1 cm long and as thick as a pencil, was cut as close as possible to the

scalp. The participants with short hair provided clippings from the posterior vertex in accordance with guidelines published by the Society of Hair Testing (SoHT; Cooper et al., 2012). Given that scalp hair grows approximately 1 cm per month, the 1 cm segment at postintervention reflects averaged cortisol secretion during the month following the training. This time window is considered appropriate for assessing cumulative HPA axis activity postintervention (Sauvé et al., 2007). Next, samples were stored in a dark environment at room temperature and shipped to the lab after all posttreatment collections. HCC was analyzed by Stress Bioanalytics, LLC, following the carefully validated method described by Bendinskas et al. (2024). In short, samples were washed with isopropanol twice, dried, weighed, milled, and extracted twice with methanol overnight; samples were evaporated, and an acetone chase ensured the dryness of samples; cortisol was measured in duplicate using enzyme-linked immunosorbent assay (ELISA; Arbor Assays, Ann Arbor, MI). The controls, the linearity test, and the parallelism test were as expected. The limit of detection (LOD) was 1.0 pg/mg. The combined extraction and detection variability in the laboratory was 8.3%. The interplate and intraplate variability were up to 10.8% each, as per ELISA manufacturer, Arbor Assays, and the crossreactivity was as follows: cortisol 100%, dexamethasone 18.8%, prednisolone 7.8%, corticosterone 1.2%, and cortisone 1.2%.

Behavioral Measures. Since AT training is known to produce a relaxation effect on the trainee, the subjective questionnaires (i.e., STAI and PSS) were given prior to AT sessions 1 and 24.

EEG Biofeedback Intervention

Alpha-Theta EEG Biofeedback Protocol. All participants completed 24 twice-weekly AT sessions over a period of 12 to 13.5 weeks. The FDA 510k registered equipment BrainMaster Atlantis (BrainMaster Technologies, Inc., Bedford, Ohio) was used for the training. Twice-weekly neurofeedback sessions were conducted within a 2-hr window in a quiet, isolated room. The active electrode was placed at Pz based on the 10–20 system referenced to A2 (right ear lobe) and grounded at A1 (left ear lobe).

Each session was 30 min long and conducted with eyes closed. During the training participants reclined comfortably in a zero-gravity chair and were instructed to relax and remain still, letting go of any control and tension during the session. The alpha-theta protocol was set up for alpha (8.5–11.5 Hz)

uptrain and theta (5–8.5 Hz) uptrain. When each band's criteria were met for 500 ms, the trainee received a reward tone. Automatic thresholding was applied allowing alpha and theta to be rewarded via auditory signals for 50% above threshold. Autothresholding used epoch length of 60 s to compute the autothreshold values. Threshold updating was set to “auto-update repeat” (after prebaseline and after each run); this protocol was set to update 10 times every 180 s. Automated thresholding was utilized with all participants as a means to control for providing the same conditions.

Two distinct tones were provided for auditory feedback for alpha (high-pitched bell) and theta (low-pitched gong sound) reinforcement. In addition, a musical instrument digital interface (MIDI) sustained sound (titled “102 Echo Drops”) played when both alpha and theta remained above threshold. MIDI modulation was based upon amplitude, producing either a softer or a louder sound depending on alpha and theta amplitudes. Before each session, the principal investigator explained the tones associated with alpha and theta and a specific sound that indicated an artifact or disrupted signal.

Sample Script. “You can get into a comfortable position. Check in with your body to release any tension in your neck and shoulders. Take a few slow breaths. Gently close your eyes. Let go of control and let the sounds guide you. This is your time to relax.”

Neurophysiological Measures

Percentage of Crossovers. As previously mentioned, a successful AT training session is indicated by the trainee's ability to relax and allow their vigilance to decrease. This can result in an alpha amplitude drop, permitting theta to surpass and produce a visible trend on the posttraining graph (aka crossover).

The occurrence of crossover activity was examined and assessed throughout all 264 sessions. Each posttraining graph was retrospectively and visually inspected by the principal investigator to determine whether the subject achieved a crossover. No blinding was applied during this process. Crossover events were identified through a combined approach involving real-time visual inspection of session spectral graphs, participant behavioral observations, and subjective self-reports. This method is consistent with prior work by Johnson et al. (2013), who linked crossover patterns with clients' internal experiences. Blinding raters without real-time contextual information may risk misinterpreting

artifacts such as eye blinks, muscle activity, or movement as alpha-theta transitions (Gruzelier, 2009). Thus, integrating physiological data with experiential reports provides a more valid indicator of meaningful crossover events, despite the limitation of nonblinded assessment.

Next, the percentage of crossovers for each participant was calculated based on the following formula: percentage of crossovers = (number of crossover sessions / total sessions) x 100. Table 1 presents a summary of the length of training for each participant, the number of sessions with crossovers, and the corresponding percentage of crossovers.

Posttraining Measurements

After concluding 24 sessions of AT training, the participants completed posttreatment measures, including STAI, PSS, and the 5-point Likert scale. To capture the time during which the brain had a chance to consolidate the learning, the participants waited 1 month before providing the last hair sample. The pre and post samples were properly labeled and stored at room temperature, in the dark (i.e., in a drawer), until they were sent to the laboratory. The pre and post samples for all participants were shipped all at once, allowing the principal investigator to be blinded about individual hair cortisol values during the active part of the protocol.

During this appointment, the participants also completed the posttraining 5-point Likert functionality scale. This subjective measurement evaluated the effects of AT training across the following categories: emotional status, sleep quality, attention span, autonomic nervous system function, and resilience, on a scale from most deteriorated to most improved (i.e., *strongly deteriorated*, *deteriorated*, *not deteriorated nor improved*, *improved*, and *strongly improved*). These categories were selected to capture areas likely influenced by the training (Appendix A).

Statistical Analysis

Each subject's raw data was entered and analyzed with the statistical program IBM-SPSS (SPSS v.26). The subjective and the biological data were not normally distributed; therefore, the Wilcoxon signed-rank test was used as a nonparametric alternative. For exploratory analyses, participants who demonstrated a reduction in HCC from pre- to postintervention were classified as responders, while those who did not were classified as nonresponders.

Results

Subjective Measures – Results of Research Question 1

The Wilcoxon signed-rank test showed statistically significant decreases in both STAI ($z = 2.02$, $p = .0025$) and PSS ($z = 2.805$, $p = .0025$) scores, supporting the hypothesis that 24 sessions of AT training will lead to a decrease in STAI and PSS scores. STAI scores decreased by 15.9 points (see Figure 3), which is a 34.7% reduction from $M = 45.82$ ($SD = 11.97$) to $M = 29.91$ ($SD = 9.05$). There was a large reduction of 12.27 points in the mean PSS from $M = 23.82$ ($SD = 5.896$) to $M = 11.55$ ($SD = 5.087$; see Figure 4). This

represents a 51.5% decrease in perceived stress following the intervention.

On the 5-point Likert functionality scale, *emotional status* ($M = 4.36$, $SD = .674$) tied with *resilience* ($M = 4.36$, $SD = .505$) for the highest mean score, while resilience had the lowest SD variability (see Table 2). The third-highest mean came with *self-awareness* ($M = 4.27$, $SD = .647$). *Autonomic functioning* showed a middle-range mean ($M = 3.91$, $SD = .831$), while *sleep* had the second-lowest mean ($M = 3.73$, $SD = .874$) and the highest variability. The category with the lowest mean was *attention* ($M = 3.82$, $SD = .874$).

Figure 3. Bar Graph of Pre to Post State and Trait Anxiety Score Changes.

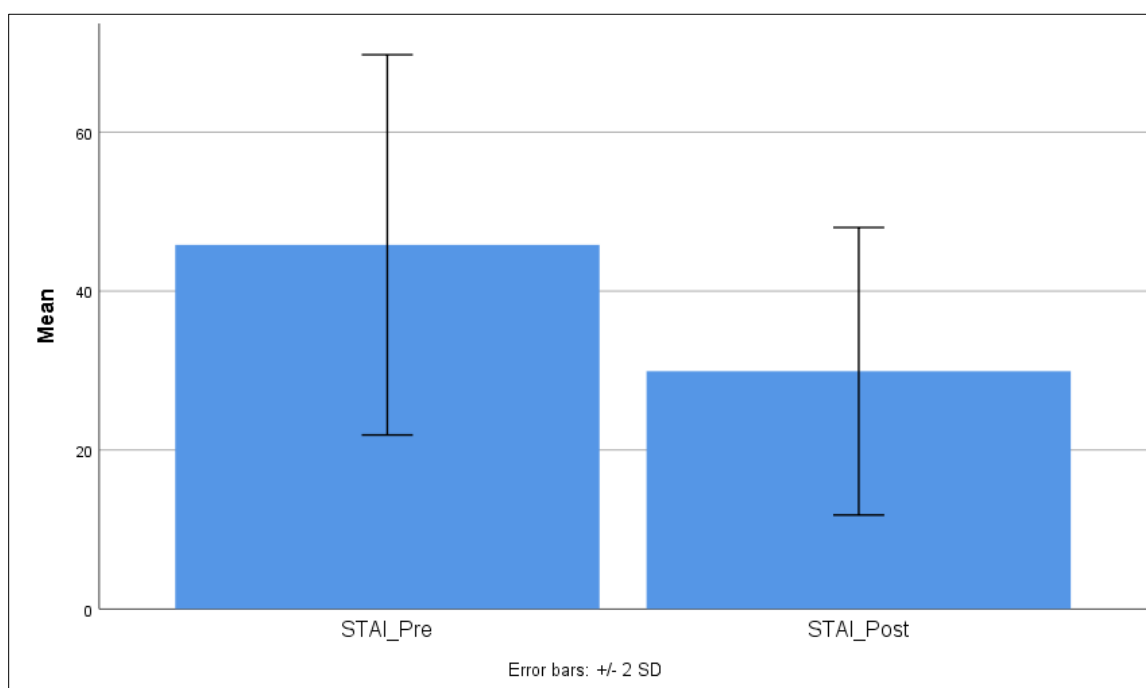
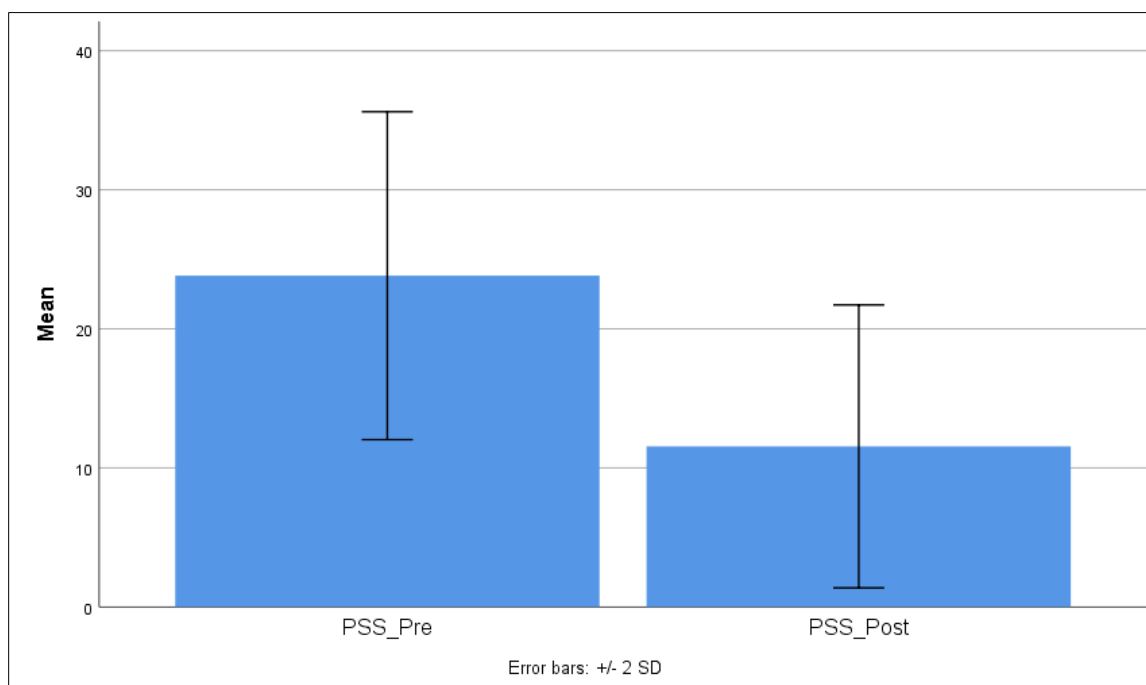


Figure 4. Bar Graph of Pre to Post Perceived Stress Scale Score Changes.**Table 2**

Descriptive Statistics for the Six Areas of Functionality From the Novel Likert Scale: Emotional Status, Sleep, Self-Awareness, Attention, Autonomic Functioning, and Resilience

Functionality	N	Min.	Max.	Mean	SD
Emotional status	11	3	5	4.36	.674
Sleep	11	3	5	3.82	.874
Self-awareness	11	3	5	4.27	.647
Attention	11	3	5	3.73	.647
Autonomic functioning	11	3	5	3.91	.831
Resilience	11	4	5	4.36	.505

Biological Measures – Results of Research Question 2

Pre- and post-HCC values ranged from 4.57 to 15.82 pg/mg and 4.63 to 40.51 pg/mg, respectively (see Table 3). During the analysis, an outlier of 40.51 pg/mg was identified. Because cortisol concentration values are sensitive to glucocorticoid prescriptions and recreational drugs, efforts were made to contact the individual in hopes that they could provide further clarification on what might have

influenced the posttraining HCC value. Unfortunately, the outlier participant did not respond, and no follow-up information was gathered. The Wilcoxon analysis was conducted for $N = 10$ (without the outlier value), but no significance was found, $z = -1.784$, $p = .037$ (one-tailed significance), $p > .01$. The median HCC decreased from 7.61 pg/mg at preintervention to 6.11 pg/mg at postintervention, with a reduction of 1.50 pg/mg (see Figure 5).

Figure 5. Median Hair Cortisol Concentration (HCC, pg/mg) at Pre- and Postintervention for N = 10 Error Bars Represent Interquartile Ranges.

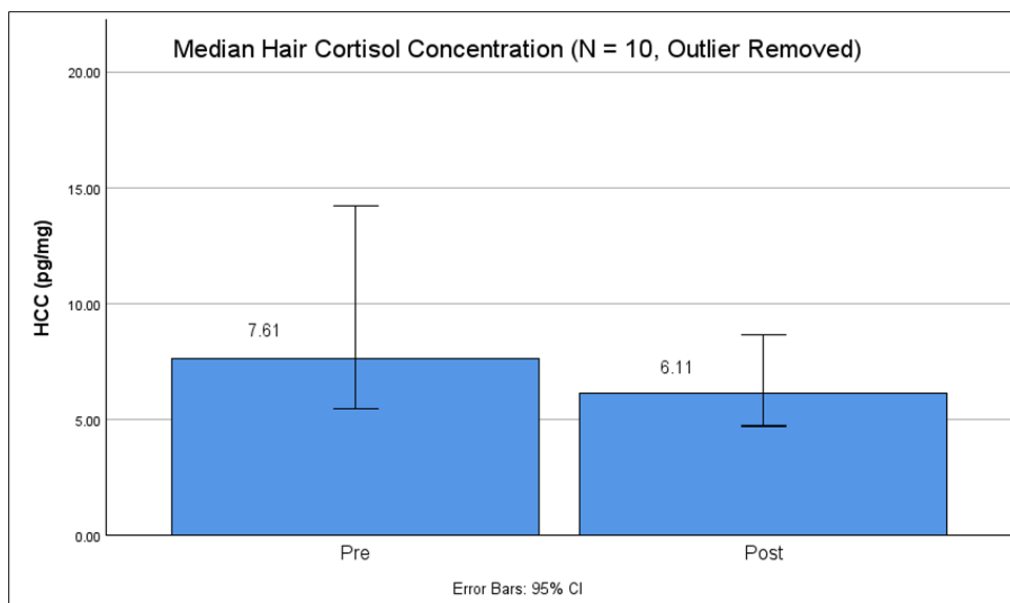


Table 3

Hair Cortisol Concentration Pre and Post Values (= Outlier)*

Participant	Lab ID	Weight (mg)	% RSD	Average	HCC (pg/mg)	Change in HCC
AT001						
Pre	X01	24.02	1.6	789.2	8.21	
Post	X02	18.88	3.0	641.1	8.49	+0.28
AT002						
Pre	X03	19.67	0.5	823.2	10.46	
Post	X04	21.26	1.5	3445.2	40.51*	+30.15
AT003						
Pre	X05	22.39	2.1	409.1	4.57	
Post	X06	19.91	4.1	478.5	6.01	+1.44
AT005						
Pre	X07	20.25	1.5	801.5	9.90	
Post	X08	23.68	1.0	445.0	4.70	-5.20
AT006						
Pre	X09	20.98	3.1	517.6	6.17	
Post	X10	22.68	5.4	556.2	6.13	-0.04
AT007						
Pre	X11	19.50	2.6	425.3	5.45	
Post	X12	21.54	0.3	462.5	5.37	-0.08
AT008						
Pre	X13	23.34	2.2	653.5	7.00	
Post	X14	20.34	2.9	495.1	6.08	-0.92

Table 3
Hair Cortisol Concentration Pre and Post Values (= Outlier)*

Participant	Lab ID	Weight (mg)	% RSD	Average	HCC (pg/mg)	Change in HCC
AT009						
Pre	X15	22.88	2.2	543.4	5.94	-1.31
Post	X16	19.46	1.6	360.7	4.63	
AT010						
Pre	X17	22.05	4.9	826.9	9.38	-0.75
Post	X18	22.87	1.3	789.2	8.63	
AT011						
Pre	X19	23.09	4.2	1461.5	15.82	-7.99
Post	X20	22.46	1.4	703.0	7.83	
AT012						
Pre	X21	21.70	5.1	1233.7	14.21	-3.39
Post	X22	20.35	3.0	880.8	10.82	

A secondary analysis was conducted on the eight participants who demonstrated a reduction in HCC from pre- to posttreatment (responders). This was performed to determine whether the observed physiological stress reduction was statistically significant within this subgroup and to explore whether responders also demonstrated greater AT learning, as indicated by crossover percentages.

The Wilcoxon signed-rank test for this subgroup revealed a significant reduction in HCC ($z = -2.521$, $p = .006$, one-tailed), indicating that participants who lowered their cortisol levels showed a consistent AT training effect. Among responders, the median HCC declined from 8.19 pg/mg to 6.11 pg/mg, representing a reduction of 2.08 pg/mg (see Figure 6). Exploratory comparison of AT learning indicated that responders had a markedly higher mean percentage of sessions with crossovers (48.94%) compared to nonresponders (36.11%). Six of the eight responders exhibited crossover percentages substantially higher than any of the nonresponders (See Table 1), suggesting that greater AT learning may be associated with the observed physiological stress reduction.

EEG Data

All participants were able to experience deep states and achieved crossovers at least one time, and six out of 11 trainees (55%) were able to produce crossovers 62.5% of the time or higher (see Table

1). There were no statistically significant correlations between crossover frequency and subjective or objective measures.

Correlations

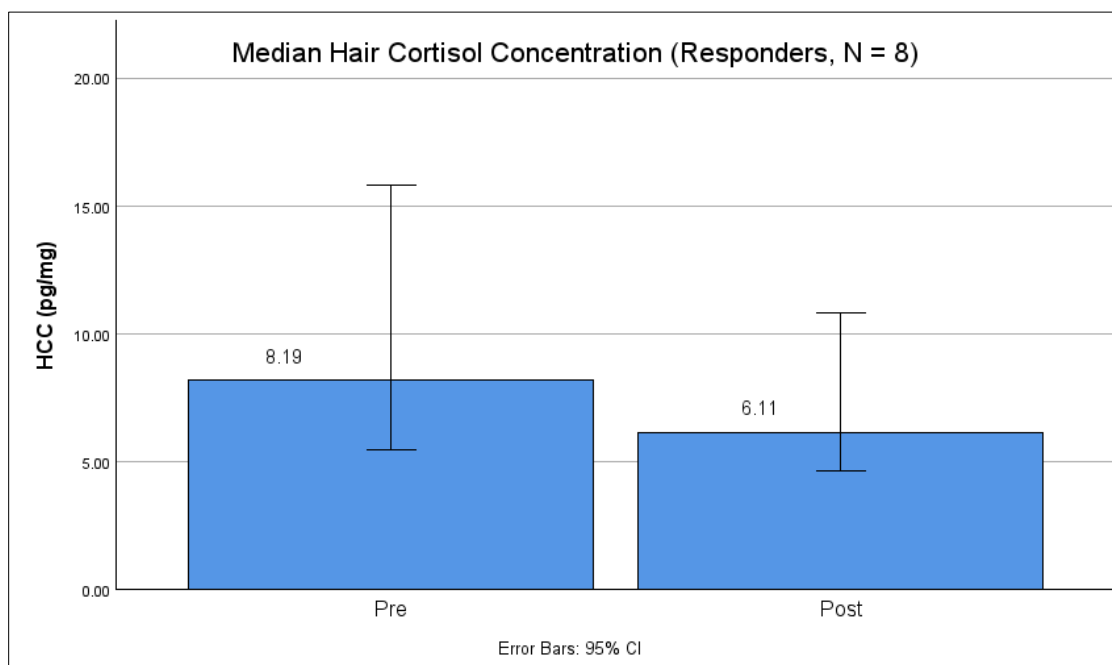
Correlations between all variables were studied to further examine the relationships between pre- and postintervention STAI, PSS, and HCC values. As seen in the correlation matrix (see Figure 7), STAI demonstrated a moderate positive correlation with PSS at both pre ($r = .596$) and postintervention ($r = .607$). Additionally, a slight negative trend was noted between STAI post and HCC post ($r = -.402$). A significant correlation was observed between PSS pre- and HCC pre ($r = -.714$, $p = .007$) and between PSS post and HCC post ($r = -.743$, $p = .004$), suggesting that higher perceived stress was associated with lower HCC levels. Lastly, HCC post had moderate insignificant positive correlation with percentage crossovers ($r = .345$, $p = .150$).

Effect Size

The magnitude of the difference between the pre- and post-STAI scores revealed Cohen's d of 1.174, which suggested a large effect (see Figure 8).

The change between the pre-PSS and post-PSS scores revealed a large effect (Cohen's d 1.655; Figure 9). The magnitude of change for HCC was .62, which indicated moderate effect size (Figure 10).

Figure 6. Median Hair Cortisol Concentration (HCC, pg/mg) at Pre- and Postintervention for N = 8 the Responders' Subgroup.



Note. Error bars represent interquartile ranges.

Figure 7. Bivariate Correlation Matrix: Relationships Between Pre and Post State-Trait Anxiety Inventory, Perceived Stress Scale, Hair Cortisol Concentration, and Percentage Crossovers.

		Correlations						
		STAI_Pre	STAI_Post	Percentage Crossovers	PSS_Pre	PSS_Post	HCC Pre-pg/mg	HCC Post-pg/mg
STAI_Pre	Pearson Correlation	1	.265	.348	.589*	-.285	-.339	-.375
	Sig. (1-tailed)		.230	.162	.037	.212	.169	.143
	N	10	10	10	10	10	10	10
STAI_Post	Pearson Correlation	.265	1	.489	.336	.529	-.293	-.260
	Sig. (1-tailed)	.230		.076	.172	.058	.205	.234
	N	10	10	10	10	10	10	10
Percentage Crossovers	Pearson Correlation	.348	.489	1	-.094	.041	.015	-.053
	Sig. (1-tailed)	.162	.076		.398	.455	.484	.443
	N	10	10	10	10	10	10	10
PSS_Pre	Pearson Correlation	.589*	.336	-.094	1	.247	-.746**	-.449
	Sig. (1-tailed)	.037	.172	.398		.246	.007	.096
	N	10	10	10	10	10	10	10
PSS_Post	Pearson Correlation	-.285	.529	.041	.247	1	-.642*	-.501
	Sig. (1-tailed)	.212	.058	.455	.246		.023	.070
	N	10	10	10	10	10	10	10
HCC Pre- pg/mg	Pearson Correlation	-.339	-.293	.015	-.746**	-.642*	1	.651*
	Sig. (1-tailed)	.169	.205	.484	.007	.023		.021
	N	10	10	10	10	10	10	10
HCC Post- pg/mg	Pearson Correlation	-.375	-.260	-.053	-.449	-.501	.651*	1
	Sig. (1-tailed)	.143	.234	.443	.096	.070	.021	
	N	10	10	10	10	10	10	10

*. Correlation is significant at the 0.05 level (1-tailed).
 **. Correlation is significant at the 0.01 level (1-tailed).

Figure 8. Paired Samples Effects Size for the Magnitude of Change for Pre- and Post-STAI.

Paired Samples Effect Sizes						
			Standardizer ^a	Point Estimate	95% Confidence Interval	
Pair 1	STAI_Pre - STAI_Post	Cohen's d	13.546	1.174	.379	1.937
		Hedges' correction	14.680	1.084	.350	1.787

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

Figure 9. Paired Samples Effects Size for the Magnitude of Change for Pre- and Post-PSS.

Paired Samples Effect Sizes						
			Standardizer ^a	Point Estimate	95% Confidence Interval	
Pair 1	PSS_Pre - PSS_Post	Cohen's d	7.417	1.655	.711	2.566
		Hedges' correction	8.038	1.527	.656	2.367

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

Figure 10. Paired Samples Effects Size for the Magnitude of Change for Pre- and Post-HCC.

Paired Samples Effect Sizes						
			Standardizer ^a	Point Estimate	95% Confidence Interval	
Pair 1	HCC Pre- pg/mg - HCC Post- pg/mg	Cohen's d	2.89446	.620	-.075	1.288
		Hedges' correction	3.16724	.567	-.068	1.177

a. The denominator used in estimating the effect sizes.
Cohen's d uses the sample standard deviation of the mean difference.
Hedges' correction uses the sample standard deviation of the mean difference, plus a correction factor.

Discussion

The present study contributed to the existing body of research on the effects of alpha-theta (AT) training by examining whether 24 AT sessions would reduce HCC and symptoms of perceived stress and anxiety.

Overall, GAD is considered a moderately stable diagnosis (Angst et al., 2009). In the present study, the within-subjects design and the use of the validated psychometric measures STAI and PSS captured individual pre- to postintervention changes, independent of diagnostic shifts that may appear

over time. The hypothesis that 24 AT neurofeedback sessions will lead to at least a 15% reduction in symptoms of anxiety and stress was statistically supported (STAI: $z = 2.02$, $p = .0025$; PSS: $z = 2.805$, $p = .0025$). Anxiety scores decreased by 34.7%, well above the predicted 15%, and perceived stress lowered by 51.5%. Both measures showed large effect sizes, indicating that the effectiveness of AT training was not due to chance. These findings suggest that AT neurofeedback has practical and meaningful implications. Furthermore, the outcomes in the subjective measures from this study align with the existing literature, which suggests that AT

training can alleviate symptoms of anxiety beyond the placebo effect (Dadashi et al., 2015; Gruzelier, 2014; N. C. Moore, 2005). The findings from the PSS scores in the current study are also consistent with previous research by Bennett et al. (2018), supporting AT training application in stress management.

Relative to studies on alternative modalities for GAD treatment, the present study demonstrated comparable results. In particular, when compared to the controlled study by Linden et al. (2005), the findings suggest that AT training may be as effective as cognitive behavioral therapy. In Linden et al.'s study, the initial treatment group showed 14.6% reduction in STAI scores and the crossover group showed an 11.6% reduction. By contrast, this pilot study showed a reduction of 34.7% in pre- to post-STAI scores, indicating AT training may be an efficacious treatment modality for GAD, especially in cases where talk therapy is not the preferred option (e.g., when there is a language or communication barrier, history of ineffective talk therapy, or when facing cognitive overload challenges). Findings involving the functionality scale should be interpreted cautiously, given the measure's exploratory nature and lack of established psychometric properties. The results were generally positive, with no deterioration in any area. The greatest improvements were in resilience and emotional status. Sleep ($M = 3.82$, $SD = .874$) showed little to no impact, and attention was least influenced ($M = 3.73$, $SD = .647$). The latter is not surprising because attention-focused neurofeedback training is often tailored to the individuals' unique frontal and prefrontal EEG function, and these areas were not targeted in this study.

The outcomes of the Likert functionality scale align with Imperatori et al. (2017), who reported that AT training enhances mentalization and increases EEG connectivity within the default mode network. Improvements observed in the categories of self-awareness and emotional status are consistent with earlier findings which link deep relaxation with well-being and emotional awareness (Boynton, 2001; Gruzelier, 2014). These EEG findings suggest that increased connectivity may support improvements in self-reflection and emotional awareness.

Even though eight out of 11 participants had reduced HCC postintervention, the statistical analysis did not support the second hypothesis that 24 AT sessions will lead to at least a 20% reduction in HCC ($p > .01$). One possible explanation for this finding is the small sample size, which is often

associated with low statistical power and a risk of committing a Type II error, and the study fails to detect the true effect. In addition, small samples also tend to show greater variability in results.

An important consideration is the interpretation of responder analyses. In the present study, responders were previously defined as participants who demonstrated a reduction in HCC from pre- to postintervention. While such subgroup analyses can introduce selection bias, they are valuable for exploring whether observed physiological changes are driven by a subset of participants rather than the full sample. By reporting both whole-sample and responder-only results, we sought to balance transparency with specificity. The significant HCC reduction observed in responders, coupled with their higher crossover percentages, points to the possibility that effective outcomes are contingent on successful protocol learning. It should be noted that the observed percentage decrease in median HCC should be interpreted in the context of approximately 11% variability inherent to the detection method. In addition, the small sample size limits generalization.

EEG Discussion

A key aspect of this investigation was the alpha-theta crossover, a state marking deep relaxation and transition into stage 1 sleep. Existing research often links crossovers to visualization and mental images. For example, Peniston viewed the crossover as an objective of the training and a "window of opportunity" for suggestibility and vivid imagery (Peniston et al., 1993). Johnson et al. (2013) distinguished two different types of crossovers: therapeutic crossovers associated with the emergence of visualizations and crossovers without "personally relevant material." In contrast, J. P. Moore et al. (2000) questioned whether alpha-theta crossovers are necessary for successful training and effective outcomes, suggesting they may not be essential.

This study analyzed 264 graphs from 11 individuals, identifying 120 sessions with crossovers. All participants experienced at least one crossover moment throughout the training and subjectively reported deep relaxation during this time. Only two instances included memory recall of a nonthreatening nature. Although there were significant reductions in the STAI and PSS scores, there were no significant correlations between the crossover percentage and the subjective measures. These findings imply that the participants experienced positive changes regardless of the number of crossovers they experienced. This

information may support J. P. Moore et al.'s (2000) argument that alpha-theta crossovers may not be a prerequisite for a therapeutic effect to occur. Further assessment of the AT protocol is required to clarify the role of alpha-theta crossovers in the efficacy of the training.

Past investigations, including this one, did not assess participants' baseline EEG characteristics, which may affect crossover accessibility. Anxiety-related EEG patterns include the low-voltage fast EEG, beta variants (i.e., frontal beta, central spindling beta, global beta, temporal beta, hypercoherence in beta), fast alpha profile, and high amplitude EEG phenotypes, as discussed by Johnstone et al. (2005). This is important because reaching crossover states for some of these profiles may be more challenging than others. For example, a fast alpha variant with posterior alpha of 12–14 Hz might appear in the low beta (12–15 Hz) frequency range and not alpha. This could cause the crossover to go undetected. In another example, a low voltage EEG may not be generating enough alpha to produce a crossover on the graph. Accounting for EEG variability may be meaningful to better outcomes in future research.

Finally, this study did not define the crossover by amplitude or duration. Future studies might adopt Johnson et al.'s (2013) model, which included specific criteria: theta exceeding alpha by at least 1 μ V, sustained for 3–10 min; beta (15–20 Hz) increase; and a subjective report for imagery. In the present study, participants reported recall of images in only two of the sessions; however, there was strong evidence that AT training alleviated symptoms associated with stress and anxiety.

Limitations

The outcomes of this study should be considered in light of some limitations. Cortisol values in the body are sensitive to factors such as (a) glucocorticoid use, (b) recreational drugs, and (c) alcohol use (Bendinskas et al., 2024). The use of any of these items can easily be overlooked or underreported throughout the study; however, they are confounding to the HCC values and can lead to exaggerated cortisol numbers. Future investigations should provide participants with a comprehensive list of substances which may compromise the cortisol readings and collect self-reports on potential usage. The functionality scale used in this study was a novel exploratory instrument designed specifically for this pilot context. As such, its psychometric properties, including reliability and validity, have not yet been established. Future research should

include rigorous evaluation of the scale's factor structure, internal consistency, and test–retest reliability to support its broader use. The visual inspection method for identifying alpha-theta crossovers was conducted without blinding because crossover identification relied on real-time review by the principal investigator integrating both spectral data and participant feedback. This approach may introduce potential bias. Future studies should use more standardized, objective thresholds and assess inter-rater reliability to strengthen the validity of crossover measurements. Although all participants completed pre- and postintervention full-cap 19-channel quantitative EEG (qEEG) assessments, these data are not included in the present study and are reserved for a future forthcoming study. The present paper reported EEG outcomes limited to alpha-theta crossover trends observed during neurofeedback sessions. The absence of qEEG results in this paper does not affect the conclusions regarding subjective and physiological stress markers.

The small sample size may lead to overgeneralization and decreases the statistical power of the study. The use of a convenience sample might have limited representation to a broader population. Next, the lack of randomization and control group may affect the reliability of the findings. Other confounding variables such as the placebo effect and research biases may also interfere with the results of the study. Lastly, the lack of similar studies limits the interpretations and comparisons of the results and the contextualization within the current body of literature.

Future Recommendations

Based on these results, the following directions for a future study are recommended: group the participants based on their specific EEG signature, include a pre- and post-qEEG to assess global normalization and synchronization, include a control group, and include individuals with the same anxiety EEG profile.

Conclusion

This investigation contributed to the existing body of literature on AT neurofeedback by integrating psychological and physiological assessments in a quest to holistically explore mind and body connections. The subjective measures, the HCC, and the EEG data provided empirical evidence about the usefulness of AT training. Furthermore, this study researched the novel hypothesis that AT training can decrease subjective measures of stress

and anxiety, as well as objective measures, such as cortisol. Overall, the results from the present study are encouraging. Collectively, the results indicated that 24 sessions of AT training can result in significant improvements in stress-related symptoms and anxiety. Although the change in cortisol measures did not reach statistical significance, eight out of 11 individuals demonstrated reduced postintervention cortisol levels. In responders, significant HCC reduction coincided with higher crossover percentages, suggesting the possibility that deeper AT learning leads to lower cortisol levels. On a functionality level, the AT training led to improved emotional status, resilience, and self-awareness, as indicated by the results from the Likert functionality scale. These findings support previous research showing that AT training can lead to increased well-being and relaxation. Finally, AT training can be a potentially viable nonpharmaceutical tool to manage symptoms of stress and anxiety and reduce arousal as effectively as cognitive behavioral therapy. Further research is recommended to replicate and expand these preliminary results.

Author Acknowledgments

K.Z. expresses sincere thanks to Dr. Mary Tracy for suggesting the inclusion of the Likert functionality scale in this study. K.Z. also gratefully acknowledges Nick Smythe for his collegial support throughout the development of this work.

Author Disclosures

K.B. is the CEO of Stress Bioanalytics, LLC. Other authors have no relevant financial or nonfinancial interests to disclose. The authors received no financial support for the submitted work. No artificial intelligence (AI) tools were used in the preparation, analysis, or writing of this manuscript. This work reflects solely the authors' original contributions.

References

- Abdian, H., Rezaei, M., Eskandari, Z., Ramezani, S., Pirzeh, R., & Dadashi, M. (2021). The effect of quantitative electroencephalography-based neurofeedback therapy on anxiety, depression, and emotion regulation in people with generalized anxiety disorder. *Basic and Clinical Neuroscience*, 12(2), 281–290. <https://doi.org/10.32598/BCN.12.2.2378.1>
- Abell, J. G., Stalder, T., Ferrie, J. E., Shipley, M. J., Kirschbaum, C., Kivimäki, M., & Kumari, M. (2016). Assessing cortisol from hair samples in a large observational cohort: The Whitehall II study. *Psychoneuroendocrinology*, 73, 148–156. <https://doi.org/10.1016/j.psyneuen.2016.07.214>
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
- Angst, J., Gamma, A., Baldwin, D. S., Ajdacic-Gross, V., & Rössler, W. (2009). The generalized anxiety spectrum: Prevalence, onset, course and outcome. *European Archives of Psychiatry and Clinical Neuroscience*, 259(1), 37–45. <https://doi.org/10.1007/s00406-008-0832-9>
- Anxiety and Depression Association of America. (2022, October 28). *Anxiety disorders—Facts & statistics*. <https://adaa.org/understanding-anxiety/facts-statistics>
- Bendinskas, K., Liu, J., Jandev, V., Careem, F. R., Hidoyatov, M., Garofalo, R., Norful, A. A., Padilla, J. J., & Schnall, R. (2024). Hair and nail cortisol levels are associated and affected by glucocorticoid use. *Psychoneuroendocrinology*, 168, Article 107139. <https://doi.org/10.1016/j.psyneuen.2024.107139>
- Bennett, C. N., Gupta, R. K., Prabhakar, P., Christopher, R., Sampath, S., Thennarasu, K., & Rajeswaran, J. (2018). Clinical and biochemical outcomes following EEG neurofeedback training in traumatic brain injury in the context of spontaneous recovery. *Clinical EEG and Neuroscience*, 49(6), 433–440. <https://doi.org/10.1177/1550059417744899>
- Berger, H. (1929). Über das elektrenkephalogramm des menschen [About the human electroencephalogram]. *Archiv für Psychiatrie und Nervenkrankheiten*, 87(1), 527–570. <https://doi.org/10.1007/BF01797193>
- Boynton, T. (2001). Applied research using alpha/theta training for enhancing creativity and well-being. *Journal of Neurotherapy*, 5(1–2), 5–18. https://doi.org/10.1300/J184v05n01_02
- Bozovic, D., Racic, M., & Ivkovic, N. (2013). Salivary cortisol levels as a biological marker of stress reaction. *Medical Archives*, 67(5), 374–377. <https://doi.org/10.5455/medarh.2013.67.374-377>
- Chang, H., Perkins, M. H., Novaes, L. S., Qian, F., Zhang, T., Neckel, P. H., Scherer, S., Ley, R. E., Han, W., & de Araujo, I. E. (2024). Stress-sensitive neural circuits change the gut microbiome via duodenal glands. *Cell*, 187(19), 5393–5412.e30. <https://doi.org/10.1016/j.cell.2024.07.019>
- Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology*, 5(7), 374–381. <https://doi.org/10.1038/nrendo.2009.106>
- Clemente-Perez, A., Makinson, S. R., Higashikubo, B., Brovarney, S., Cho, F. S., Urry, A., Holden, S. S., Wimer, M., Dávid, C., Fenno, L. E., Acsády, L., Deisseroth, K., & Paz, J. T. (2017). Distinct thalamic reticular cell types differentially modulate normal and pathological cortical rhythms. *Cell Reports*, 19(10), 2130–2142. <https://doi.org/10.1016/j.celrep.2017.05.044>
- Cooper, G. A. A., Kronstrand, R., & Kintz, P. (2012). Society of Hair Testing guidelines for drug testing in hair. *Forensic Science International*, 218(1–3), 20–24. <https://doi.org/10.1016/j.forsciint.2011.10.024>
- Costa, M. A., Gadea, M., Hidalgo, V., Pérez, V., & Sanjuán, J. (2016). An effective neurofeedback training, with cortisol correlates, in a clinical case of anxiety. *Universitas Psychologica*, 15(5). <https://doi.org/10.11144/Javeriana.upsy15-5.entc>
- Crick, F. (1984). Function of the thalamic reticular complex: The searchlight hypothesis. *Proceedings of the National Academy of Sciences of the United States of America*, 81(14), 4586–4590. <https://doi.org/10.1073/pnas.81.14.4586>
- Dadashi, M., Birashk, B., Tareman, F., Asgarnejad, A. A., & Momtazi, S. (2015). Effects of increase in amplitude of occipital alpha & theta brain waves on global functioning level of patients with GAD. *Basic and Clinical Neuroscience*, 6(1), 14–20.
- Edwards, S. (2013). Alpha theta meditation: phenomenological, neurophysiologic, mindfulness, mood, health and sport implications: Social psychology. *African Journal for Physical Health Education, Recreation, and Dance*, 19(2), 419–434.
- Favretto, D., Cooper, G. A. A., Andraus, M., Sporkert, F., Agius, R., Appenzeller, B., Baumgartner, M., Binz, T., Cirimele, V., Kronstrand, R., del Mar Ramirez, M., Strano-Rossi, S., Uhl, M., Vincenti, M., & Yegles, M. (2023). The Society of Hair Testing consensus on general recommendations for hair

- testing and drugs of abuse testing in hair. *Drug Testing and Analysis*, 15(9), 1042–1046. <https://doi.org/10.1002/dta.3526>
- Gadea, M., Aliño, M., Hidalgo, V., Espert, R., & Salvador, A. (2020). Effects of a single session of SMR neurofeedback training on anxiety and cortisol levels. *Neurophysiologie Clinique*, 50(3), 167–173. <https://doi.org/10.1016/j.neucli.2020.03.001>
- Glantz, S. A. (2012). *Primer of biostatistics* (7th ed.) [CD-ROM software]. McGraw Hill Medical.
- Gruzelier, J. (2009). A theory of alpha/theta neurofeedback, creative performance enhancement, long distance functional connectivity and psychological integration. *Cognitive Processing*, 10(Suppl. 1), 101–109. <https://doi.org/10.1007/s10339-008-0248-5>
- Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. I: A review of cognitive and affective outcome in healthy participants. *Neuroscience & Biobehavioral Reviews*, 44, 124–141. <https://doi.org/10.1016/j.neubiorev.2013.09.015>
- Halassa, M. M., & Acsády, L. (2016). Thalamic inhibition: Diverse sources, diverse scales. *Trends in Neurosciences*, 39(10), 680–693. <https://doi.org/10.1016/j.tins.2016.08.001>
- Imperator, C., Della Marca, G., Amoroso, N., Maestoso, G., Valenti, E. M., Massullo, C., Carbone, G. A., Contardi, A., & Farina, B. (2017). Alpha/theta neurofeedback increases mentalization and default mode network connectivity in a non-clinical sample. *Brain Topography*, 30(6), 822–831. <https://doi.org/10.1007/s10548-017-0593-8>
- Johnson, M., & Bodenhamer-Davis, E. (2017). The therapeutic crossover in A/T training neurofeedback: Temporal and spectral components of access to levels of consciousness. In A. Martins-Mourao & C. Kerson (Eds.), *Alpha theta neurofeedback training in the 21st century: A handbook for clinicians and researchers* (2nd ed., pp. 141–172). A FNNR Publication.
- Johnson, M. L., Bodenhamer-Davis, E., Bailey, L. J., & Gates, M. S. (2013). Spectral dynamics and therapeutic implications of the theta/alpha crossover in alpha-theta neurofeedback. *Journal of Neurotherapy*, 17(1), 3–34. <https://doi.org/10.1080/10874208.2013.758968>
- Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG and Neuroscience*, 36(2), 99–107. <https://doi.org/10.1177/155005940503600209>
- Kamiya, J. (1971). Biofeedback training in voluntary control of EEG alpha rhythms. *California Medicine*, 115(3), 44.
- Kerson, C. (2017). The neurophysiology of trauma. In A. Martins-Mourao & C. Kerson (Eds.), *Alpha-theta in the 21st century* (pp. 131–132). Academic Press.
- Kropotov, J. (2009). Chapter 4—Frontal midline theta rhythm. In *Quantitative EEG, event-related potentials and neurotherapy* (pp. 77–95). Elsevier Inc. <https://doi.org/10.1016/B978-0-12-374512-5.00004-9>
- Lenze, E. J., Mantella, R. C., Shi, P., Goate, A. M., Nowotny, P., Butters, M. A., Andreescu, C., Thompson, P. A., & Rollman, B. L. (2011). Elevated cortisol in older adults with generalized anxiety disorder is reduced by treatment: A placebo-controlled evaluation of escitalopram. *The American Journal of Geriatric Psychiatry*, 19(5), 482–490. <https://doi.org/10.1097/JGP.0b013e3181ec806c>
- Linden, M., Zuberhagen, D., Baer, T., Franke, U., & Schlattmann, P. (2005). Efficacy of cognitive behaviour therapy in generalized anxiety disorders: Results of a controlled clinical trial (Berlin CBT-GAD Study). *Psychotherapy and Psychosomatics*, 74(1), 36–42. <https://doi.org/10.1159/000082025>
- Marcuse, L. V., Fields, M. C., & Yoo, J. (2016). The normal adult EEG. In L. V. Marcus, M. C. Fields, & J. Yoo (Eds.), *Rowan's primer of EEG* (2nd ed., pp. 39–66). Elsevier. <https://doi.org/10.1016/B978-0-323-35387-8.00002-0>
- Marzbani, H., Marateb, H. R., & Mansourian, M. (2016). Neurofeedback: A comprehensive review on system design, methodology and clinical applications. *Basic and Clinical Neuroscience*, 7(2), 143–158. <https://doi.org/10.15412/J.BCN.03070208>
- McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840(1), 33–44. <https://doi.org/10.1111/j.1749-6632.1998.tb09546.x>
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87(3), 873–904. <https://doi.org/10.1152/physrev.00041.2006>
- Moore, J. P., Trudeau, D. L., Thuras, P. D., Rubin, Y., Stockley, H., & Dimond, T. (2000). Comparison of alpha-theta, alpha and EMG neurofeedback in the production of alpha-theta crossover and the occurrence of visualizations. *Journal of Neurotherapy*, 4(1), 29–42. https://doi.org/10.1300/J184v04n01_04
- Moore, N. C. (2005). The neurotherapy of anxiety disorders. *Journal of Adult Development*, 12(2–3), 147–154. <https://doi.org/10.1007/s10804-005-7031-y>
- Niedermeyer, E. (1999). Sleep and the EEG. In E. Niedermeyer & F. Lopes Da Silva (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (4th ed., pp. 174–189). Williams and Wilkins.
- Noushad, S., Ahmed, S., Ansari, B., Mustafa, U. H., Saleem, Y., & Hazrat, H. (2021). Physiological biomarkers of chronic stress: A systematic review. *International Journal of Health Sciences*, 15(5), 46–59.
- Núñez, A., & Buño, W. (2021). The theta rhythm of the hippocampus: From neuronal and circuit mechanisms to behavior. *Frontiers in Cellular Neuroscience*, 15, Article 649262. <https://doi.org/10.3389/fncel.2021.649262>
- Nunez, P. L. (2017). Electroencephalography (EEG). In *Reference Module in Neuroscience and Biobehavioral Psychology*. Elsevier Inc. <https://doi.org/10.1016/B978-0-12-809324-5.03049-2>
- Peniston, E. G., & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta-endorphin levels in alcoholics. *Alcoholism, Clinical and Experimental Research*, 13(2), 271–279. <https://doi.org/10.1111/j.1530-0277.1989.tb00325.x>
- Peniston, E. G., & Kulkosky, P. J. (1991). Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related post-traumatic stress disorder. *Medical Psychotherapy*, 4, 47–60.
- Peniston, E. G., Marrinan, D. A., Deming, W. A., & Kulkosky, P. J. (1993). EEG alpha-theta brainwave synchronization in Vietnam theater veterans with combat-related post-traumatic stress disorder and alcohol abuse. *Advances in Medical Psychotherapy*, 6(7), 37–50.
- Raymond, J., Varney, C., Parkinson, L. A., & Gruzelier, J. H. (2005). The effects of alpha/theta neurofeedback on personality and mood. *Cognitive Brain Research*, 23(2–3), 287–292. <https://doi.org/10.1016/j.cogbrainres.2004.10.023>
- Sauvé, B., Koren, G., Walsh, G., Tokmakejian, S., & Van Uum, S. H. M. (2007). Measurement of cortisol in human hair as a biomarker of systemic exposure. *Clinical and Investigative Medicine*, 30(5), E183–E191. <https://doi.org/10.25011/cim.v30i5.2894>
- Saxby, J., & Peniston, E. G. (1995). EEG biofeedback and alcohol use disorders: A review and pilot study. *Journal of Neurotherapy*, 1(2), 35–47. https://doi.org/10.1300/J184v01n02_04
- Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Stermann, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy*, 15(4), 292–304. <https://doi.org/10.1080/10874208.2011.623089>
- Shonkoff, J. P., Garner, A. S., the members of Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, & Section on Developmental and Behavioral Pediatrics, Siegel,

- B. S., Dobbins, M. I., Earls, M. F., Garner, A. S., McGuinn, L., Pascoe, J., & Wood, D. L. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, 129(1), e232–e246. <https://doi.org/10.1542/peds.2011-2663>
- Smith, S. J. M. (2005). EEG in the diagnosis, classification, and management of patients with epilepsy. *Journal of Neurology, Neurosurgery and Psychiatry*, 76(Suppl. 2), ii2–ii7. <https://doi.org/10.1136/jnnp.2005.069245>
- Stalder, T., Steudte-Schmiedgen, S., Alexander, N., Klucken, T., Vater, A., Wichmann, S., Kirschbaum, C., & Miller, R. (2017). Stress-related and basic determinants of hair cortisol in humans: A meta-analysis. *Psychoneuroendocrinology*, 77, 261–274. <https://doi.org/10.1016/j.psyneuen.2016.12.017>
- Thomas, C. L., & Cassady, J. C. (2021). Validation of the state version of the state-trait anxiety inventory in a university sample. *SAGE Open*, 11(3). <https://doi.org/10.1177/21582440211031900>
- Vaitl, D., Birbaumer, N., Gruzelier, J., Jamieson, G. A., Kotchoubey, B., Kübler, A., Lehmann, D., Miltner, W. H. R., Ott, U., Pütz, P., Sammer, G., Strauch, I., Strehl, U., Wackermann, J., & Weiss, T. (2005). Psychobiology of altered states of consciousness. *Psychological Bulletin*, 131(1), 98–127. <https://doi.org/10.1037/0033-2909.131.1.98>
- Zani, A., Tumminelli, C., & Proverbio, A. M. (2020). Electroencephalogram (EEG) alpha power as a marker of visuospatial attention orienting and suppression in normoxia and hypoxia: An exploratory study. *Brain Sciences*, 10(3), Article 140. <https://doi.org/10.3390/brainsci10030140>

Received: September 4, 2025

Accepted: February 11, 2026

Published: September 28, 2026

Appendix A

Figure A1. Posttraining Functionality Scale Questionnaire.

Post- training Questionnaire

Name: _____ Date: _____

Which areas of functioning do you feel were influenced by Alpha Theta training?

	Strongly Deteriorated	Deteriorated	Neither Deteriorated nor Improved	Improved	Strongly Improved
Emotional Status (e.g. rumination, anxiety, negative thoughts, stress-related anxiety, etc.)	☐	☐	☐	☐	☐
Sleep	☐	☐	☐	☐	☐
Self-Awareness	☐	☐	☐	☐	☐
Attention Span	☐	☐	☐	☐	☐
Autonomic Nervous System Function (body responses to stress, e.g. nausea, sweating, blood pressure, "fight-flight" responses etc.)	☐	☐	☐	☐	☐
Resilience (i.e., the ability to work creatively with stress rather than allowing stress to affect performance)	☐	☐	☐	☐	☐
Other:	☐	☐	☐	☐	☐

Effectiveness of Infra-Low Frequency Neurofeedback on Postconcussion Symptoms in Combat Veterans: Results of a Delayed Intervention Analysis

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Abstract

Introduction. The purpose of this study was to ascertain the impact infra-low frequency neurofeedback (ILF NFB) had on a group of combat veterans with postconcussive symptoms who served as a control group in a randomized controlled trial (RCT) and later completed a delayed intervention procedure. **Methods.** After completing the control procedures (eight weekly 15-min health-related discussions and four assessment sessions), veterans were invited to join a delayed intervention (twenty 30-min sessions of ILF NFB and three assessment sessions) group. Of 38 control group members, 32 went on to complete the delayed intervention procedures (16% attrition). **Results.** Comparing baseline to end of treatment measures, findings were clinically and statistically significant for headache ($p < .0001$), sleep ($p < .0001$) and attention ($p < .0044$). Additional measures of interest were also significantly improved following ILF NFB, including quality of life ($p < .0001$), depressive symptoms ($p < .0001$), and symptoms of posttraumatic stress disorder ($p = .0001$). Significant results remained at the 2-month follow-up on all measures, despite a history of chronicity for many years and prior treatment failure. **Conclusion.** ILF NFB holds promise to be a safe and effective intervention for those who suffer with postconcussive symptoms of chronic headache, sleep, and attention disorders.

Keywords: infra-low frequency neurofeedback; mTBI; postconcussive symptoms; veterans; sleep; headache; endogenous neuromodulation

Citation: Carlson, J., Tyrrell, C. J., Fiame, B., Nunokawa, C., Ross, W., & Schaper, K. (2026). Effectiveness of infra-low frequency neurofeedback on postconcussions symptoms in combat veterans: Results of a delayed intervention analysis. *NeuroRegulation*, 13(3), 239–255. <https://doi.org/10.15540/nr.13.3.239>

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Introduction

Traumatic brain injury (TBI) is a common malady in the United States with over 4 million citizens experiencing a TBI annually, of which 90% are considered mild TBI (mTBI; Berman et al., 2023). About 40% of those with mTBI suffer from chronic symptoms (Voormolen et al., 2018) and often experience significant economic and functional impact affecting the ability to fully participate in their roles (e.g., work, family; Gaudette et al., 2022;

Sharma, 2022). A special subset of those with mTBI are service members who were part of recent post-9/11 military operations. It has been estimated that over a 20-year period from 2001 to 2021, about 90% of 441,639 veterans diagnosed with TBI had an mTBI (Whiteneck et al., 2024). However, rather than blunt head injuries (e.g., from motor vehicle accidents or athletics) that usually cause mTBIs in civilians, the most common cause of TBI by combat veterans was exposure to explosive blasts from devices such as improvised explosive devices,

rocket-propelled grenades, and landmines. The whole-body exposure to the pressurization shock waves of the blasts causes very complex injuries in the brain, involving both focal injuries (direct impact of the brain's surface on the bony protuberances of the skull and body pressure pushing up into the skull cavity) and diffuse injuries (stretching and twisting of axons and blood vessels by shearing forces; Chapman & Diaz-Arrastia, 2014). In addition, there are frequently secondary, longer-duration brain injuries related to the activation of molecular and biochemical responses that were once thought to be self-limiting (i.e., lasting hours or days postinjury). These complex brain injuries often result in abnormal brain signaling, including impairments to normal brain connectivity and electrical brain waves and patterns, as well as disruptions of intra- and interhemispheric communication and inflammatory processes that last much longer, leading to chronic and debilitating symptoms (Chapman & Diaz-Arrastia, 2014; Hayes et al., 2016). These types of heterogeneous and chronic symptom profiles have been identified in veterans diagnosed with mTBI due to blast exposure (Rowland et al., 2024). A recent review of the literature further validates the consistent and compelling evidence of the long-term effects of blast-related TBI in terms of poorer psychological health, greater healthcare utilization and disability levels, impacts on brain structure and function, and greater headache impact on daily life (Miller et al., 2024).

Data from the Military Health System Data Repository indicated that the frequent comorbidities with mTBI were headache, cognitive deficits, sleep-related problems, anxiety disorders, and posttraumatic stress disorder (PTSD; Agimi et al., 2024). When these symptoms persist, they can negatively impact veterans' self-rated health occupational status (ability to return to work, school) social functioning and quality of life (QOL; MacGregor et al., 2013; Morissette et al., 2011; Sakamoto et al., 2021; Schiehser et al., 2015). Because of the complexity of the brain injury and prevalence of comorbid complications, combat-related mTBIs can prove difficult to treat. Therefore, developing and implementing effective strategies to reduce the persistent symptoms associated with mTBI is of critical importance.

According to the 2021 VA/DoD clinical practice guidelines for the management and rehabilitation of postacute mTBI (Eapen et al., 2022), the current treatment for postconcussive symptoms (PCS) remains symptom focused. Veterans with migraine headaches associated with mTBI are often treated

with abortive agents (e.g., Triptans) and preventive medications (e.g., anticonvulsants and tricyclics; Theeler & Erickson, 2012). Cognitive dysfunction and insomnia are treated with cognitive rehabilitation programs, cognitive behavior therapy, occupational therapy, and medications (Ayalon et al., 2007). Symptom management results in addressing one concern at a time with treatments that may be temporary, not fully tackling the concern, or may even result in additional side effects and complications. While cognitive rehabilitation and psychological support are widely used to treat mTBI PCS, neither has been shown to be effective in addressing the core brain deficits associated with mTBI (Duff, 2004). Since symptoms often return and persist, it is clear that improved treatment options for these persistent PCS need to be developed for veterans with mTBI (Morissette et al., 2011).

A more targeted approach to the treatment of TBI would be to enhance latent neuroplasticity which may establish a more normalized or stable brain (DeFina et al., 2009). The ability to recruit neuroplastic mechanisms in the service of rehabilitation has been demonstrated by neurofeedback (NFB), as reflected in microstructural changes in white and gray matter (Enriquez-Geppert et al., 2013; Ghaziri et al., 2013; Ros et al., 2013). NFB has been shown to contribute to functional rehabilitation by restoring network connectivities in areas of the brain that may have been impaired, rehabilitative changes that appear to be sustained over the longer term (Ibric et al., 2009). Functional magnetic resonance imaging (fMRI) studies further validate that NFB may be useful in promoting recovery from neurological disorders rooted in abnormal patterns of brain connectivity (Haller et al., 2013; Koush et al., 2013). Hence, NFB, a noninvasive and nonpharmacological modality, may be used to improve functionality by remediating abnormal network relationships, including both steady-state and dynamic connectivity (Haller et al., 2013).

ILF NFB, a novel type of NFB, was initially conceived in 2006 and has developed over time to its present level of clinical maturity (Othmer & Othmer, 2020; Othmer, 2023). ILF NFB primarily utilizes the slow cortical potential domain, which refers to brainwave frequencies below 0.1 Hz (Othmer & Othmer, 2020). It requires a very specialized type of instrumentation that can isolate and provide information back to the brain about its functioning in the infra-low frequency regime. The Cygnet NFB instrument (<https://www.beemedic.com/cygnet.html>), an example of equipment that has the

ILF capability to access brainwave frequencies as low as .000001 (Othmer, 2023) was used in this study. Since ILF NFB differs from conventional NFB with respect to focus, mechanisms of action, and the role of both therapist and client, this innovative type of NFB is still new to most neuroscience and NFB experts and practitioners, most of whom still work with methods grounded in traditional operant conditioning.

Unlike traditional NFB, ILF NFB recruits latent neuroplasticity by way of feeding back real-time information on the time course of the frequency-delimited slow cortical potential, as derived from the differential signal from two cortical sites (bipolar montage). The use of bipolar montage reveals the dynamics of selected critical linkages within the intrinsic connectivity networks, and on that basis the brain adjusts its activity level, moderates neuronal excitability, and alters connectivity relationships. Since the brain is reacting to, and acting upon, slowly varying intrinsic network activity, this process is most appropriately referred to as endogenous neuromodulation. This is the essential distinction that sets ILF NFB apart from conventional NFB, which involves explicit targeting in what is typically an operant conditioning design. ILF NFB fundamental mechanism of action is the renormalization of the steady-state and dynamic functional connectivity of the resting state networks. Since the low frequencies establish the context for the activation and interaction of neuronal assemblies, they present a potent and most efficient target for functional renormalization (Buzsáki & Watson, 2012; Monto et al., 2008).

NFB has been used since the 1960s for symptoms related to mTBI. Duff (2004), in his extensive review of the literature, suggests that since the 1960s, studies using NFB (EEG biofeedback or neurotherapy) have shown that patients can be taught to recover normal functioning in brains with excessively slow wave activity, which is often observed in PCS. In a 2013 review of the literature, May et al. used a 10-level classification rubric to classify the research literature of NFB and mTBI, with 10 being the highest level (e.g., randomized controlled trials [RCT]) and 1 being the lowest (e.g., case study/anecdotal evidence). They found two studies at level 5 (randomized waitlist or intention to treat), six studies at level 3 (historical control), ten studies at level 2 (no control group) and five studies at level 1 (case study/anecdotal evidence). Of the 23 studies reviewed, all found NFB to positively impact symptoms associated with mTBI (attention, memory, quality of life, sleep, motor control, coordination,

depression, headaches). May et al. (2013) called for more RCT to further support the efficacy of NFB treatments. Recent studies have continued to demonstrate support for the use of NFB with symptoms related to mTBI. Nelson and Esty (2012) found in their small study ($n = 7$) that neurotherapy significantly reduced depression, somatic and memory/attention symptoms, when employed with Operation Enduring Freedom and Operation Iraqi Freedom veterans diagnosed with TBI and PTSD. A study conducted in 2013 demonstrated that NFB was able to enhance quality of life and perceived control in 29 service members with mTBI and PTSD (Strang & Chae, 2013). In a control group/waitlist study conducted in 2014, sixty mTBI participants (aged 18–49 years old) received NFB, and it was found that 20 sessions of NFB significantly improved quality of life (Reddy et al., 2014). Munivenkatappa et al. in 2014 provided further validation of the ability of NFB to enhance structural and functional connectivity and cognitive scores of mTBI patients. Arns and Kenemans in 2014 reported in their review of the literature regarding attentional and sleep disorders that NFB is associated with improved sleep quality and sleep onset. After a thorough meta-analysis of NFB research, Larsen and Sherlin rated NFB "probably" efficacious for the treatment of mTBI symptoms (2013).

Since ILF NFB is a more recently developed technology, to date there have been few published studies that have demonstrated the efficacy of ILF NFB relating to veterans with mTBI who experience chronic PCS including headache, insomnia, and attention dysfunction. Previous research suggested that ILF NFB positively affected change in postconcussive symptom severity (Carlson & Ross, 2021; Legarda et al., 2022). Other studies found that ILF NFB decreased incidence of both migraines and tension-type headaches (Arina et al., 2022; Dobrushina et al., 2017). However, these studies' findings have limited generalizability due to methodological limitations such as selection bias (Legarda et al., 2022) and small sample size (Arina et al., 2022; Carlson & Ross, 2021; Dobrushina et al., 2017). Though not in a population with mTBI, ILF NFB has also demonstrated clinical efficacy in addressing symptoms of depression (Grin-Yatsenko et al., 2018). There are also several publications on the efficacy of ILF NFB in clinical settings (Benson & LaDou, 2015; Kirk & Dahl, 2022; Legarda et al., 2011; McMahon, 2020; Othmer & Othmer, 2009; Shapero & Prager, 2020).

This paper presents additional analysis of data from veterans who completed all control group

procedures as well as all delayed intervention procedures during a 5-year RCT. The RCT was conducted with veterans who experienced an mTBI while participating in post-9/11 military operations to evaluate the impact of ILF NFB on chronic PCS of headache, insomnia, and attention dysfunction. Initial results of this trial that included all randomized participants using an intention-to-treat analysis demonstrated significant differences between the intervention and control groups with strong effect sizes at end of treatment on all outcome measures (Carlson et al., 2025). Because of the known probable effectiveness of ILF NFB for veterans with mTBI by the research team from previous pilot study and clinical practice experience, the team felt an ethical compulsion to design the study to include a delayed intervention arm of the study. For this analysis, it was hypothesized that ILF NFB would improve veterans' headache, sleep, and attentional symptoms above and beyond those results received while serving in the control group.

Methods

The initial RCT study was a parallel RCT, which used block randomization set of four (2 treatment, 2 control) to ensure that each group had a similar number of participants. The biostatistician created the blocks and placed group assignments in individual security envelopes. Once the control group completed all control group procedures, they were invited to enter the delayed intervention group.

Research Ethics

This study was performed in line with the principles of the Declaration of Helsinki. All procedures of this study were performed in compliance with relevant laws and institutional guidelines and was approved by the Veterans Administration Pacific Islands Health Care System Institutional Review Board Approval Number: 2019-06-JC/Promise 0003.

Inclusion and Exclusion Criteria

Participants included veterans who had received a diagnosis of mTBI associated with post-9/11 military operations, were between the ages of 18 and 65, and experienced the PCS symptoms of interest (chronic headaches, insomnia, and attention difficulties). To be eligible, participants also needed to be able to read and write English, comprehend what was read, and follow directions. The diagnosis of mTBI was verified for each veteran via electronic health records. Veterans were excluded if they were under the age of 18 or over the age of 65; were pregnant at the time of the study; had a severe TBI diagnosis or impaired decision-making capacity;

were unable to comply with study visit schedule; or endorsed active suicidal intent on the Columbia Suicide Severity Rating Scale (Posner et al., 2011).

To recruit veterans, letters about the study with its contact information were sent to eligible veterans that were identified by a clinical informatics data pull after a waiver of Health Insurance Portability and Accountability Act was obtained. Two weeks after the letter was sent, a member of the research team telephoned the veteran to answer any questions. Also, veterans could be referred to the study by providers, or veterans could self-refer based flyers and other marketing materials advertising the study. Veterans who completed the control group procedures were invited to enter the delayed intervention arm.

Setting and Location

The study was conducted from 2020 to 2025 at a mid-sized outpatient Veterans Health Administration facility located in Oahu, Hawaii. Four private research spaces in compliance with human research standards (e.g., privacy, safety), were utilized for the consenting, treatments, and assessment sessions.

Consenting and Randomization Procedure

The privacy rights of potential participants were observed, and all interested veterans who met the eligibility criteria underwent an informed consent procedure which included information about the opportunity for veterans in the control group to enter a delayed intervention group once all control group procedures were completed. All those consented were administered the Columbia Suicide Severity Rating Scale (Posner et al., 2011). Anyone who screened positive for suicidality was redirected to same-day mental health services available in the clinic. Those veterans who did not screen positive for active suicide intent were randomized into the intervention or control group by the research coordinator who used the block of four security envelopes until the four were distributed, then the next block of four would be used. Once randomized, the participant was considered enrolled in the study and scheduled for the baseline assessment.

Primary Outcome Measures

For the RCT, the outcome assessment measures were administered to the intervention and control groups at baseline, midpoint, end of treatment, and at a 2-month follow-up by the research assistant or research coordinator. Once the control group procedures were completed, the 2-month follow-up outcome assessment served as the baseline outcome assessment for the delayed intervention

group. Those in the delayed intervention group completed additional assessments after 10 sessions of ILF NFB, after completion of all 20 sessions, and 2 months after completion of ILF NFB treatment. All outcome assessments for the delayed intervention group were administered by the research assistant or research coordinator.

Headache Measures.

- Headache Impact Test (HIT-6; Kosinski et al., 2003) is a 6-item measure describing people's feelings and impact of their headache over the past month. The score range is 36–78, with lower scores indicating less headache impact. The clinically significant change score index is 2.3 points (Coeytaux et al., 2006).
- TBI-Quality of Life Headache Pain Short form (TBI-QOL Headache; Tulskey et al., 2019) is a 10-item instrument that measures headache pain over the past week. The raw score range is 10–50, with lower scores indicating less pain. The clinically significant change score index is 7 points (Portz et al., 2017).

Sleep Measures.

- Insomnia Severity Index (ISI; Bastien et al., 2001) is a 7-item measure, assessing the severity, perception, and consequences of the person's insomnia over the past 2 weeks. Insomnia scores are as follows: 8–14, subthreshold insomnia; 15–21, moderate severe insomnia; and 22–28, severe insomnia. The clinically significant change score index is 3 points (Yang et al., 2009).
- Sleep Disturbance short form (NEURO QOL Sleep; Cella et al., 2012) is an 8-item form that measures a person's reported sleep disturbance over the past 7 days. The raw score range is 8–40, with lower scores indicating less disturbance. The clinically significant change score index is 7 points (Kozlowski et al., 2016)

Attention Measure. QIKtest Continuous Performance Test (QIKtest; Versace, 2014) is a computerized visual continuous performance 21-min test. The Accuracy Index was used as the attention measure which reflects sustained attention and impulse control. Sustained attention is determined by omission errors and outliers in regard to response times, and impulse control is determined by commission errors and anticipatory errors. The Accuracy Index score range is 165–460 with a

higher score suggesting better attention. The clinically significant change score index is 7 points.

Secondary Outcome Measures

QOL Measures.

- QOL After Brain Injury (QOLIBRI; Truelle et al., 2010) is a 37-item questionnaire that assesses life since the brain injury. The raw score range is 0–100, with higher scores suggesting greater satisfaction. The clinically significant change score index is a 30% shift.
- Satisfaction with social roles and activities - short form (NEUROQOL TBI Satisfaction; Cella et al., 2012) is an 8-item instrument that measures a person's satisfaction with social roles and activities over the past seven days. The raw score range is 8–40, with higher scores indicating greater satisfaction. The clinically significant change score index is 3.7 points (Portz et al., 2017).
- Ability to participate in social roles and activities short form (NEUROQOL TBI Ability; Cella et al., 2012) is an 8-item instrument that measures a person's perception of their capability to participate in social roles and activities in the past seven days. The raw score range is 8–40, with higher scores suggesting an increased ability. The clinically significant change score index is 3.9 (Portz et al., 2017).

Variables of Interest.

- Depression, Anxiety and Stress Scale–21 (DASS-21; Lovibond & Lovibond, 1995) is a 21-item instrument that assesses depressive, anxious, and stress symptoms over the past week. Total score range is 0–63, with the following interpretive ranges: 17–20, mild distress; 21–25, moderate distress; 26–29, severe distress; and 30+, extremely severe distress. The clinically significant change score index is 12.7 points (Ronk et al., 2013).
- Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001) is a 9-item instrument that assesses depression. The score range is 0–27, with a score of 10 suggesting moderate depression and lower scores suggesting fewer depressive symptoms. The clinically significant change score index is 5 points (Kroenke, 2012).
- PTSD Checklist (PCL-5; Weathers et al., 2013) is a 20-item instrument that assesses the symptoms of PTSD in the past month. The score range is 0–80, with a score of 31–

33 suggesting probable PTSD, and lower scores indicating less PTSD symptoms. The clinically significant change score index is 10 points (Weathers et al., 2013).

- General Symptom Inventory (GSI; Carlson, 2021) is a 117-item instrument that assesses general health symptoms over the past month. Each item is a 5-point scale with 0 = *did not apply to me at all* to 4 = *applied to me very much, or most of the time*. Total score range is 0–464, with lower scores suggesting less severity or less frequency of the item. The clinically significant change score index is a 30% change.

Sample Size. For the RCT, it was determined that 72 participants (36 in each group, intervention and control) were needed to attain the goals of the study. Up to 100 veterans were projected to be recruited to achieve the 72 enrolled participants to complete the study. Literature on NFB studies of patients with TBI have reported dropout rates ranging from 10% to 30% (Nelson & Esty, 2012; Zoefel et al., 2011). Assuming a dropout rate of 28% and a moderate autocorrelation of 0.6 among repeated measures, this sample size ensured the detection of an average difference of at least 0.49 standard deviations with a power of 80% in the NFB intervention group compared to the control group using a two-tailed significance level of 0.05. For the delayed intervention analyses, the sample size consisted of those control group completers ($n = 38$), who were willing and able to enter the delayed intervention group.

Procedures

The procedures for control group whose members then became the delayed intervention group are as follows:

Control Group. Control group participants received eight phone calls (one call/week) over the course of 8–10 weeks. Calls were completed by one of the four clinical investigators. Each call was approximately 15 min, and one health topic was discussed. The health topics discussed included sleep hygiene, basic nutritional concepts, beverage choices, positive thinking, thought reframing, fitness, daily calming activity, and enhancement of focus strategies. A script for each of these topics was used to guide each call.

Delayed Intervention Group. The delayed intervention group participants completed 20 half-hour ILF NFB sessions, typically receiving three sessions per week over an 8- to 10-week period. ILF

NFB treatments were provided by one of four licensed healthcare employees, who had received substantial ILF NFB training. The principal investigator was one of the four ILF NFB providers with extensive experience in NFB as well as Board Certification in Neurofeedback by the Biofeedback Certification International Alliance. The other three ILF NFB providers received formal and ongoing optimal response frequency (ORF) precision training and supervision from the principal investigator. Reliability between the four ILF NFB providers was established using a procedure developed by the principal investigator to determine the level of fidelity of providers' decisions in response to clinical data from a case study presented. This procedure yielded an inter-rater skill-level reliability of .95 among the providers. A coaching script was used by the ILF NFB providers during the ILF NFB sessions to keep the interactions approach consistent with all the participants.

All procedures were explained in advance to participants and voluntary participation affirmed. At the first session, participants also filled out a clinical symptom checklist of five symptoms (Carlson & Ross, 2021). The first three symptoms were specific to the study aims (headache, sleep, and attention), while the additional two items were specific to each participant's self-identified symptoms of interest and selected at their initial assessment session. Participants rated these five items on an 11-point Likert scale (0–10, where 0 was *No Issue* and 10 was *Worst Issue*); the initial ratings were reflective of their experience over the past month, while in each subsequent session, participants indicated their experience of those symptoms that day. ILF NFB providers were never privy to participants' assessment outcome measures data during the course of the study, so these recurring clinical symptom ratings, as well as other feedback provided by participants on their experience within and between sessions, were the clinical data that ILF NFB providers used to make decisions about the treatment protocol.

The precision model of ILF NFB, based on the Othmer ORF protocol (Othmer & Othmer, 2020), was further refined by the principal investigator and utilized for the participants in this study. The ORF protocol establishes through an iterative process the specific frequency point within the ILF spectrum that is optimal for resolution of the symptoms being experienced in real time during the session (e.g., tension or pain in shoulders, racing thoughts). The iterative process includes the prompting of participants every 2–3 min to describe their

subjective experience of the specific symptoms identified and, based on these descriptions, the ILF NFB provider carefully interprets its meaning and methodically adjusts the frequency (higher or lower) until the unique ORF is identified for each participant. Typically, a participant's ORF was identified within the first 4–6 sessions; symptom tracking over the course of treatment further verified that the participant's ORF had been identified. More information about the ILF NFB procedures followed is outlined in the report of the pilot study (Carlson & Ross, 2021).

Participants completed 30 min of ILF NFB at each session, during which they were instructed to look at the computer monitor; the moving images on the screen provided prompt feedback to participants about their brain functioning as ascertained by electrodes placed on the scalp (Huster et al., 2013). Working with bipolar montage, the brain gets feedback (neuroceptively) on the dynamic differential activation at the two sites. This information is sufficient for the brain to regulate its state of activation by way of a whole-brain response that over time allows for resolution of subjective symptoms and enhanced functioning. Electrode placement followed the 10–20 international system of electrode placement (Hughes, 1994). Per study protocol, sites used were right parietal (T4–P4), interhemispheric (T3–T4), right frontal (T4–Fp2), and left frontal (T3–Fp1), though the combination of sites was individualized to the participant based on their response to the intervention. The unique ORFs for all participants in this study were identified and ranged from .000004 to .973096 Hz, with a median frequency of .002825 Hz.

Statistical Analysis

The initial between-subjects' results of the RCT are reported in Carlson et al. (2025). The present analysis represented an additional objective of the RCT, which was to evaluate the effectiveness of ILF NFB for participants who were initially randomized into the control condition and later received ILF NFB as a delayed intervention.

This analysis assessed outcome scores at the endpoint and 2-month follow-up assessments, comparing participants following ILF NFB treatment to their own assessment scores from their control group participation. The control group's 2-month follow-up assessment served as the baseline for the delayed intervention analyses. A paired-sample *t*-test was used to evaluate the mean difference in outcome scores between treatments, at 5% type-I error rate. Additionally, mean scores were estimated

at multiple time points, including midtreatment, with corresponding 95% confidence intervals (CIs). The analysis followed a per-protocol approach, including only participants who completed the treatment phases and the required assessments. It is important to note that the reported 95% CIs serve as guidelines only, as no adjustments were made for multiple comparisons across time points. All statistical analyses were performed using SAS software, version 9.4M8.

Results

Participants

Of the 44 randomly assigned to the control group, 38 participants completed the control group procedures per protocol, with 32 participants completing the delayed intervention per protocol, an attrition rate of 16%. No adverse event was related to the treatment; most were related to other illnesses (e.g., COVID-19) that forced rescheduling appointments, and no participant withdrew because of the intervention. Figure 1 demonstrates the complete details of participant flow through this study.

The average age of participants was 45 years ($SD = 8.7$). The majority were male (89.5%), and the two most frequently reported ethnic group identified were biracial/multiracial (31.6%) and White (23.7%). Table 1 includes additional details on participant demographics, as well as baseline scores on the variables of interest for the present analyses.

Primary Variables of Interest Analyses

The study's main hypothesis was that participants would demonstrate significant improvement in symptoms related to headaches, sleep disturbance, and difficulty with attention following completion of the delayed intervention when compared to their initial assessments on these variables during participation in treatment-as-usual control conditions. Paired-sample *t*-tests supported this hypothesis. A paired *t*-test demonstrated that veterans reported improved headache symptoms after completing NFB treatment at both the endpoint assessment, $p < .0001$, $d = 1.28$; and after a 2-month delay, $p < .0001$, $d = 1.01$. This pattern was also true for the TBI QOL Headache Pain measure at the conclusion of treatment, $p = .0001$, $d = 1.12$; and at the follow-up assessment, $p < .0001$, $d = 0.94$. Paired *t*-tests also demonstrated significantly improved subjective report of sleep following NFB treatment at the endpoint assessment, $p < .0001$, $d = 1.34$; and at the 2-month follow-up assessment $p < .0001$, $d = 0.98$. This

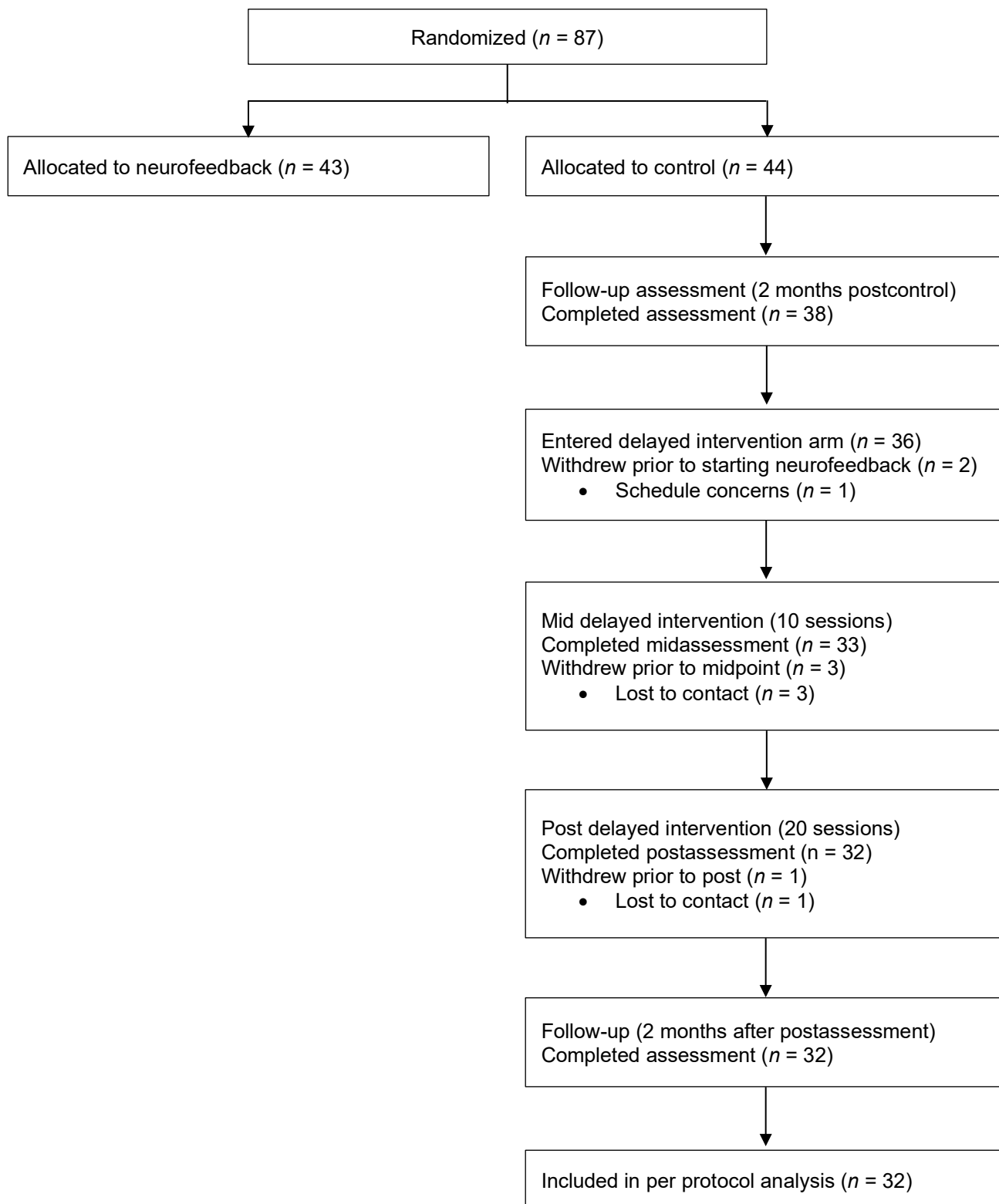
Figure 1. CONSORT Delayed Intervention Participant Flow Diagram.

Table 1
Baseline Participant Characteristics and Test Scores

Control Group Completers (N = 38)	
Participant Demographics	
Age	45.5 (8.7)
Gender (Male)	34 (89.5%)
Self-identified Ethnicity	
African/Black	7 (18.4%)
European/White	9 (23.7%)
Asian	5 (13.2%)
Native American	0 (0%)
Hispanic	2 (5.3%)
Hawaiian/Pacific Islander	1 (2.6%)
Biracial/Multiracial	12 (31.6%)
Refused/Unknown	2 (5.3%)
Delayed Intervention Completers Primary Variables of Interest at Baseline (n = 32)	
HIT-6	62.6 (5.6)
TBI QOL Headache Pain	32.2 (8.5)
ISI	16.9 (5.6)
Neuro QOL Sleep Disturbance	25.1 (6.0)
QIK-Test	259.3 (44.9)
Delayed Intervention Completers Secondary Variables of Interest at Baseline (n = 32)	
QOLIBRI	46.3 (17.2)
Neuro QOL Participate	24.4 (5.7)
Neuro QOL Satisfaction	21.9 (6.1)
PCL-5	36.5 (18.7)
PHQ-9	13.0 (5.7)
DASS-21	22.9 (12.4)
GSI	149.7 (19.0)

Note. Participant features observed at baseline are summarized as counts with percentages of the total indicated in parentheses, with the exception of age. Participant age and test scores observed at baseline are summarized as average with standard deviation indicated in parentheses.

pattern also held true for the Neuro QOL Sleep Disturbance subtest, which demonstrated improvement in scores following NFB treatment compared to the control group procedure at endpoint, $p < .0001$, $d = 1.43$; and at the 2-month assessment, $p < .0001$, $d = 1.10$. Finally, a paired

t -test showed significant improvement in attention at completion of the NFB treatment compared to completion of the control group protocol, $p = .0197$, $d = 0.43$; and at the 2-month follow-up assessment, $p = .0497$, $d = 0.32$. See Table 2 for additional details of these analyses.

Table 2
*Result**

Measure	Delayed Intervention Baseline	Assessment Point	Post Delayed Intervention	<i>p</i> -value	Cohen's <i>d</i> **
Primary Variables of Interest					
HIT6	62.6 (60.2, 65.1)	End	51.8 (48.3, 55.2)	<.0001	1.28
		Follow-up	54.0 (50.5, 57.5)	<.0001	1.01
TBI QOL Headache Pain	32.2 (28.8, 35.5)	End	21.2 (17.8, 24.6)	<.0001	1.12
		Follow-up	22.7 (19.1, 26.3)	<.0001	0.94
ISI	16.9 (15.0, 18.8)	End	8.6 (6.2, 11.0)	<.0001	1.34
		Follow-up	10.7 (8.2, 13.2)	<.0001	0.98
Neuro QOL Sleep Disturbance	25.1 (23.3, 26.9)	End	16.9 (14.8, 19.1)	<.0001	1.43
		Follow-up	18.6 (16.2, 20.9)	<.0001	1.10
QIK-Test	259.3 (241.2, 277.4)	End	281.4 (263.1, 299.6)	.0197	0.43
		Follow-up	276.4 (255.8, 297.0)	.0497	0.32
Secondary Variables of Interest					
QOLIBRI	50.8 (45.4, 56.2)	End	66.9 (59.9, 73.9)	<.0001	0.91
		Follow-up	64.0 (55.6, 72.3)	<.0001	0.67
Neuro QOL Participate	25.2 (23.4, 27.0)	End	29.0 (26.7, 31.3)	<.0001	0.66
		Follow-up	29.0 (26.3, 31.7)	.0030	0.60
Neuro QOL Satisfaction	23.3 (21.3, 25.4)	End	28.2 (25.4, 31.0)	<.0001	0.70
		Follow-up	28.2 (25.4, 31.1)	.0013	0.70
PCL-5	36.5 (30.6, 42.4)	End	19.8 (13.3, 26.2)	<.0001	0.94
		Follow-up	20.2 (13.9, 26.4)	<.0001	0.93
PHQ-9	13.0 (11.1, 15.0)	End	6.1 (4.3, 8.0)	<.0001	1.23
		Follow-up	7.0 (4.7, 9.2)	<.0001	0.99
DASS-21	22.9 (19.0, 26.8)	End	10.3 (6.5, 14.2)	<.0001	1.10
		Follow-up	12.9 (8.5, 17.2)	<.0001	0.84
GSI	149.7 (130.5, 168.9)	End	82.0 (58.9, 105.1)	<.0001	1.11
		Follow-up	91.3 (66.9, 115.7)	<.0001	0.93

*Result Table displays mean outcome scores at three time points during the delayed intervention—baseline (i.e., follow-up of the control phase), end of treatment, and follow-up—with corresponding 95% confidence intervals. The reported *p*-values reflect within-group differences in mean scores at the end of the delayed intervention and at the 2-month follow-up compared with the delayed-intervention baseline. Analyses were conducted using a per-protocol approach. Within-group comparisons were performed using paired-sample *t*-tests, with corresponding *p*-values and Cohen's *d* effect sizes reported.

** Cohen's *d* effect size Legend: 0–0.2 = not relevant; 0.2–0.5 = Small Effect Size; 0.5–0.8 = Moderate Effect Size; 0.8–1.3 = Large Effect Size; 1.3+ = Very Large Effect Size

Secondary Variables of Interest Analyses

In addition to the study’s main variables of interest, participants provided self-report data regarding quality of life, trauma-related symptoms, depressive symptoms, and generalized symptoms. The hypothesis again was that participants would demonstrate improvement on these secondary variables of interest following their participation on NFB treatment compared to their scores upon completion of the initial control group protocol. This hypothesis was supported for all variables, as results showed improvement above and beyond that seen upon completion of the control group procedure on all variables. In regard to quality of life, participants had significantly improved scores on the QOLIBRI, with participants reporting higher scores upon completion of treatment compared to completion of the control condition both at the endpoint assessment, $p < .0001$, $d = 0.91$; and at the 2-month follow-up, $p = .0004$, $d = 0.67$. This was also true on the Neuro QOL Participate subtest upon completing NFB treatment at the end of the study, $p < .0001$, $d = 0.66$; and at the 2-month follow-up, $p = .003$, $d = 0.60$. A similar pattern was found for the Neuro QOL Satisfaction subtest, with participants demonstrating higher scores upon completion of the treatment condition than the control condition, $p < .0001$, $d = 0.70$; and at the 2-month follow-up, $p = .0013$, $d = 0.70$.

In regard to subjective symptom reports, veterans reported a significant reduction in trauma-related symptoms on the PCL-5 upon completion of NFB

versus the control condition at the endpoint assessment, $p < .0001$, $d = 0.94$; and at the 2-month follow-up, $p < .0001$, $d = 0.93$. Participants also demonstrated significantly lower scores on the PHQ-9 in the NFB condition than in the control condition at the endpoint assessment, $p < .0001$, $d = 1.23$; and at the 2-month follow-up, $p < .0001$, $d = 0.99$. Participants also reported less subjective stress on the DASS-21 following the treatment condition compared to the control condition at the end of the study, $p < .0001$, $d = 1.10$; and at the 2-month follow-up, $p < .0001$, $d = 0.84$. The participants indicated fewer general symptoms on the GSI or less severity of symptoms at the end of the study, $p < .0001$, $d = 1.11$; and at the 2-month follow-up, $p < .0001$, $d = 0.93$. See Table 2 for additional details on these analyses.

Clinical impact of an intervention can also be assessed by determining if there were clinically significant change shifts in scores on outcome measures. As seen in Table 3, there were clinically significant change scores for most of the outcome measures.

Additionally, we looked at the average difference between scores at the end of treatment and scores at the 2-month follow-up assessment to examine whether the clinically significant benefits were maintained after the conclusion of treatment (Table 4). Overall, the observed differences were not substantial based on the confidence intervals displayed.

Table 3
Average Change of Delayed Intervention Group From Its Control Group Results With Clinically Significant Change Indices for Each Measure and Percentage of Responders for Each

Assessment Tool	End of Treatment Score	Baseline Score	Average Change in Delayed Intervention Group*	Clinically Significant Change Index	Percentage of Responders (95% CI)**
HIT-6	51.8	62.6	−10.8	2.3 points	87.5 (71.0, 96.5)
TBI QOL Headache Pain	21.2	32.2	−11	7 points	68.8 (50.0, 83.9)
ISI	8.6	16.9	−8.3	3 points	68.8 (50.0, 83.9)
Neuro QOL Sleep Disturbance	16.9	25.1	−8.2	7 points	56.2 (37.7, 73.6)
QIK Test	281.4	259.3	22.1	7 points	61.3 (42.2, 78.2)
QOLIBRI	66.9	50.8	16.1 (32%)	30%	53.1 (34.7, 70.9)

Table 3

Average Change of Delayed Intervention Group From Its Control Group Results With Clinically Significant Change Indices for Each Measure and Percentage of Responders for Each

Assessment Tool	End of Treatment Score	Baseline Score	Average Change in Delayed Intervention Group*	Clinically Significant Change Index	Percentage of Responders (95% CI)**
Neuro QOL Participate	29.0	25.2	3.8	3.9 points	37.5 (21.1, 56.3)
Neuro QOL Satisfaction	28.2	23.3	4.9	3.7 points	56.2 (37.7, 73.6)
PCL-5	19.8	36.5	-16.7	10 points	59.4 (40.6, 76.3)
PHQ-9	6.1	13.0	-6.9	5 points	59.4 (40.6, 76.3)
DASS-21	10.3	22.9	-12.6	12.7 points	46.9 (29.1, 65.3)
GSI	82.0	149.7	-66.7 (45% change)	30%	68.8 (50.0, 83.9)

*Negative scores noted in this column indicate the direction of effect. Only the change score itself is relevant to this table and not the direction of the effect.

**Percentage of subjects who showed a clinically significant improvement in outcome scores at the end of the NFB phase compared with the follow-up of the control phase, with corresponding 95% Clopper–Pearson exact binomial confidence intervals. Analyses were conducted using a per-protocol approach.

Table 4

Average Difference in Outcome Scores Between 2-Month Follow-Up and the End of Treatment Phase With CI Intervals*

Assessment Tool	Average difference (95% CI)
HIT-6	2.3 (-0.9, 5.3)
TBI QOL Headache Pain	1.6 (-1.5, 4.7)
ISI	2.1 (0.5, 3.6)
Neuro QOL Sleep Disturbance	1.6 (-0.2, 3.5)
QIK-Test	-5.4 (-17.4, 6.6)
QOLIBRI	-2.9 (-7.7, 1.9)
Neuro QOL Participate	0 (-2.1, 2.1)
Neuro QOL Satisfaction	0 (-2.3, 2.3)
PCL-5	0.4 (-4.2, 5.0)
PHQ-9	0.8 (-0.6, 2.3)
DASS-21	2.5 (-0.3, 5.4)
GSI	9.2 (-5.8, 24.3)

* Difference = 2-month follow-up – end of treatment. Analyses were conducted using a per-protocol approach.

Discussion

This analysis focused on participants who completed the control and delayed intervention procedures to assess the impact of ILF NFB on symptoms commonly experienced by veterans who have experienced mTBI, headaches, sleep, and attention disorders. In addition to these specific variables of interest, additional assessment measures capture data on participants' QOL, and symptoms related to PTSD and depression. Veterans, who were originally from the control group of a larger RCT, completed a delayed ILF NFB intervention. Participants demonstrated statistically significant improvements in all areas assessed when compared to their own control group data at baseline. The associated effect sizes varied from small (attention), moderate (QOL related to life satisfaction and participation in roles), large (headache, sleep, QOL after brain injury, PTSD at follow-up, depression, anxiety, and stress and general symptoms experience), and very large (sleep) at posttreatment. Most of the comparisons demonstrated a large effect size. In addition to demonstrating practical significance, the improvements seen in the delayed intervention group show clinically significant effects. The results of delayed intervention group support the hypothesis that ILF NFB would improve in headache, sleep, and attentional symptoms above and beyond those results received while serving in the control group. Secondary outcomes measures captured broader impacts of ILF NFB on psychosocial functioning, mood, and general health, providing insight into the intervention's effect on overall well-being.

This study further validates existing research that has supported ILF NFB as an efficacious clinical intervention (Arina et al., 2022; Carlson et al., 2025; Carlson & Ross, 2021; Dobrushina et al., 2017; Legarda et al., 2022). This research study used ILF NFB successfully as an intervention for veterans with mTBI, who are well known to be a treatment-resistant group, for which effective treatments have been difficult to identify (Agimi et al., 2024). This research project also verifies that the use of the precision iterative, idiosyncratic approach to identifying each individual's ORF is an effective approach in ILF NFB. The implementation of the ILF NFB precision model by the providers, which was an individualized, precise data driven approach in determining the ORF for each participant, was an important aspect of the study as well, since finding the ORF resulted in the greatest symptom reduction by the participants.

To our knowledge, this is the only analysis of an ILF NFB delayed intervention group that was involved in a RCT. By conducting an additional analysis of control group members, subsequent exposure to ILF NFB provided another source of data that further serves to validate ILF NFB as an effective treatment. It was an ethical commitment of the investigators in this study to offer those participants randomized into the control group treatment even though it impacted time involvement and study costs. Throughout the study, the participants were encouraged to continue in treatment as usual and other treatments that may affect the study's variables of interest. Another strength of the study was the implementation of procedures to achieve an expert skill level among the ILF NFB providers, minimizing the confounding factor of provider proficiency level negatively impacting study outcomes. Even though the control group members were not mandated to continue into the delayed intervention group, most control group members choose to enter. Of note for participants who dropped out, the majority (85%) did so during the timeframe of COVID-19 (between March 2020 and May 2023). Loss of jobs, reassignments, change of schedules, and moving out of state were among the reasons for attrition. The demographic diversity of the sample, including a substantial proportion of biracial/multiracial participants, enhances the generalizability of findings to the broader veteran population. The low attrition rate and absence of intervention-related adverse events further support the feasibility and safety of ILF NFB.

A limitation of the present study's design is that the delayed intervention group had an extra 2 months of clinical contact, and continuous, therapeutic contact with a professional could have contributed to findings. There is also the potential that the expectations of participants in the intervention group were impacted by the novelty of ILF NFB technology. There is some evidence that taking part in a clinical trial using innovative technology may in and of itself bias participant expectations and thus, potentially, their outcomes (Lawton et al., 2019). While the delayed intervention group demonstrated significant improvement in symptoms over the course of study when compared to its results as a control group, results should be interpreted with these factors in mind.

Although the QIK-test measure of attention was statistically significant at end of treatment and at the 2-month follow-up, the small effect size result is of concern. This finding may be related to practice effects of completing the QIK test seven times by the participants across the course of the study. Of note,

recent research has indicated that the use of objective tests to capture attention issues in veterans with mTBI has not been found as relevant to the veteran experience as subjective measures (Ord et al., 2025).

Future research should focus on further understanding the factors that contribute to improvements seen in response to ILF NFB therapy. To explore whether and to what degree nonspecific effects of participating in this treatment impact participants' outcomes, a research design utilizing sham ILF NFB for the control condition, a technology now available, may be helpful. Given the substantial clinical effects evident in this study, fMRI studies may assist in understanding the brain connectivity pathways that may have renormalized with this treatment modality.

Based on the clinical impact observed in the present study, future research may also demonstrate utility of ILF NFB in broader populations other than veterans with mTBI in those areas where our results demonstrated potential for treatment, such as chronic sleep, headache, and attention disorders, as well as for other physiological and psychological issues that this study provided initial data.

Conclusion

ILF NFB holds promise to be a safe and effective intervention for those who continue to suffer with postconcussive symptoms of chronic headache, sleep, and attention disorders. Secondary outcomes from the present study also suggest that ILF NFB may be an effective intervention for symptoms associated with depression and PTSD, in addition to having a significant positive impact on subjective QOL in veterans with mTBI. Given the absence of intervention-related adverse events and the high rate of clinically significant improvement, ILF NFB should be considered for further study and potential integration into multidisciplinary care for veterans with persistent postconcussive symptoms.

Author Acknowledgments

The authors want to express deep appreciation and acknowledgement to the veterans who participated in the study, VAPIHCS Research and Development Service, especially Sedra Graves, BA, for all of her support during the 5 years of the study. A special acknowledgement to Siegfried Othmer, PhD, and the late Sue Othmer, BA, BCN, for their enormous contribution to the science and clinical development and use of ILF NFB. A very deep appreciation and acknowledgement is extended to Applied

Neurophysics for their gracious offer to provide our veterans with EEG Expert Reports for the QIK-Test results.

Author Declarations

This work was supported by Merit Review Award # NURC-002-19S from the United States Department of Veterans Affairs Clinical Science Research and Development Services. This funding source was not involved in any part of the development or execution of study or publication thereof. This study was registered in the United States Clinical Trials registry (NCT04195685). There are no financial interests nor any other type of conflicts of interest by any of the authors. There was no usage of AI in the development or production of the manuscript. Data available by request to corresponding author.

References

- Agimi, Y., Hai, T., Gano, A., Stuessi, K., Gold, J., Kaufman, R., & McKinney, G. (2024). Clinical trajectories of comorbidity associated with military-sustained mild traumatic brain injury: Pre- and post-injury. *Journal of Head Trauma Rehabilitation*, 39(6), E564–E575. <https://doi.org/10.1097/HTR.0000000000000934>
- Arina, G. A., Dobrushina, O. R., Shvetsova, E. T., Osina, E. D., Meshkov, G. A., Aziatskaya, G. A., Trofimova, A. K., Efremova, I. N., Martunov, S. E., & Nikolaeva, V. V. (2022). Infra-low frequency neurofeedback in tension-type headache: A cross-over sham-controlled study. *Frontiers of Human Neuroscience*, 16, Article 891323. <https://doi.org/10.3389/fnhum.2022.891323>
- Arns, M., & Kenemans, J. L. (2014). Neurofeedback in ADHD and insomnia: Vigilance stabilization through sleep spindles and circadian networks. *Neuroscience & Biobehavioral Reviews*, 44, 183–194. <https://doi.org/10.1016/j.neubiorev.2012.10.006>
- Ayalon, L., Borodkin, K., Dishon, L., Kanety, H., & Dagan, Y. (2007). Circadian rhythm sleep disorders following mild traumatic brain injury. *Neurology*, 68(14), 1136–1140. <https://doi.org/10.1212/01.wnl.0000258672.52836.30>
- Bastien, C., Vallieres, A., & Morin, C. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, 2(4), 297–307. [https://doi.org/10.1016/s1389-9457\(00\)00065-4](https://doi.org/10.1016/s1389-9457(00)00065-4)
- Benson, A., & LaDou, T. W. (2015). The use of neurofeedback for combat veterans with post-traumatic stress. In H. W. Kirk (Ed.), *Restoring the brain neurofeedback integrative approach to health (1st ed)*. CRC Press. <https://doi.org/10.1201/b18671>
- Berman, R., Spencer, H., Boese, M., Kim, S., Radford, K., & Choi, K. (2023). Loss of consciousness and righting reflex following traumatic brain injury: Predictors of post-injury symptom development (a narrative review). *Brain Sciences*, 13(5), Article 750. <https://doi.org/10.3390/brainsci13050750>
- Buzsáki, G., & Watson, B. O. (2012). Brain rhythms and neural syntax: Implications for efficient coding of cognitive content and neuropsychiatric disease. *Dialogues in Clinical Neuroscience*, 14(4), 345–367. <https://doi.org/10.31887/DCNS.2012.14.4/gbuzsaki>
- Carlson, J. (2021). *General symptom inventory* [Unpublished]. Clinical Practice Tool.
- Carlson, J., & Ross, G. W. (2021). Neurofeedback impact on chronic headache, sleep, and attention disorders experienced by veterans with mild traumatic brain injury: A pilot study.

- Applied Psychophysiology & Biofeedback*, 49(1), 2–9. <https://doi.org/10.5298/1081-5937-49.01.01>
- Carlson, J. Ross, G. W., Tyrrell, C., Fiamé, B., Nunokawa, C., Sirirwardhana, C., & Schaper, K. (2025). Infra-low frequency neurofeedback impact on post-concussive symptoms of headache, insomnia and attention disorder: Results of a randomized control trial. *EXPLORE*, 21(2), Article 103137. <https://doi.org/10.1016/j.explore.2025.103137>
- Cella, D., Lai, J. S., Nowinski, C. J., Victorson, D., Peterman, A., Miller, D., Bethoux, F., Heinemann, A., Rubin, S., Cavazos, J. E., Reder, A. T., Sufit, R., Simuni, T., Holmes, G. L., Siderowf, A., Wojna, V., Bode, R., McKinney, N., Podrabsky, T., ... Moy, C. (2012). Neuro-QOL brief measures of health-related quality of life for clinical research in neurology. *Neurology*, 78(23), 1860–1867.
- Chapman, J. C., & Diaz-Arastia, R. (2014). Military traumatic brain injury: A review. *Alzheimer's Dementia*, 10(3S), S97–S104. <https://doi.org/10.1016/j.jalz.2014.04.012>
- Coeysaers, R. R., Kaufman, J. S., Chao, R., Mann, J. D., & DeVellis, R. F. (2006). Four methods of estimating the minimal important difference score were compared to establish a clinically significant change in Headache Impact Test. *Journal of Clinical Epidemiology*, 59(4), 374–380. <https://doi.org/10.1016/j.jclinepi.2005.05.010>
- DeFina, P., Fellus, J., Polito, M. Z., Thompson, J. W. G., Moser, R. S., & DeLuca, J. (2009). The new neuroscience frontier: Promoting neuroplasticity and brain repair in traumatic brain injury. *The Clinical Psychologist*, 23(8), 1391–1399. <https://doi.org/10.1080/13854040903058978>
- Dobrushina, O., Arina, G., Osina, E., & Aziatskaya, G. (2017). Clinical and psychological confirmation of stabilizing effect of neurofeedback in migraine. *European Psychiatry*, 41(S1), S253–S253. <https://doi.org/10.1016/j.eurpsy.2017.02.045>
- Duff, J. (2004). The usefulness of quantitative EEG (QEEG) and neurotherapy in the assessment and treatment of post-concussion syndrome. *Clinical EEG Neuroscience*, 35, 198–209. <https://doi.org/10.1177/155005940403500410>
- Eapen, B. C., Bowles, A. O., Sall, J., Lang, A. E., Hoppes, C. W., Stout, K. C., Kretzmer, T., & Cifu, D. X. (2022). The management and rehabilitation of post-acute mild traumatic brain injury. *Brain Injury*, 36(5), 693–702. <https://doi.org/10.1080/02699052.2022.2033848>
- Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2013). Boosting brain functions: Improving executive functions with behavioral training, neurostimulation, and neurofeedback. *International Journal of Psychophysiology*, 88(1), 1–16. <https://doi.org/10.1016/j.ijpsycho.2013.02.001>
- Gaudette, É., Seabury, S. A., Temkin, N., Barber, J., DiGiorgio, A. M., Markowitz, A. J., Manley, G. T., & TRACK-TBI Investigators. (2022). Employment and economic outcomes of participants with mild traumatic brain injury in the TRACK-TBI study. *JAMA Network Open*, 5(6), Article e2219444. <https://doi.org/10.1001/jamanetworkopen.2022.19444>
- Ghaziri, J., Tucholka, A., Larue, V., Blanchette-Sylvestre, M., Reyburn, G., Gilbert, G., Lévesque, J., & Beauregard, M. (2013). Neurofeedback training induces changes in white and gray matter. *Clinical EEG Neuroscience*, 44(4), 265–272. <https://doi.org/10.1177/1550059413476031>
- Grin-Yatsenko, V., Othmer, S., Ponomarev, V., Evdokimov, S., Konoplev, Y., & Kropotov, J. (2018). Infra-low frequency neurofeedback in depression: Three case studies. *NeuroRegulation*, 5(1), 30–42. <https://doi.org/10.15540/nr.5.1.30>
- Haller, S., Kopel, R., Jhooti, P., Haas, T., Scharnowski, F., Lovblad, K. O., Scheffler, K., & Van De Ville, D. (2013). Dynamic reconfiguration of human brain functional networks through neurofeedback. *NeuroImage*, 81, 243–252. <https://doi.org/10.1016/j.neuroimage.2013.05.019>
- Hayes, J. P., Bigler, E. D., & Verfaellie, M. (2016). Traumatic brain injury as a disorder of brain connectivity. *Journal of International Neuropsychological Society*, 22(2), 120–137. <https://doi.org/10.1017/S1355617715000740>
- Hughes, J. R. (1994). *EEG in Clinical Practice (2nd edition)*. Butterworth-Heinemann.
- Huster, R. J., Mokom, Z. N., Enriquez-Geppert, S., & Herrmann, C. S. (2014). Brain-computer interfaces for EEG neurofeedback: Peculiarities and solutions. *International Journal of Psychophysiology*, 91(1), 36–45. <https://doi.org/10.1016/j.ijpsycho.2013.08.011>
- Ibric, V. L., Dragomirescu, L. G., & Hudspeth, W. J. (2009). Real-time changes in connectivities during neurofeedback. *Journal of Neurotherapy*, 13(3), 156–165. <https://doi.org/10.1080/10874200903118378>
- Kirk, H. W., & Dahl, M. G. (2022). Infra low frequency neurofeedback training for trauma recovery: A case report. *Frontiers of Human Neuroscience*, 16, Article 905823. <https://doi.org/10.3389/fnhum.2022.905823>
- Kosinski, M., Bayliss, M., Björner, Ware, J. E. Jr., Garber, W. H., Batenhors, A., Cady, R., Dahlöf, C. G., Dowson, A., & Tepper, S. (2003). A six-item short-form survey for measuring headache impact: The HIT-6. *Quality of Life Research*, 12(8), 963–974. <https://doi.org/10.1023/a:1026119331193>
- Koush, Y., Rosa, M. J., Robineau, F., Heinen, K., Rieger, S. W., Weiskopf, N., Vuilleumier, P., Van De Ville, D., & Scharnowski, F. (2013). Connectivity-based neurofeedback: Dynamic causal modeling for real-time fMRI. *NeuroImage*, 81, 422–430. <https://doi.org/10.1016/j.neuroimage.2013.05.010>
- Kozłowski, A. J., Cella, D., Nitsch, K. P., & Heinemann, A. W. (2016, April). Evaluating individual change with the Quality of Life in Neurological Disorders (Neuro-QoL) Short Forms. *Archives of Physical Medicine and Rehabilitation*, 97(4), 650–654.e8. <https://doi.org/10.1016/j.apmr.2015.12.010>
- Kroenke, K. (2012). Enhancing the clinical utility of depression screening. *Canadian Medical Association Journal*, 184(3), 281–282. <http://doi.org/10.1503/cmaj.112004>
- Kroenke, K., Spitzer, R. and Williams, J. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16(9), 606–613. <https://doi.org/10.1046/j.1525-1497.2001.016009606.x>
- Larsen, S., & Sherlin, L. (2013). Neurofeedback: An emerging technology for treating central nervous system dysregulation. *Psychiatric Clinics of North America*, 36(1), 163–168. <https://doi.org/10.1016/j.psc.2013.01.005>
- Lawton, J., Blackburn, M., Breckenridge, J., Hallowell, N., Farrington, C., & Rankin, D. (2019). Ambassadors of hope, research pioneers and agents of change—Individuals' expectations and experiences of taking part in a randomized trial of an innovative health technology: Longitudinal qualitative study. *Trials*, 20(1), Article 289. <https://doi.org/10.1186/s13063-019-3373-9>
- Legarda, S. B., Lahti, C. E., McDermott, D., & Michas-Martin, A. (2022). Use of novel concussion protocol with infralow frequency neuromodulation demonstrates significant treatment response in patients with persistent post-concussion symptoms: A retrospective study. *Frontiers of Human Neuroscience*, 16, Article 894758. <https://doi.org/10.3389/fnhum.2022.894758>
- Legarda, S. B., McMahon, D., Othmer, S., & Othmer, S. (2011). Clinical neurofeedback: Case studies, proposed mechanism, and implications for pediatric neurology practice. *Journal of Child Neurology*, 26(8), 1045–1051. <https://doi.org/10.1177/0883073811405052>
- Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy*, 33(3), 335–343. [https://doi.org/10.1016/0005-7967\(94\)00075-u](https://doi.org/10.1016/0005-7967(94)00075-u)
- MacGregor, A. J., Dougherty, A. L., Tang, J. J., & Galarneau, M. R. (2013). Postconcussive symptom reporting among US combat veterans with mild traumatic brain injury from

- Operation Iraqi Freedom. *The Journal of Head Trauma Rehabilitation*, 28(1), 59–67. <https://doi.org/10.1097/HTR.0b013e3182596382>
- May, G., Benson, R., Balon, R., & Boutros, N. (2013). Neurofeedback and traumatic brain injury: A literature review. *Annals of Clinical Psychiatry*, 25(4), 289–296.
- McMahon, D. (2020). Neurofeedback in an integrative medical practice. In H. W. Kirk (Ed.), *Restoring the brain neurofeedback as an integrative approach to health* (2nd ed.). Routledge.
- Miller, A. R., Martindale, S. L., Rowland, J. A., Walton, S., Talmy, T., & Walker, W. C. (2024). Blast-related mild TBI: LIMBIC-CENC focused review with implications commentary. *NeuroRehabilitation*, 55(3), 329–345. <https://doi.org/10.3233/NRE-230268>
- Monto, S., Palva, S., Voipio, J., & Palva, J. M. (2008). Very slow EEG fluctuations predict the dynamics of stimulus detection and oscillation amplitudes in humans. *The Journal of Neuroscience*, 28(33), 8268–8272. <https://doi.org/10.1523/JNEUROSCI.1910-08.2008>
- Morissette, S. B., Woodward, M., Kimbrel, N. A., Meyer, E. C., Kruse, M. I., Dolan, S., & Gulliver, S. B. (2011). Deployment related TBI, persistent post-concussive symptoms, PTSD, and depression in OEF/OIF veterans. *Rehabilitation Psychology*, 56(4), 340–350. <https://doi.org/10.1037/a0025462>
- Munivenkatappa, A., Rajeswaran, J., Bhagavatula, I. D., Bennet, N., & Upadhyay, N. (2014). EEG neurofeedback therapy: Can it attenuate brain changes in TBI? *NeuroRehabilitation*, 35(3), 481–484. <https://doi.org/10.3233/NRE-141140>
- Nelson, D. V., & Esty, M. L. (2012, Spring). Neurotherapy of traumatic brain injury/posttraumatic stress symptoms in OEF/OIF veterans. *Journal Neuropsychiatry Clinical Neuroscience*, 24(2), 237–240. <https://doi.org/10.1176/appi.neuropsych.11020041>
- Ord, A. S., Martindale, S. L., Jenks, E. R., & Rowland, J. A. (2025). Subjective cognitive complaints and objective cognitive functioning in combat veterans: Effects of PTSD and deployment mild TBI. *Applied Neuropsychology: Adult*, 32(5), 1400–1406. <https://doi.org/10.1080/23279095.2023.2280807>
- Othmer, S. (2023). Endogenous neuromodulation at infra-low frequencies. In D. R. Chertier, M. B. Dellinger, J. R. Evans, & H. K. Budzynski (Eds.), *Introduction to quantitative EEG and neurofeedback* (3rd ed.), Chapter 17, (pp. 283–299). Academic Press. <https://doi.org/10.1016/B978-0-323-89827-0.00001-2>
- Othmer, S., & Othmer, S. F. (2009). Post traumatic stress disorder—The neurofeedback remedy. *Biofeedback*, 37(1), 24–31. <https://doi.org/10.5298/1081-5937-37.1.24>
- Othmer, S., & Othmer, S. F. (2020). Toward a theory of infra-low frequency neurofeedback. In H. Kirk (Ed.), *Restoring the brain neurofeedback as an integrative approach to health* (2nd ed.). Taylor and Francis.
- Portz, J. M. P., Sherer, M., Kisala, M. A., Tulskey, D., Leon-Novelo, M., & Ngan, E. (2017). Responsiveness of the Traumatic Brain Injury-Quality of Life (TBI-QOL) Measurement System. *Archives of Physical Medicine and Rehabilitation*, 101(1), 54–61. <https://doi.org/10.1016/j.apmr.2017.11.018>
- Posner, K., Brown, G. K., Stanley, B., Brent, D. A., Yershova, K. V., Oquendo, M. A., Currier, G. W., Melvin, G. A., Greenhill, L., Shen, S., & Mann, J. J. (2011). The Columbia–Suicide Severity Rating Scale: Initial validity and internal consistency findings from three multisite studies with adolescents and adults. *American Journal of Psychiatry*, 168(12), 1266–1277. <https://doi.org/10.1176/appi.ajp.2011.10111704>
- Ronk, F. R., Korman, J. R., Hooke, G. R., & Page, A. C. (2013). Assessing clinical significance of treatment outcomes using the DASS-21. *Psychological Assessment*, 25(4), 1103–1110. <https://doi.org/10.1037/a0033100>
- Ros, T., Théberge, J., Frewen, P. A., Kluetsch, R., Densmore, M., Calhoun, V. D., & Lanius, R. A. (2013). Mind over chatter: Plastic up-regulation of the fMRI salience network directly after EEG neurofeedback. *NeuroImage*, 65, 324–335. <https://doi.org/10.1016/j.neuroimage.2012.09.046>
- Rowland, J. A., Stapleton-Kotloski, J. R., Godwin, D. W., Hamilton, C. A., & Martindale, S. L. (2024). The functional connectome and long-term symptom presentation associated with mild traumatic brain injury and blast exposure in combat veterans. *Journal of Neurotrauma*, 41(23–24), 2513–2527. <https://doi.org/10.1089/neu.2023.0315>
- Sakamoto, M. S., Delano-Wood, L., Schiehser, D. M., & Merritt, V. C. (2021). Predicting veteran health-related quality of life following mild traumatic brain injury. *Rehabilitation Psychology*, 66(4), 461–469. <https://doi.org/10.1037/rep0000392>
- Schiehser, D. M., Twamley, E. W., Liu, L., Matevosyan, A., Filoteo, J. V., Jak, A. J., Orff, H. J., Hanson, K. L., Sorg, S. F., & Delano-Wood, L. (2015). The relationship between post-concussive symptoms and quality of life in veterans with mild to moderate traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 30(4), E21–E28. <https://doi.org/10.1097/HTR.0000000000000065>
- Shapiro, E. J., & Prager, J. P. (2020). ILF neurofeedback and alpha-theta training in a multidisciplinary chronic pain program. In *Restoring the brain neurofeedback as an integrative approach to health* (2nd ed.). Routledge.
- Sharma, T. L. (2022). Returning to work after mild traumatic brain injury—Considering the impact of employer support. *JAMA Network Open*, 5(6), Article e2219454. <https://doi.org/10.1001/jamanetworkopen.2022.19454>
- Strang, J., & Chae, H. (2013). Poster 108 Neuropsychological rehabilitation to enhance quality of life, perceived control and psychological well-being. *Archives of Physical Medicine and Rehabilitation*, 94(10), Article E48.
- Theeler, B. J., & Erickson, J. C. (2012). Post-traumatic headache in military personnel and veterans of the Iraq and Afghanistan conflicts. *Current Treatment Options in Neurology*, 14, 36–49. <https://doi.org/10.1007/s11940-011-0157-2>
- Truelle, J.-L., Koskinen, S., Hawthorne, G., Sarajuuri, J., Formisano, R., Von Wild, K., Neugebauer, E., Wilson, L., Gibbons, H., Powell, J., Bullinger, M., Höfer, S., Maas, A., Zitnay, G., Von Steinbuechel, N., & The Qolibri Task Force (2010). Quality of life after traumatic brain injury: The clinical use of the QOLIBRI, a novel disease-specific instrument. *Brain Injury*, 24(11), 1272–1291. <https://doi.org/10.3109/02699052.2010.506865>
- Tulskey, D. S., Tyner, C. E., Boulton, A. J., Kisala, P. A., Heinemann, A. W., Roth, E. J., & Carlozzi, N.E. (2019). Development of the TBI-QOL headache pain item bank and short form. *Journal of Head Trauma Rehabilitation*, 34(5), 298–307. <https://doi.org/10.1097/HTR.0000000000000532>
- Versace, M. (2014). QIKTest report on EEG Expert: Introduction and overview. EEG Expert. https://www.eegexpert.net/index_content.asp?page=reports
- Voormolen, D., Cnossen, M., Polinder, S., von Steinbuechel, N., Vos, P. E., & Haagsma, J. A. (2018). Divergent classification methods of post-concussion syndrome after mild traumatic brain injury: Prevalence rates, risk factors, and functional outcome. *Journal of Neurotrauma*, 35(11), 1233–1241. <https://doi.org/10.1089/neu.2017.5257>
- Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). *The PTSD checklist for DSM-5 (PCL-5)*. National Center for PTSD. <https://www.ptsd.va.gov/>
- Whiteneck, G., Williams, W., Almeida, E., Bidelsbach, D., Culpepper, W., Picon, L. M., Eagye, C. B., & Dr Mellick, D. (2024). Two decades of Department of Veterans Affairs traumatic brain injury care and benefits for veterans of post-9/11 conflicts. *Journal of Head Trauma Rehabilitation*, 39(5), E462–E469. <https://doi.org/10.1097/HTR.0000000000000952>

Yang, M., Morin, C. M., Schaefer, M., & Wallenstein, G. V. (2009). Interpreting score differences in the Insomnia Severity Index: Using health-related outcomes to define the minimally important difference. *Current Medical Research and Opinion*, 25(10), 2487–2494. <https://doi.org/10.1185/03007990903167415>

Zoefel, B., Huster, R. J., & Herrmann, C. S. (2012). Neurofeedback training of the upper alpha frequency band in EEG improves cognitive performance. *NeuroImage*, 54(2), 1427–1431. <https://doi.org/10.1016/j.neuroimage.2010.08.078>

Received: October 30, 2025

Accepted: March 28, 2026

Published: September 28, 2026

Efficacy of Neurofeedback in Adults With Posttraumatic Stress Disorder

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Abstract

Objective. To evaluate, under PRISMA, the clinical efficacy, safety/acceptability, and mechanistic evidence of neurofeedback (NF) for adult posttraumatic stress disorder (PTSD), stratified by modality and comparator, and to meta-analyze posttreatment severity. **Method.** I conducted an a priori systematic review following PRISMA 2020 and PRISMA-S. Sources were MEDLINE/PubMed, Cochrane CENTRAL, PsycINFO, ClinicalTrials.gov, WHO-ICTRP, and medRxiv, plus citation chasing. Eligible studies were parallel randomized controlled trials in adults with PTSD, comparing NF versus wait-list/treatment-as-usual (TAU), active, or sham. The primary outcome was posttreatment PTSD severity. Risk of bias (RoB 2) and certainty (GRADE) were assessed. Random-effects meta-analysis used REML with Knapp–Hartung adjustment. **Results.** Five trials met inclusion; three provided comparable data for pooling. The combined effect favored NF with minimal heterogeneity. Larger clinical signals appeared versus wait-list/TAU; double-blind sham/active contrasts were smaller or inconclusive yet showed mechanistic change (DMN/SN normalization; amygdala down-regulation). No NF-related serious adverse events were reported; dropout was low–moderate. GRADE certainty for posttreatment severity was low–moderate. **Conclusions.** NF is a promising, well-tolerated adjunct for adult PTSD; larger double-blind trials with active/sham comparators, longer follow-up, and standardized protocols are needed to establish specificity and durability of effects.

Keywords: neurofeedback; EEG-neurofeedback; trauma; PTSD; EEG; fMRI; randomized controlled trial

Citation: González-Rivera, J. A. (2026). Efficacy of neurofeedback in adults with posttraumatic stress disorder. *NeuroRegulation*, 13(3), 256–269. <https://doi.org/10.15540/nr.13.3.256>

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Posttraumatic stress disorder (PTSD) is more than a diagnostic category; it is a condition that persistently undermines well-being, relationships, and social participation. At the population level, most people will experience at least one potentially traumatic event across their lives; only a fraction will develop PTSD, with lifetime prevalence between 3% and 6%, depending on exposure type and sociocultural context (Kessler et al., 2017; Koenen et al., 2017). The functional burden is substantial: PTSD is associated with psychiatric and medical comorbidity, occupational and relational impairment, and greater use of health services. Its impact is also unevenly distributed. Armed conflict, interpersonal violence, and disasters—together with gaps in access to mental health care—raise risk and deepen inequities (Kessler et al., 2017; Koenen et al., 2017).

Current practice guidelines prioritize trauma-focused psychotherapies—including trauma-focused cognitive behavioral therapy, cognitive processing therapy, and prolonged exposure—and, when indicated, selective serotonin reuptake inhibitors (SSRI) or serotonin and norepinephrine reuptake inhibitors (SNRI) as alternatives or adjuncts (American Psychological Association [APA], 2017; Hamblen et al., 2019; U.S. Department of Veterans Affairs [VA], 2023). Yet real-world care shows partial/nonresponse and dropout rates near 20%, with variability across settings and populations (Varker et al., 2021). This gap—together with the phenotypic heterogeneity of PTSD (e.g., dissociation, sustained hyperarousal, sleep and emotion-regulation disturbances)—underscores the need for adjunctive strategies that engage

neurobiological mechanisms, enhance self-regulation, and improve adherence.

Within this landscape, neurofeedback (NF) has gained traction as a brain self-regulation intervention grounded in real-time feedback of neural signals. Broadly, NF translates a brain marker—oscillatory power in EEG or BOLD signal in fMRI—into contingent sensory feedback and reinforces changes in the desired direction, enabling volitional control over a target activity (Enriquez-Geppert et al., 2017; Sitaram et al., 2017). In EEG-NF, protocols typically train frequency bands (alpha, theta, SMR), normalized indices (z-score), or connectivity metrics; in real-time fMRI (rt-fMRI) NF, training may target limbic regions (e.g., amygdala), networks, or multivariate patterns. Despite technical diversity, all variants share the principle of practice-dependent learning: repeated signal-feedback pairing fosters neuroplasticity that—ideally—generalizes beyond the training context.

The neurobiological plausibility of NF in PTSD aligns with large-scale network models. Reproducible alterations have been documented in the default mode network (DMN), the salience network (SN), and the central executive network (CEN): DMN relates to self-referential processing and self-coherence; a hyper- or hyporeactive SN to threat-detection biases and dysregulated interoception; and CEN deficits to impaired cognitive control and emotion regulation. This framework helps explain core PTSD symptoms—hypervigilance, re-experiencing, avoidance, affective numbing, and dissociation—and supports approaches that recalibrate balance between prefrontal systems and limbic substrates (Lanius et al., 2015). Under this logic, NF operates as a circuit-targeted intervention aimed at restoring activation and connectivity patterns.

Progress in the field has been methodological as well as technical. The CRED-nf checklist summarizes best practices to strengthen internal validity: pre-registration, justified sample sizes, appropriate controls (including sham where feasible), blinding—ideally double-blind for fMRI-NF—a priori specification of clinical and neurophysiological outcomes, and explicit learning criteria that demonstrate actual control over the target signal (Ros et al., 2020). In rt-fMRI-NF, methodological reviews emphasize critical parameters—feedback latency, temporal smoothing, signal quality, interface design—and the need for transfer tests without feedback to demonstrate generalization (Fede et al., 2020). This rigor is not

merely formal: in contexts where nonspecific factors (expectancy, therapeutic interaction, attention, motivation) can contribute to improvement, controlling for them is essential to isolate specific effects (Thibault & Raz, 2017).

Safety/acceptability is another key axis. Available evidence suggests good tolerability and low frequency of serious adverse events; even so, standardized monitoring and systematic reporting are recommended (Luctkar-Flude & Groll, 2015). In PTSD—where interventions centered on traumatic memory can be emotionally demanding—an adjunct that moderates hyperarousal and supports autonomic regulation could improve therapeutic engagement and retention in first-line psychotherapies (APA, 2017; Hamblen et al., 2019; VA, 2023).

Despite its promise, the literature points to four tensions that justify a rigorous review. First, heterogeneity in protocols—EEG band-based, z-score/LORETA, multivariate approaches in fMRI—dose (number/duration of sessions), target signals, and populations complicates direct comparison and quantitative synthesis without stratifying by modality and comparator. Second, designing credible sham and active comparators remains challenging: sham must avoid inadvertently training the same network, and active comparators (e.g., cardiovascular biofeedback) are essential to distinguish specific from nonspecific effects (Ros et al., 2020; Thibault & Raz, 2017). Third, verifying learning and its transfer to clinical contexts requires objective metrics beyond symptom reduction (e.g., volitional control in no-feedback tasks or ecological evidence). Fourth, integrating mechanistic markers (e.g., DMN/SN/CEN connectivity changes, amygdala modulation) as mediators or coprimary outcomes can clarify how and for whom NF adds value (Fede et al., 2020; Lanius et al., 2015).

Finally, frameworks such as Research Domain Criteria (RDoC) promote a transdiagnostic, circuit-centered view (e.g., negative valence or threat, arousal or regulatory systems, cognitive control). Aligning NF targets with RDoC domains—e.g., assessing the extent to which training reduces salience hyperreactivity or strengthens executive control—facilitates mechanistic hypotheses, response prediction, and dose optimization by subpopulation (Cuthbert, 2014; Morris et al., 2014). In a disorder as heterogeneous as PTSD, this stratification is particularly pertinent.

In summary, this review is motivated by four factors: (a) unmet clinical need in PTSD despite first-line care; (b) network/circuit plausibility for NF; (c) recent methodological advances; and (d) a favorable safety profile. I synthesize clinical efficacy and mechanistic evidence in adults, stratified by modality (EEG vs. fMRI) and comparator (wait-list, active, sham), and appraise safety/acceptability. My goal is to determine—rigorously and transparently—the extent to which NF improves PTSD symptoms and which neurobiological mechanisms may account for its effects, to inform practice and set priorities for future research.

Methods

Study Design

I conducted a systematic review to address a focused clinical question: to what extent is NF effective, safe/acceptable, and mechanistically supported for adult PTSD? The review was planned a priori and followed PRISMA 2020 (Page et al., 2021) and PRISMA-S (Rethlefsen et al., 2021). Before screening, I specified the PICO question, eligibility criteria, outcome hierarchy, and a statistical plan (narrative synthesis and meta-analysis when appropriate). Because I am the sole author, I

implemented safeguards to mitigate selection/extraction bias: a two-time-point pass for key steps, an operational decision guide, and a decision log.

Protocol and Process Documentation

The a priori protocol guided all phases—information sources and per-database strategies; two-stage screening and deduplication; a structured extraction template with prespecified derivation rules; outcome-level RoB 2; and the statistical plan (effect measures, random-effects modelling, heterogeneity and sensitivity analyses, and GRADE).

Protocol Transparency. The a priori protocol—including PICO, eligibility criteria, verbatim search strings, outcomes, and the statistical plan—is fully disclosed within this article (Table 1 and Methods). To mitigate risks of post hoc decisions, Table 1 provides an operational summary of each component together with a dated “Deviations/Notes” column that records clarifications and their rationale; no deviation altered the primary contrast or the prespecified outcome hierarchy. Verbatim search strings and search dates per source are listed in Table 2.

Table 1 <i>A Priori Protocol (Operational Summary) With Deviations/Notes</i>		
Component	Operational specification	Deviations/Notes
PICO and objectives	Adults (≥18) with PTSD; NF (EEG-bands/z-score/LORETA or connectivity; EEG amygdala Electrical FingerPrint [amyg-EFP]; rt-fMRI-NF) vs. wait-list/TAU, active, or sham. Primary outcome: posttreatment PTSD severity (CAPS-5 > CAPS-IV > PCL-5 > PCL). Secondary outcomes: remission; depression/anxiety; function/quality of life; adverse events; acceptability (dropout).	2025-10-25: First-use definition of amyg-EFP added in Eligibility. No change to PICO or outcome hierarchy.
Eligibility criteria	Parallel RCTs; Spanish/English; follow-ups analyzed separately. Exclude non-RCTs, pediatric samples, case series/reports, nonretrievable data, and combined interventions when the specific NF effect is not isolable.	2025-10-25: Clarified exclusion of nonseparable combined interventions and language scope (ES/EN). No impact on primary analysis.

Table 1*A Priori Protocol (Operational Summary) With Deviations/Notes*

Component	Operational specification	Deviations/Notes
Information sources	MEDLINE/PubMed; Cochrane CENTRAL; APA PsycNet (PsycINFO); ClinicalTrials.gov; WHO-ICTRP; medRxiv; backward/forward citation chasing.	2025-10-25: PRISMA counts reconciled by “lanes” (databases/registers vs other searches). Search dates per source documented in Table 2.
Search strategies (PRISMA-S)	Hybrid approach (MeSH/Thesaurus + free text); trial-oriented operators (random, trial, sham); RCT filters when feasible; verbatim strings and dates reported in Table 2.	2025-10-25: Harmonized “wait-list/TAU” spelling; ensured verbatim strings are included. No substantive change to logic.
Screening and selection	Two-stage screening (title/abstract → full text); deduplication by DOI/PMID/registry with manual check; one exclusion reason per full text; study-level consolidation for multiple reports.	2025-10-25: PRISMA numbers specified in text: duplicates = 9; records screened (left lane) = 273; other sought = 1, assessed = 1, excluded (protocol) = 1.
Data extraction	Structured template capturing design/context, sample/diagnosis, intervention/comparator (sessions; minutes/session), outcomes, AEs/acceptability; derivation rules (CI→SD; median/range/IQR→mean/SD; CI→log[HR]/SE).	2025-10-25: Table 3 expanded with “Sessions (<i>n</i>)”, “Minutes per session”, “Withdrawals (NF/Control)”; AE terms aligned to each trial’s definitions.
Effect measures	SMD (Hedges’ <i>g</i>) for continuous outcomes; MD when all trials share the same scale; RR for dichotomous; HR for time-to-event. Direction coded consistently (negative = favors NF); 95% CIs reported.	2025-10-25: Sign convention and scale hierarchy made explicit; no analytic change.
Synthesis plan	Primary analysis stratified by comparator (NF vs wait-list/TAU; NF vs active/sham). Random-effects (REML + Knapp–Hartung); report <i>Q</i> , <i>I</i> ² , <i>τ</i> ² and 95% prediction interval (PI) when <i>k</i> ≥ 3 (IntHout et al., 2016). Across-comparator pooled estimate treated as exploratory.	2025-10-25: Elevated comparator-stratified analysis to primary; added 95% PI reporting. Global pool labeled exploratory.
Risk of bias (RoB 2)	RoB 2 by outcome (posttreatment severity; follow-up when available); particular attention to blinding and comparator type.	2025-10-25: Clarified that RoB 2 informed sensitivity sets and GRADE.
Certainty of evidence (GRADE)	Certainty by outcome; downgrades for risk of bias, imprecision, indirectness, publication bias.	2025-10-25: Linked indirectness to comparator/modality mixing and imprecision to small samples/near-null CIs.

Note. The protocol was established a priori and guided all stages of the review in accordance with PRISMA 2020 and PRISMA-S. Locations of each protocol component are detailed across Methods; this embedded, dated ‘Deviations/Notes’ column makes the plan auditable within the article.

Eligibility Criteria

I included parallel randomized controlled trials (RCTs) enrolling adults (≥18 years) with PTSD diagnosed by standardized interview or validated instruments. The intervention had to be NF for PTSD (EEG-NF by bands/z-score/LORETA or connectivity; EEG amygdala Electrical FingerPrint (amyg-EFP); rt-fMRI-NF). Accepted comparators were wait-list/TAU, credible sham, or active (e.g., HRV-biofeedback). The primary outcome was posttreatment PTSD severity, with a prespecified hierarchy CAPS-5 > CAPS-IV > PCL-5 > PCL; secondary outcomes were remission, depression/anxiety, function/quality of life, adverse events, and acceptability (all-cause dropout). Follow-ups (≥ 1–3 months) were analyzed separately. I excluded nonrandomized studies, pediatric samples, case series/case reports, abstracts without retrievable data, and combined interventions when the specific effect of NF was not isolable. No date limits; languages Spanish/English. Operational rules included prioritizing the primary measure or the most standardized scale per the hierarchy; using between-group posttreatment differences as the main contrast (pre–post changes only as sensitivity when arm-level post means/SDs

were missing); combining like arms to avoid double counting; and deduplicating before screening.

Information Sources and Search Strategies

To maximize coverage and minimize omissions, I searched—planned a priori—MEDLINE/PubMed, Cochrane CENTRAL, and APA PsycNet (PsycINFO); for ongoing/unpublished trials, ClinicalTrials.gov and WHO-ICTRP; and, for grey literature, medRxiv. Per the Cochrane Handbook (Higgins et al., 2023), I complemented this with backward citation chasing (reference lists) and forward citation searching (cited-by). Searches were designed a priori with a hybrid approach (MeSH/Thesaurus + free text), adapted to each portal’s syntax, and recorded verbatim (exact strings, fields/operators, limits), complying with PRISMA-S (Page et al., 2021; Rethlefsen et al., 2021). Across sources, I combined PTSD and NF terms (including EEG, LORETA, amyg-EFP, rt-fMRI), with operators that prioritize trials (random, trial, sham) and, when feasible, RCT filters. Table 2 provides dates and verbatim strings for each source to ensure unambiguous reproducibility.

Table 2
Bibliographic Search (PRISMA-S): Query Strings by Source, Fields, and Limits

Source/portal	Exact string	Fields / operators	Limits / filters	Date & time
MEDLINE / PubMed	("post-traumatic stress disorder" OR PTSD[Title/Abstract] OR "posttraumatic stress"[Title/Abstract]) AND (neurofeedback[Title/Abstract] OR "EEG neurofeedback"[Title/Abstract] OR LORETA[Title/Abstract] OR "z-score"[Title/Abstract])	Title / Abstract, MeSH; AND/OR; quotation marks	Study type when available	25-Oct-2025, 11:40
Cochrane CENTRAL	(PTSD OR "post-traumatic stress") AND (neurofeedback OR LORETA)	Title / Abstract; RCT filters	RCT; humans	25-Oct-2025, 11:55
PsycINFO	AB((posttraumatic stress disorder OR PTSD) AND (neurofeedback OR LORETA))	Thesaurus + AB/TI	Adults	25-Oct-2025, 12:20
ClinicalTrials.gov	PTSD AND (neurofeedback OR EEG OR LORETA OR fMRI OR amygdala OR EFP)	Registry fields	Interventional; adults	25-Oct-2025, 13:00
WHO-ICTRP	PTSD AND (neurofeedback OR EEG OR LORETA)	Simple term search	Trials; adults	25-Oct-2025, 13:10
medRxiv	"PTSD and neurofeedback"	Free text	–	25-Oct-2025, 13:20

Note. All searches were conducted in Spanish and English.

Screening and Study Selection

I applied a two-stage workflow (title/abstract → full text). Deduplication used DOI/PMID/registry IDs with manual verification; multiple reports of the same trial were consolidated at the study level. Each full-text exclusion received a single, explicit reason (e.g., nonseparable combined intervention; inadequate comparator; nonclinical outcomes; duplicate; protocol without results). As the sole author, I used a two-time-point pass, an operational guide, and a decision log. The PRISMA figure reports records identified, deduplicated, screened, assessed, excluded with reason, and included; the exclusion table lists reasons.

Data Extraction

I planned extraction a priori and executed it with a standardized template to ensure traceability and consistency. I captured design/context (year, country, registry, RCT type, blinding), sample/diagnosis (*n* per arm, age, sex, PTSD criteria/method), intervention/comparator (NF modality, target, number/duration of sessions, parameters; wait-list/TAU, sham, active), and outcomes (instruments, time points, results), plus adverse events and acceptability. For continuous outcomes (CAPS-5/CAPS-IV, PCL-5/PCL), I recorded means, *SDs*, and *n* per arm at post; when only pre–post changes were available, I used them as sensitivity. For dichotomous outcomes (remission, AEs) I recorded events/total; for time-to-event, HR with 95% CIs/SE. To preserve comparability, I applied the scale hierarchy and prioritized the primary measure (Higgins et al., 2023). For missing statistics, I derived *SD* from CI/SE (algebraic inversion); estimated mean/*SD* from median/range/IQR (Wan et al., 2014); and obtained log(HR)/SE from CI or *p* (Higgins et al., 2023). I checked internal consistency across text, tables, and figures; when discrepant, I privileged tables and documented decisions. As sole author, I used double-pass checks, predefined rules, and cross-checks (e.g., sum of arm *n* vs. total *N*; reported “dose” vs. session counts).

Effect Measures and Statistical Synthesis

For continuous outcomes, I used standardized mean differences (Hedges’ *g*) with small-sample correction; when all trials within a contrast used the same scale (e.g., CAPS), I preferred mean differences (MD) to retain unit interpretability (Hedges & Olkin, 1985; Higgins et al., 2023). Effect direction was coded consistently (negative values = lower severity favoring NF), and I reported 95% CIs. For dichotomous outcomes, I used risk ratios (RR); for time-to-event, hazard ratios (HR; or log[HR]/SE

derived when needed) following standard transformations (Higgins et al., 2023).

The primary synthesis was stratified by comparator—NF vs wait-list/TAU and NF vs active/sham—to minimize indirectness in clinical interpretation. When two or more clinically comparable trials were available within a stratum, I performed random-effects meta-analysis, estimating τ^2 with REML (Viechtbauer, 2005) and using Knapp–Hartung adjustments for confidence intervals; as sensitivity, I re-estimated with DerSimonian–Laird (DerSimonian & Laird, 1986; Knapp & Hartung, 2003). I reported *Q*, I^2 , τ^2 , and—when $k \geq 3$ —a 95% prediction interval (PI) for the pooled effect (IntHout et al., 2016). Subgroups were prespecified (NF modality; comparator; population: veterans vs. civilians), and meta-regression was considered only if $k \geq 10$ (moderators: number of sessions, blinding, NF target, RoB; Thompson & Higgins, 2002). I assessed robustness via leave-one-out analyses, exclusion of studies with high overall RoB 2, MD instead of SMD when a common scale was used, and *r* pre–post = 0.30/0.50/0.70 scenarios when deriving change *SDs*. Small-study/publication bias tests (funnel, Egger/Begg; Begg & Mazumdar, 1994; Egger et al., 1997) were planned only if $k \geq 10$; in case of asymmetry, trim-and-fill was treated as exploratory (Duval & Tweedie, 2000). All analyses were conducted in *R* (metafor, meta) with verification in Python (statsmodels), aiming for stable, clinically interpretable, and transparent estimates.

Risk of Bias Assessment

I assessed risk of bias with RoB 2 by outcome (posttreatment severity and, when available, follow-up), per the Cochrane Handbook and the original guidance (Higgins et al., 2023; Sterne et al., 2019). The five domains (randomization; deviations; missing data; outcome measurement; selective reporting) were judged low, some concerns, or high, with textual justifications and source (table/figure/registry). Particular attention was paid to blinding and comparator type (better profile with sham/active than with TAU/wait-list). These judgments informed the narrative, sensitivity analyses, and GRADE; when feasible, overall risk (low vs. nonlow) was explored as a moderator. Nonrandomized studies cited for context (e.g., safety/mechanism) were assessed with ROBINS-I (Sterne et al., 2016) and not meta-analyzed.

Certainty of Evidence (GRADE)

I rated certainty per outcome using GRADE: starting at high for RCTs and downgrading for risk of bias,

inconsistency, indirectness, imprecision, and publication bias; exceptionally, upgrading was considered for large effects or dose–response (Guyatt et al., 2011; Schünemann et al., 2022). The SoF table presents point estimates, 95% CIs, certainty (high/moderate/low/very low), and rationale notes.

Handling Missing Data and Multiplicity

When data were missing, I prioritized extraction from the article (text, tables, figures, supplements) and, as needed, applied standardized transformations (CI/SE→SD; median/range/IQR→mean/SD; CI→log(HR)/SE). Multiplicity was managed a priori: scale hierarchy (CAPS-5 > CAPS-IV > PCL-5 > PCL), primary measure when duplicates existed, combination of like arms, and posttreatment as the primary contrast (pre–post changes used only as sensitivity). This approach balances rigor and comparability and minimizes post hoc choices (Higgins et al., 2023; Wan et al., 2014).

Ethics and Transparency

The review used public sources and did not require ethics approval. A priori planning, verbatim

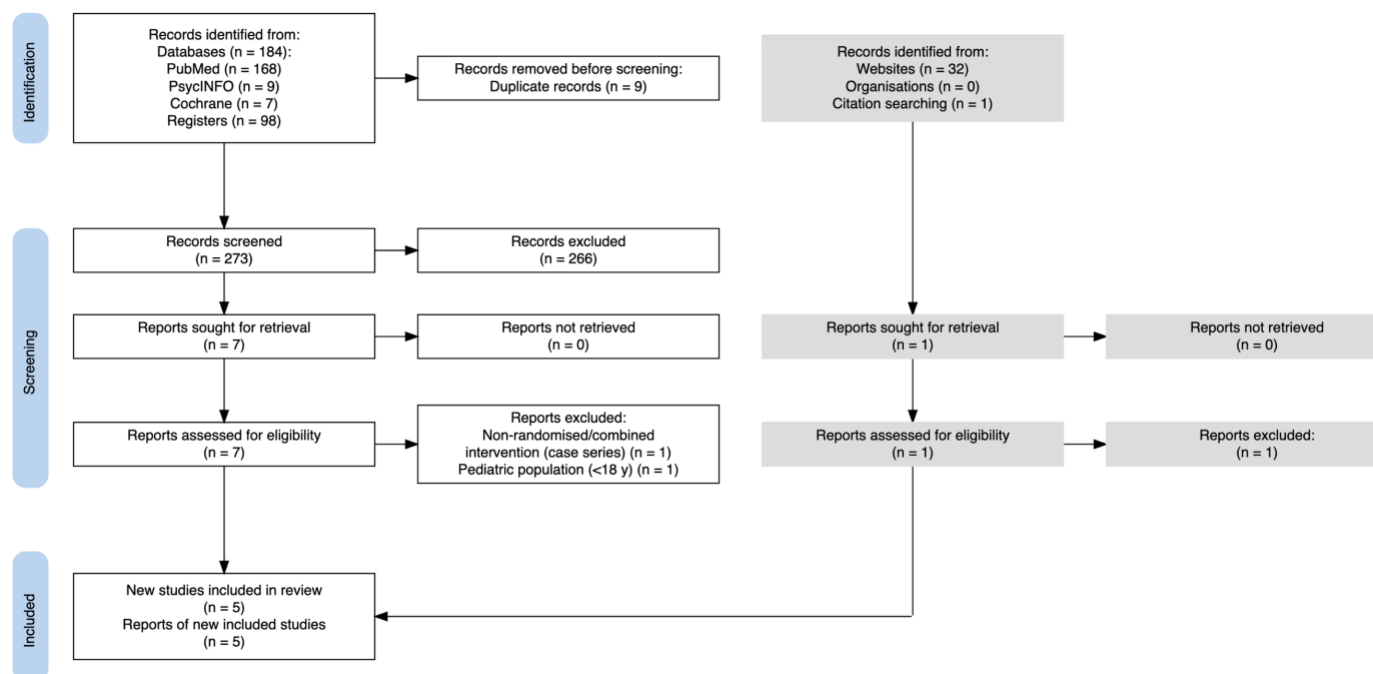
documentation of search strategies (PRISMA-S), and recognized tools (RoB 2, GRADE) ensure transparency and auditability (Page et al., 2021; Rethlefsen et al., 2021). All relevant methodological decisions—derivation rules, outcome hierarchies, and eligibility criteria—are reported within the manuscript and tables so the process can be readily verified.

Results

Study Selection

The identification, screening, and eligibility process followed PRISMA 2020 and is summarized in Figure 1. After deduplication and strict application of a priori criteria, five randomized controlled trials (RCTs) in adults with PTSD were included in the qualitative synthesis, of which three contributed comparable data to the quantitative synthesis for the posttreatment severity outcome. Full-text exclusions were assigned a single, verifiable reason (e.g., nonrandomized study with a combined intervention, pediatric population, protocol/registered report without results), ensuring traceability.

Figure 1. PRISMA 2020 Flow Diagram of the Study Selection Process.



Characteristics of Included Studies

The five RCTs evaluated NF in adults with PTSD and included (a) EEG-NF by bands versus wait-list/TAU (Leem et al., 2021; van der Kolk et al., 2016); (b) LORETA z-score NF versus an active control (HRV-biofeedback; Bell et al., 2019); (c) EEG alpha-rhythm NF versus sham (Nicholson et al., 2020); and (d) real-time fMRI NF targeting amygdala versus sham (Zhao et al., 2023). Protocols ranged from 3 to 24 sessions (≈30–60 min) and employed standardized posttreatment severity measures. Safety and acceptability (standardized). No NF-related serious adverse events were reported in any trial. All-cause withdrawals were low to moderate and generally comparable across arms; where counts were available (e.g., van der Kolk et al., 2016: 6/24 vs. 2/22), findings did not suggest excess attrition in NF groups. Because several trials did not report denominators consistently, a pooled RR was not computed; Table 3 reports withdrawal

counts (NF/Control) and session dose (sessions; minutes per session) for transparency. Table 3 also summarizes, without redundancy, the essential features (modality, dose, comparators, instruments, and time points).

Risk of Bias (RoB 2)

Outcome-level appraisal used RoB 2 according to the Cochrane Handbook. Table 4 presents domain-level judgments: trials with wait-list/TAU comparators showed some concerns/high risk in deviations from intended interventions and measurement of the outcome (lack of blinding and reliance on self-report), whereas sham, double-blind RCTs (Nicholson et al., 2020; Zhao et al., 2023) showed low risk in randomization and outcome measurement, with the caveat of modest sample sizes. These judgments informed sensitivity analyses (excluding studies with high overall RoB) and the GRADE rating.

Table 3
Characteristics of Included Randomized Controlled Trials

Study (country)	Design / blinding	NF modality and dose	Comparator	N (NF / C)	Primary outcome (time)
van der Kolk et al., 2016 (USA)	Parallel RCT; partial assessor blinding	EEG-NF, 24 sessions over 12 weeks	Wait-list / TAU	24 / 22	CAPS (post; +1-month follow-up)
Leem et al., 2021 (Korea)	Parallel RCT; assessor-blinded	NSRT (EEG-NF), 16 sessions over 8 weeks	Wait-list	10 / 9	PCL-5-K (post)
Bell et al., 2019 (USA)	Parallel RCT; open-label	LORETA z-score NF, 15 sessions	Active control (HRV-biofeedback)	12 / 11	PCL-5 (post)
Nicholson et al., 2020 (Canada)	Parallel RCT; double-blind	EEG alpha-rhythm NF, ~20 weeks	Sham EEG	18 / 18	CAPS-5 (post; 3-month follow-up)
Zhao et al., 2023 (China)	Parallel RCT; double-blind	rt-fMRI amygdala down-regulation, 3 sessions	Sham	14 / 11	fMRI amygdala control (primary); PCL (secondary)

Note. NF = neurofeedback; RCT = randomized controlled trial; TAU = treatment as usual; WL = wait-list; EEG-NF = electroencephalography-based neurofeedback; LORETA = low-resolution brain electromagnetic tomography; HRV = heart-rate variability; rt-fMRI = real-time functional MRI; CAPS = Clinician-Administered PTSD Scale; PCL-5 = PTSD Checklist for DSM-5; DMN/SN = default-mode/salience networks.

Table 4*Risk of Bias (RoB 2) by Domain for the Primary Posttreatment Clinical Outcome*

Study (country)	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall risk of bias
Bell et al., 2019 (USA)	Some concerns	Some concerns	Low	Some concerns	Low	Some concerns
Leem et al., 2021 (Korea)	Low	Some concerns	Low	Some concerns	Low	Some concerns
Nicholson et al., 2020 (Canada)	Low	Low	Low	Low	Low	Low
van der Kolk et al., 2016 (USA)	Some concerns	High	Low	Some concerns	Low	High
Zhao et al., 2023 (China)	Low	Low	Low	Low	Low	Low

Quantitative Synthesis

Primary Stratified Analysis. For NF versus wait-list/TAU (two RCTs), the pooled effect was $g = -1.07$ (95% CI [-1.59, -0.55]); the 95% PI was not computed for $k = 2$. For NF versus an active comparator (one RCT with comparable posttreatment means), the study-level effect was $g = -0.97$ (95% CI [-1.83, -0.10]); additional *sham* RCTs reported mechanistic improvements but lacked arm-level post means/SDs for pooling

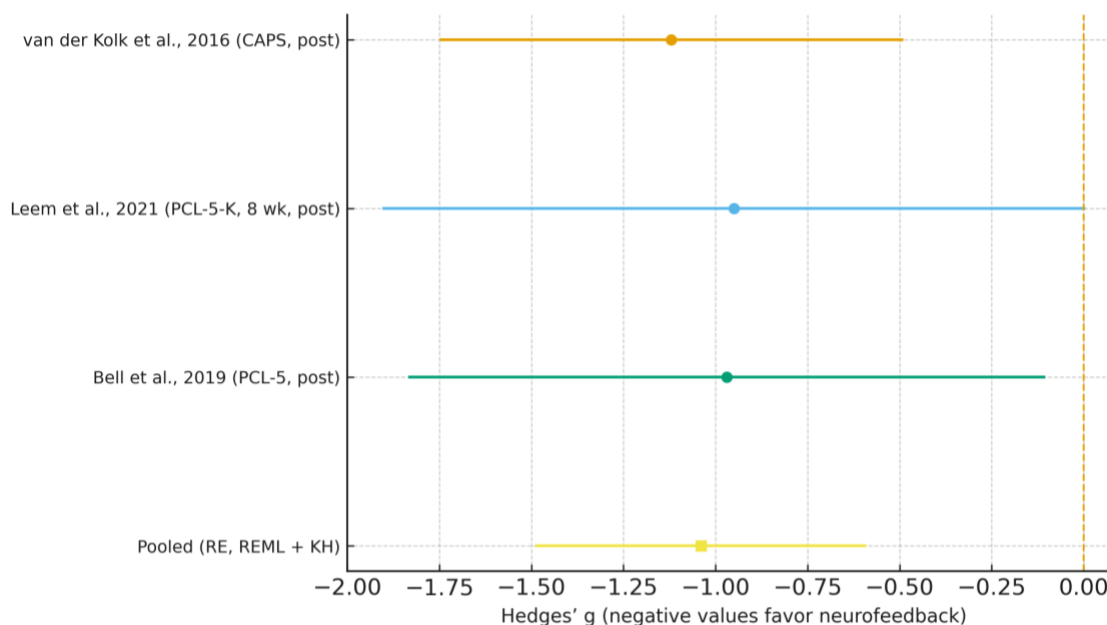
(Nicholson et al., 2020; Zhao et al., 2023). As an exploratory across-comparator estimate, the pooled effect was $g = -1.04$ (95% CI [-1.49, -0.59]; 95% PI [-6.40, 4.32]) with minimal heterogeneity ($Q = 0.119$; $I^2 = 0\%$; $\tau^2 = 0.0000$). Re-estimation with DerSimonian–Laird did not change direction or magnitude. No small-study asymmetry tests (Egger/Begg) were performed due to $k < 10$. Table 5 reports study-level effects and pooled estimates; Figure 2 shows the corresponding forest plot.

Table 5*Posttreatment Effects (Hedges' g) by Study and Pooled Estimate*

Study (country)	Scale (time)	N (NF / Control)	Hedges' g	95% CI
Bell et al., 2019 (USA)	PCL-5 (post)	12 / 11	-0.97	[-1.83, -0.10]
Leem et al., 2021 (Korea)	PCL-5-K (8 weeks, post)	10 / 9	-0.95	[-1.90, 0.01]
van der Kolk et al., 2016 (USA)	CAPS (post)	24 / 22	-1.12	[-1.74, -0.48]
Pooled (random-effects, REML + Knapp–Hartung)			-1.04	[-1.49, -0.59]

Note. Heterogeneity for the exploratory across-comparator pool: $Q = 0.119$, $df = 2$; $I^2 = 0\%$; $\tau^2 = 0.0000$. The pooled contrast NF vs wait-list/TAU (Leem et al., 2021; van der Kolk et al., 2016) yielded $g = -1.07$ ($\tau^2 \approx 0$). Trials with sham contributed mechanistic outcomes but lacked comparable arm-level post means/SDs; they are synthesized narratively and in RoB 2/GRADE. Abbreviations: PTSD = posttraumatic stress disorder; CAPS = Clinician-Administered PTSD Scale; PCL-5(-K) = PTSD Checklist for DSM-5 (Korean version); NF = neurofeedback.

Figure 2. Forest Plot of Posttreatment Effects (Hedges' G) by Study and Pooled Estimate (Random-Effects, REML With Knapp–Hartung Adjustment).



Certainty of Evidence (GRADE)

Outcome-level GRADE ratings are summarized in Table 6 (SoF). For posttreatment severity, overall certainty was low to moderate, with downgrades for risk of bias (unblinded passive comparators), imprecision (small samples; one CI approaching the null), and applied heterogeneity related to comparator/modality diversity, despite minimal statistical heterogeneity in the meta-analysis. For remission and safety/acceptability, certainty ranged from moderate to low, primarily due to imprecision and variability in adverse event and withdrawal reporting.

Discussion

The primary objective of this work was to evaluate, under PRISMA standards, the clinical efficacy, safety/acceptability, and mechanistic underpinnings of NF for adult PTSD, distinguishing modalities (EEG band/LORETA/rt-fMRI) and comparator types (wait-list/TAU, active controls, sham), and quantitatively integrating the posttreatment severity outcome where data were comparable. Taken together, the findings support a dual reading: on the one hand, there are signals of clinical benefit versus passive comparators; on the other, there is consistent mechanistic evidence—normalization of DMN/SN connectivity and amygdala down-regulation—from double-blind sham trials in which between-group clinical superiority is not always

confirmed (Bell et al., 2019; Leem et al., 2021; Nicholson et al., 2020; van der Kolk et al., 2016; Zhao et al., 2023). This combination suggests that NF has specific potential while coexisting with nonspecific components that become more visible when the comparator is passive.

In trials comparing EEG-NF with wait-list/TAU, I observed posttreatment symptom reductions that, by magnitude and consistency, are clinically meaningful (Leem et al., 2021; van der Kolk et al., 2016). This pattern aligns with the meta-analysis of the three trials with comparable data, which yielded a pooled effect size of $g = -1.04$ (95% CI = -1.49 to -0.59) with minimal heterogeneity, indicating that—when the contrast is not sham—NF is associated with a clinically relevant improvement. It is reasonable to link this signal to the self-regulatory learning promoted by training—enhanced attentional control, reduced hyperarousal, and greater emotional tolerance—and to consider that “dose” (number and duration of sessions) contributes to consolidation; indeed, protocols ranged from 3 to 24 sessions, used standardized posttreatment measures, and in some cases included brief follow-ups showing short-term maintenance (e.g., 1 month in van der Kolk et al., 2016). Overall, the data suggest that NF can provide a tangible clinical benefit in the immediate posttreatment period, particularly against minimal-intervention conditions.

Table 6*Summary of Findings (SoF) and Certainty of Evidence (GRADE)*

Outcome (measure, time)	Comparison	Studies (participants)	Effect estimate (95% CI)*	Certainty (GRADE)	Reasons for rating†
PTSD symptom severity (posttreatment; SMD)	Neurofeedback (EEG/LORETA) vs. WL/TAU/active	3 RCTs (N = 88)	SMD -1.04 (-1.49 to -0.59)	Low–Moderate	↓ risk of bias (a); ↓ imprecision (b); ↓ indirectness / heterogeneity of comparators/modalities (c)
Remission (diagnostic status; posttreatment)	Neurofeedback vs. comparator	2 RCTs (n not pooled)	Directionally favors NF (not pooled)	Low	↓ imprecision (d); ↓ inconsistency (e)
Safety and acceptability (serious AEs; withdrawals)	Neurofeedback vs. comparator	4–5 RCTs (n not pooled)	No NF-related serious AEs reported; withdrawals low–moderate and similar across arms	Moderate–Low	↓ imprecision / under-reporting (f); follow-up short (g)

Note. NF = neurofeedback; RCT = randomized controlled trial; PTSD = posttraumatic stress disorder; SMD = standardized mean difference; WL/TAU = wait-list/treatment as usual; AE = adverse event.* = Standardized mean differences (Hedges' g) from posttreatment group means/SDs; negative values indicate lower PTSD severity in the neurofeedback group (see Table 5 and Figure 2); † = Reasons: (a) Some concerns/high risk in deviation/measurement domains for WL/TAU trials (RoB 2; Table 4), (b) small sample sizes; one study's CI borders the null, (c) mixture of comparators (WL/TAU, active) and modalities (EEG bands, LORETA) limits directness despite low statistical heterogeneity, (d) sparse data and variable definitions of remission, (e) effects not consistently superior to comparators across trials, (f) adverse-event reporting heterogeneous; few events overall, and (g) short follow-up in most trials.

When NF was contrasted with more stringent controls—active (e.g., HRV-biofeedback) or sham—between-group effects were more modest or inconclusive, yet plausible mechanistic markers emerged. In LORETA z-score versus HRV-biofeedback, both arms improved, although the posttreatment standardized between-group effect favored NF (Bell et al., 2019). In double-blind sham designs, Nicholson et al. (2020) reported within-group symptom reduction accompanied by DMN/SN connectivity normalization, whereas Zhao et al. (2023) showed greater amygdala control with rt-fMRI and symptom decreases in both arms, without a significant between-group difference at post. This dual reading—clear clinical benefit versus wait-list/TAU and mechanistic signals in sham-controlled trials—is compatible with a potential specific effect of NF that coexists with nonspecific components (e.g., expectancy, attention, therapeutic alliance) that are more apparent when the control is passive. Methodologically, it also underscores that comparator strength conditions the observable effect

size: the tighter the control of expectancy and training context, the narrower the short-term between-group differences, which does not negate within-group change nor the biological anchoring of the procedure (Bell et al., 2019; Leem et al., 2021; Nicholson et al., 2020; van der Kolk et al., 2016; Zhao et al., 2023).

Regarding safety and acceptability, the included trials did not report NF-related serious adverse events, and dropout rates were low to moderate and comparable across arms (Bell et al., 2019; Leem et al., 2021; Nicholson et al., 2020; van der Kolk et al., 2016; Zhao et al., 2023). This profile supports the adjunctive use of NF, especially when self-regulation and hyperarousal reduction are key therapeutic aims.

From a clinical standpoint, these results help delineate when, how, and for whom NF may add value. Given its tolerability and acceptability, NF can be integrated as an adjunct in adults with PTSD—

particularly in individuals with partial response to trauma-focused CBT or EMDR, with marked physiological hyperreactivity, with difficulty sustaining exposure due to autonomic dysregulation, or with a need to train attentional and somatic control before or during trauma-focused psychotherapy. Modality selection can be guided by clinical goals and feasibility: EEG-NF by bands and LORETA z-score are more accessible options and, against passive comparators, tend to exhibit a stronger clinical signal; rt-fMRI offers a direct mechanistic anchor (amygdala/networks) and may be best reserved for specialized centers or therapeutic-mechanistic purposes (e.g., documenting neural mediators of change). Implementation benefits from a multimodal plan: embedding NF as a module in a stepped approach (e.g., 12–24 sessions of 30–60 min for autonomic stabilization and regulation training, followed by exposure or EMDR) is reasonable, provided systematic outcome monitoring (e.g., CAPS-5 or PCL-5), explicit targets, and dose adjustments according to response. Honest psychoeducation—NF does not replace first-line therapies but can potentiate them—and shared decision-making are essential, particularly because short-term superiority versus sham remains mixed. In brief, NF can be considered a regulation tool that helps facilitate and sustain trauma-focused work within evidence-based frameworks, with low risk and realistic expectations.

This review has several strengths: a transparent search and documentation process (PRISMA/PRISMA-S), a priori criteria and outcome hierarchies, RoB 2 assessment by outcome, random-effects meta-analysis (τ^2 via REML with Knapp–Hartung adjustment), and a GRADE synthesis linking estimates, precision, and methodological quality. Nonetheless, the certainty of the posttreatment effect is low to moderate, constrained by small samples, applied heterogeneity (modality/comparator/dosing), and risk of bias in trials with passive controls (lack of blinding and predominant reliance on self-report), as well as short follow-ups and the inability to assess asymmetry with $k < 10$. These limitations circumscribe generalizability and highlight the need for larger, double-blind RCTs with credible active/sham comparators, 6- to 12-month follow-ups, standardized protocols (target, parameters, “dose”), and patient-centered outcomes (e.g., remission and functioning). In parallel, it is a priority to examine moderators (number of sessions, modality/target, population—veterans vs. civilians) and neurophysiological mediators (EEG/fMRI) to specify how and for whom NF adds value.

In conclusion, in adults with PTSD, NF shows posttreatment symptom benefits versus passive comparators, a favorable safety profile, and mechanistic coherence with circuit models. However, the certainty of the specific effect—particularly versus active/sham—is low to moderate and requires more rigorous, longitudinal trials to pin down its magnitude and durability. In the short term, positioning NF as an adjunct within a multimodal plan, with explicit self-regulation goals and periodic outcome monitoring, is a reasonable strategy aligned with the current evidence (Bell et al., 2019; Leem et al., 2021; Nicholson et al., 2020; van der Kolk et al., 2016; Zhao et al., 2023).

Author Disclosures

This is self-funded research. The author declares that there were no conflicts of interest in the preparation of this manuscript. I used ChatGPT to translate limited sections of the manuscript. All AI-assisted outputs were checked for accuracy, edited by the author, and approved in the final version. The author takes full responsibility for the content. The author is responsible for all statements made in this article.

References

- American Psychological Association. (2017). *Clinical practice guideline for the treatment of posttraumatic stress disorder (PTSD) in adults*. <https://www.apa.org/ptsd-guideline>
- Begg, C. B., & Mazumdar, M. (1994). Operating characteristics of a rank correlation test for publication bias. *Biometrics*, 50(4), 1088–1101. <https://doi.org/10.2307/2533446>
- Bell, A. N., Moss, D., & Kallmeyer, R. J. (2019). Healing the neurophysiological roots of trauma: A controlled study examining LORETA Z-score neurofeedback and HRV biofeedback for chronic PTSD. *NeuroRegulation*, 6(2), 54–70. <https://doi.org/10.15540/nr.6.2.54>
- Cuthbert, B. N. (2014). The RDoC framework: Facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. *World Psychiatry*, 13(1), 28–35. <https://doi.org/10.1002/wps.20087>
- DerSimonian, R., & Laird, N. (1986). Meta-analysis in clinical trials. *Controlled Clinical Trials*, 7(3), 177–188. [https://doi.org/10.1016/0197-2456\(86\)90046-2](https://doi.org/10.1016/0197-2456(86)90046-2)
- Duval, S., & Tweedie, R. (2000). Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics*, 56(2), 455–463. <https://doi.org/10.1111/j.0006-341X.2000.00455.x>
- Egger, M., Davey Smith, G., Schneider, M., & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *BMJ*, 315(7109), 629–634. <https://doi.org/10.1136/bmj.315.7109.629>
- Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2017). EEG-neurofeedback as a tool to modulate cognition and behavior: A review tutorial. *Frontiers in Human Neuroscience*, 11, Article 51. <https://doi.org/10.3389/fnhum.2017.00051>
- Fede, S. J., Dean, S., Manuweera, T., & Momenan, R. (2020). A guide to literature-informed decisions in the design of real-time fMRI neurofeedback studies: A systematic review. *Frontiers in Human Neuroscience*, 14, Article 60. <https://doi.org/10.3389/fnhum.2020.00060>

- Guyatt, G. H., Oxman, A. D., Vist, G. E., Kunz, R., Falck-Ytter, Y., Alonso-Coello, P., & Schünemann, H. J. (2011). GRADE guidelines: 1. Introduction—GRADE evidence profiles and summary of findings tables. *Journal of Clinical Epidemiology*, 64(4), 383–394. <https://doi.org/10.1016/j.jclinepi.2010.04.026>
- Hamblen, J. L., Norman, S. B., Sonis, J., Phelps, A., Bisson, J. I., Nunes, V. D., Megnin-Viggars, O., Forbes, D., Riggs, D. S., & Schnurr, P. P. (2019). A guide to guidelines for the treatment of posttraumatic stress disorder in adults: An update. *Psychotherapy*, 56(3), 359–373. <https://doi.org/10.1037/pst0000231>
- Hedges, L. V., & Olkin, I. (1985). *Statistical methods for meta-analysis*. Academic Press.
- Higgins, J. P. T., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M. J., & Welch, V. A. (Eds.). (2023). *Cochrane handbook for systematic reviews of interventions* (Version 6.3). Cochrane/Wiley. <https://doi.org/10.1002/9781119536604>
- IntHout, J., Ioannidis, J. P. A., Rovers, M. M., & Goeman, J. J. (2016). Plea for routinely presenting prediction intervals in meta-analysis. *BMJ Open*, 6(7), Article e010247. <https://doi.org/10.1136/bmjopen-2015-010247>
- Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Benjet, C., Bromet, E. J., Cardoso, G., Degenhardt, L., de Girolamo, G., Dinolova, R. V., Ferry, F., Florescu, S., Gureje, O., Haro, J. M., Huang, Y., Karam, E. G., Kawakami, N., Lee, S., Lepine, J.-P., Levinson, D., ... Koenen, K. C. (2017). Trauma and PTSD in the WHO World Mental Health Surveys. *European Journal of Psychotraumatology*, 8(sup5), Article 1353383. <https://doi.org/10.1080/20008198.2017.1353383>
- Knapp, G., & Hartung, J. (2003). Improved tests for a random-effects meta-regression with a single covariate. *Statistics in Medicine*, 22(17), 2693–2710. <https://doi.org/10.1002/sim.1482>
- Koenen, K. C., Ratanatharathorn, A., Ng, L., McLaughlin, K. A., Bromet, E. J., Stein, D. J., Karam, E. G., Meron Ruscio, A., Benjet, C., Scott, K., Atwoli, L., Petukhova, M., Lim, C. C. W., Al-Hamzawi, A., Borges, G., de Girolamo, G., Degenhardt, L., Demyttenaere, K., Florescu, S., ... Kessler, R. C. (2017). Posttraumatic stress disorder in the World Mental Health Surveys. *Psychological Medicine*, 47(13), 2260–2274. <https://doi.org/10.1017/S0033291717000708>
- Lanius, R. A., Frewen, P., Tursich, M., Jetly, R., & McKinnon, M. C. (2015). Restoring large-scale brain networks in PTSD and related disorders: A proposal for neuroscientifically-informed treatment interventions. *European Journal of Psychotraumatology*, 6(1), Article 27313. <https://doi.org/10.3402/ejpt.v6.27313>
- Leem, J., Cheong, M. J., Lee, H., Cho, E., Lee, S. Y., Kim, G. W., & Kang, H. W. (2021). Effectiveness, cost-utility, and safety of neurofeedback self-regulating training in patients with post-traumatic stress disorder: A randomized controlled trial. *Healthcare*, 9(10), Article 1351. <https://doi.org/10.3390/healthcare9101351>
- Luctkar-Flude, M., & Groll, D. (2015). A systematic review of the safety and effect of neurofeedback on fatigue and cognition. *Integrative Cancer Therapies*, 14(4), 318–340. <https://doi.org/10.1177/1534735415572886>
- Morris, S. E., Rumsey, J. M., & Cuthbert, B. N. (2014). Rethinking mental disorders: The role of learning and brain plasticity. *Restorative Neurology and Neuroscience*, 32(1), 5–23. <https://doi.org/10.3233/RNN-139015>
- Nicholson, A. A., Ros, T., Densmore, M., Frewen, P., Neufeld, R. W. J., Théberge, J., Jetly, R., & Lanius, R. A. (2020). A randomized, controlled trial of alpha-rhythm EEG neurofeedback in posttraumatic stress disorder: Preliminary evidence of decreased symptoms and restored default-mode and salience-network connectivity using fMRI. *NeuroImage: Clinical*, 28, Article 102490. <https://doi.org/10.1016/j.nicl.2020.102490>
- Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., ... Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, 372, n71. <https://doi.org/10.1136/bmj.n71>
- Rethlefsen, M. L., Kirtley, S., Waffenschmidt, S., Ayala, A. P., Moher, D., Page, M. J., & Koffel, J. B. (2021). PRISMA-S: An extension to the PRISMA statement for reporting literature searches in systematic reviews. *Systematic Reviews*, 10, Article 39. <https://doi.org/10.1186/s13643-020-01542-z>
- Ros, T., Enriquez-Geppert, S., Zotev, V., Young, K. D., Wood, G., Whitfield-Gabrieli, S., Wan, F., Vuilleumier, P., Vialatte, F.-B., Tsytsarev, V., Sur, M., Serman, M. B., Steiner, N. J., Sterzer, P., Strehl, U., Sitaram, R., Scharnowski, F., Rubia, K., Rosa, A., ... Thibault, R. T. (2020). Consensus on the reporting and experimental design of clinical and cognitive-behavioral neurofeedback studies (CRED-nf checklist). *Brain*, 143(6), 1674–1685. <https://doi.org/10.1093/brain/awaa009>
- Schünemann, H. J., Brožek, J., Guyatt, G., & Oxman, A. D. (Eds.). (2022). *GRADE handbook for grading quality of evidence and strength of recommendations*. GRADE Working Group. <https://gdt.gradepro.org/app/handbook/handbook.html>
- Sitaram, R., Ros, T., Stoeckel, L. E., Haller, S., Scharnowski, F., Lewis-Peacock, J., Weiskopf, N., Blefari, M. L., Rana, M., Oblak, E., Birbaumer, N., & Sulzer, J. (2017). Closed-loop brain training: The science of neurofeedback. *Nature Reviews Neuroscience*, 18(2), 86–100. <https://doi.org/10.1038/nrn.2016.164>
- Sterne, J. A. C., Hernán, M. A., Reeves, B. C., Savović, J., Berkman, N. D., Viswanathan, M., Henry, D., Altman, D. G., Ansari, M. T., Boutron, I., Carpenter, J. R., Chan, A.-W., Churchill, R., Deeks, J. J., Hróbjartsson, A., Kirkham, J., Jüni, P., Loke, Y. K., Pigott, T. D., ... Higgins, J. P. T. (2016). ROBINS-I: A tool for assessing risk of bias in non-randomised studies of interventions. *BMJ*, 355, i4919. <https://doi.org/10.1136/bmj.i4919>
- Sterne, J. A. C., Savović, J., Page, M. J., Elbers, R. G., Blencowe, N. S., Boutron, I., Cates, C. J., Cheng, H.-Y., Corbett, M. S., Eldridge, S. M., Hernán, M. A., Hopewell, S., Hróbjartsson, A., Junqueira, D. R., Jüni, P., Kirkham, J. J., Lasserson, T., Li, T., McAleenan, A., ... Higgins, J. P. T. (2019). RoB 2: A revised tool for assessing risk of bias in randomized trials. *BMJ*, 366, i4898. <https://doi.org/10.1136/bmj.i4898>
- Thibault, R. T., & Raz, A. (2017). The psychology of neurofeedback: Clinical intervention even if applied placebo. *American Psychologist*, 72(7), 679–688. <https://doi.org/10.1037/amp000118>
- Thompson, S. G., & Higgins, J. P. T. (2002). How should meta-regression analyses be undertaken and interpreted? *Statistics in Medicine*, 21(11), 1559–1573. <https://doi.org/10.1002/sim.1187>
- U.S. Department of Veterans Affairs [VA]. (2023). *VA/DoD clinical practice guideline for the management of posttraumatic stress disorder and acute stress disorder (Version 4.0)*. VA/DoD Clinical Practice Guideline Working Group. <https://www.healthquality.va.gov/guidelines/MH/ptsd/>
- van der Kolk, B. A., Hodgdon, H., Gapen, M., Musicaro, R., Suvak, M. K., Hamlin, E., & Spinazzola, J. (2016). A randomized controlled study of neurofeedback for chronic PTSD. *PLoS ONE*, 11(12), Article e0166752. <https://doi.org/10.1371/journal.pone.0166752>
- Varker, T., Jones, K. A., Arjmand, H.-A., Hinton, M., Hiles, S. A., Freijah, I., Forbes, D., Kartal, D., Phelps, A., Bryant, R. A., McFarlane, A., Hopwood, M., & O'Donnell, M. (2021). Dropout from guideline-recommended psychological treatments for posttraumatic stress disorder: A systematic review and meta-analysis. *Journal of Affective Disorders*

- Reports*, 4, Article 100093. <https://doi.org/10.1016/j.jadr.2021.100093>
- Viechtbauer, W. (2005). Bias and efficiency of meta-analytic variance estimators in the random-effects model. *Journal of Educational and Behavioral Statistics*, 30(3), 261–293. <https://doi.org/10.3102/10769986030003261>
- Wan, X., Wang, W., Liu, J., & Tong, T. (2014). Estimating the sample mean and standard deviation from the sample size, median, range and/or interquartile range. *BMC Medical Research Methodology*, 14, Article 135. <https://doi.org/10.1186/1471-2288-14-135>
- Zhao, Z., Duek, O., Seidemann, R., Gordon, C., Walsh, C., Romaker, E., Koller, W. N., Horvath, M., Awasthi, J., Wang, Y., O'Brien, E., Fichtenholtz, H., Hampson, M., & Harpaz-Rotem, I. (2023). Amygdala downregulation training using fMRI neurofeedback in post-traumatic stress disorder: A randomized, double-blind trial. *Translational Psychiatry*, 13(1), Article 177. <https://doi.org/10.1038/s41398-023-02467-6>

Received: October 27, 2025

Accepted: January 18, 2026

Published: September 28, 2026

In Vivo Neuroimaging Evidence of Prefrontal Cortex Dysfunction in Adolescents With Substance Use: A Systematic Review

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Abstract

Background. Adolescence marks a critical phase for neurodevelopment, particularly in the prefrontal cortex (PFC), which underpins executive function and impulse control. This period coincides with the vulnerability to substance use, which has a significant impact on brain maturation. In vivo neuroimaging techniques provide valuable insights into the structural and functional alterations in the PFC associated with adolescent substance use. This review aims to synthesize the available literature focusing on the PFC dysfunction in adolescent substance users. **Methods.** A comprehensive search was conducted across peer-reviewed, Scopus-indexed databases, including PubMed, Web of Science, and Google Scholar. Eligible studies included adolescents aged 10–19 years with reported substance use like alcohol, nicotine, cannabis, opioids, stimulants, or polysubstance use, assessed via structural or functional neuroimaging techniques (MRI, fMRI, DTI, PET). Data were extracted on study design, sample characteristics, imaging modalities, and findings related to PFC dysfunction. **Results.** Sixteen studies met the inclusion criteria. Structural MRI studies reported reduced cortical thickness and gray matter volume in the dorsolateral and orbitofrontal regions among the cannabis and alcohol users. Functional imaging studies consistently showed the hypoactivation of PFC circuits during executive function tasks, along with the altered resting-state connectivity within the default mode and reward networks. **Conclusion.** This evidence suggests that adolescent substance use is linked with structural, functional, and neurochemical alterations in the PFC, which may further affect executive functioning and impulse control. Thus, it highlights the need for longitudinal studies to understand the neurodevelopmental vulnerabilities and highlight the importance of early prevention and intervention strategies targeting adolescent substance users.

Keywords: adolescents; substance use; prefrontal cortex; neuroimaging methods; PRISMA

Citation: Dinesh, D., & Unnikrishnan, S. (2026). In vivo neuroimaging evidence of prefrontal cortex dysfunction in adolescents with substance use: A systematic review. *NeuroRegulation*, 13(3), 270–279. <https://doi.org/10.15540/nr.13.3.270>

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Introduction

Adolescence is a critical developmental period characterized by rapid brain development and maturation, particularly in regions involved in cognitive and emotional processing (Clark et al., 2008). During this time, significant neuronal growth occurs, resulting in both structural and functional changes in the brain. This period also marks behaviors such as social interaction, exploration, novelty seeking, and mostly high-risk-taking behaviors (Boer et al., 2024). Thus, often these

periods of heightened neuroplasticity also represent a vulnerability to substance use, including early exposure to alcohol, nicotine, cannabis or marijuana, and other psychoactive substances. Such exposure has been shown to disrupt the typical neurodevelopmental trajectories, particularly affecting the prefrontal cortex (PFC) region, which is primarily responsible for executive functions such as planning, decision-making, impulse control, and reward regulation. These alterations in the PFC functioning during adolescence not only hinder cognitive control but also hint at the risk of

developing substance use disorders in adulthood (Ferdous, 2022). In 2019, the prevalence of alcohol consumption among 15- to 19-year-olds remained high worldwide (22%) with very little gender differences and a tendency to increase from initially low levels in some regions (World Health Organization, 2024).

The field of addiction research has long focused on impairments in self-regulation and maladaptive processing, both of which are closely linked to PFC dysfunction. Studies have shown that drug use increases activity in the PFC regions when related to drug cues, and decreases non-drug-related activity, especially during the episodes of bingeing and periods of craving in addiction (Goldstein & Volkow, 2011). Advancements in *in vivo* neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and diffusion tensor imaging (DTI), have thus provided compelling evidence for these dysfunctions marked by the consumption of such psychoactive substances. These methods consistently report hypoactivation of the PFC during cognitive control tasks, followed by disruptions in limbic-striatal connectivity and various other structural abnormalities (Li et al., 2009). These neurobiological alterations are thought to contribute to the continued and compulsive use of substances. Other cognitive domains, including memory (10% lower recall in heavy drinkers), attention, and processing speed, also show significant decline with the involvement of substance use like alcohol. Some studies also show that even after 4 weeks of abstinence, regular adolescent marijuana users showed deficits in learning, working memory, cognitive flexibility, and error monitoring (Squeglia et al., 2009).

Given the context of growing technological access and social influence, adolescent substance use continues to rise, which further links early use to later-life addiction (Silveri et al., 2016). It is therefore essential to synthesize the neuroimaging findings that illuminate the neural mechanisms underlying this vulnerability. Despite a growing body of neuroimaging research investigating the neural correlates of adolescent substance use, findings remain scattered and inconsistent. Methodological issues in these studies are important to consider, such as variability in imaging techniques and defining of abstinent periods (Squeglia et al., 2009). Some studies report structural deficits (Goldstein & Volkow, 2011), others highlight functional hypoactivation or disrupted connectivity (Goldstein & Volkow, 2011) within the PFC, while others report no significant change. This inconsistency limits our

understanding of the precise neural alterations associated with adolescent substance use and impedes the development of targeted interventions. Hence, this review aims to (a) consolidate the present evidence of PFC dysfunction in adolescents with substance use, (b) examine how these alterations contribute to addiction risk, and (c) identify the methodological limitations for prevention and treatment research.

Methods

This systematic review was performed under the 2020 PRISMA guidelines, where the data were retrieved and compiled to understand, identify, describe, and characterize various structural and functional changes in the PFC, one of which is the main focus of the study.

Information Sources

An initial pool of information was collected using a serial search of articles, performed using various electronic databases like Google Scholar, Semantic Scholar, PubMed, BASE (Bielefeld Academic Search Engine), Web of Sciences, and ResearchGate. This was done to identify the relevant studies for the review. Additionally, reference search from other studies was manually conducted. Data limit of 2010 to 2025, a 15-year boundary was considered to ensure good quality, with extensive findings culminating access to the width and breadth of the data. The selection of these took place between August 11–20, 2025.

Search Strategy

The keywords search used the free-text keywords using Boolean operators in various database searches which included (“Adolescents”) AND (“substance use” OR “drug use” OR “cannabis” OR “nicotine” OR “alcohol” OR “marijuana”) AND (“prefrontal cortex dysfunction” OR “PFC” OR “executive function dysfunction” OR “impulse control difficulties”) AND (“In-Vivo neuroimaging” OR “MRI” OR “fMRI” OR “DTI” OR “PET” OR “MRS”).

Study Selection and Categorization Process

The inclusion criteria included all the studies involving adolescents whose ages ranged between 10–19 years (as per World Health Organization); participants with current or past substance use; participants using one or more of the following substances: alcohol, cannabis, nicotine, opioids, stimulants, inhalants, substance use as defined by diagnostic criteria (DSM-5, ICD-10) or validated screening tools, or any self-reported usage evidence of PFC dysfunction based on *in vivo* neuroimaging

findings; neuroimaging modalities may include fMRI, structural MRI, DTI, PET, etc., both structural and functional outcomes of PFC dysfunction. All cross-sectional, longitudinal, Masters dissertation, doctoral theses, and book chapters were extensively considered.

All identified records were imported into Mendeley/Zotero reference manager, and duplicates were removed. All animal studies were also removed. They were further screened for title and abstract screening. Of those which were retrieved, full text screening was conducted using the inclusion criteria. The study selection process, including the identification, screening, eligibility assessment, and inclusion of studies, is presented in Figure 1.

Data Extraction

A standardized data extraction sheet was done using Microsoft Excel to collect details on author(s) and year of study; theoretical or conceptual framework; research questions, hypotheses, and objectives; methodology (sample, research design, neuroimaging modality); analysis and major findings with respect to structural or functional PFC outcomes, conclusion, limitations, and suggestions for future research methods.

Data Synthesis

A narrative synthesis was used because of the heterogeneity in neuroimaging techniques, outcome measures, and types of substance use. The studies were categorized on the basis of structural (e.g., cortical thickness, gray matter volume) abnormalities, task-based and resting-state fMRI functional activation patterns, changes in connectivity (DTI, functional connectivity), and neurochemical or metabolic (MRS, PET). Patterns across studies were compared where there was enough homogeneity and converging evidence pointed out.

Results

Theme 1: Structural Brain Alterations in Adolescent Substance Use

Studies have shown several neurostructural differences in adolescents who often engage in early substance use. Lopez-Larson et al. (2011) found that the cortical thickness in the prefrontal and insular regions had significantly reduced among the adolescent marijuana users, which shows that these regions are crucial for decision-making and interoceptive awareness (see Figure 2). A study by Cheetham et al. (2012) reported that reduced orbitofrontal volumes predicted future initiation of

cannabis use during a 4-year follow-up, indicating that adolescents may be vulnerable to experimentation due to preexisting vulnerabilities in neural structure. Further studies by Cheetham et al. (2014) found volumetric dissimilarities in the anterior cingulate cortex related to alcohol consumption, which again supports structural brain predictors of risk trajectories.

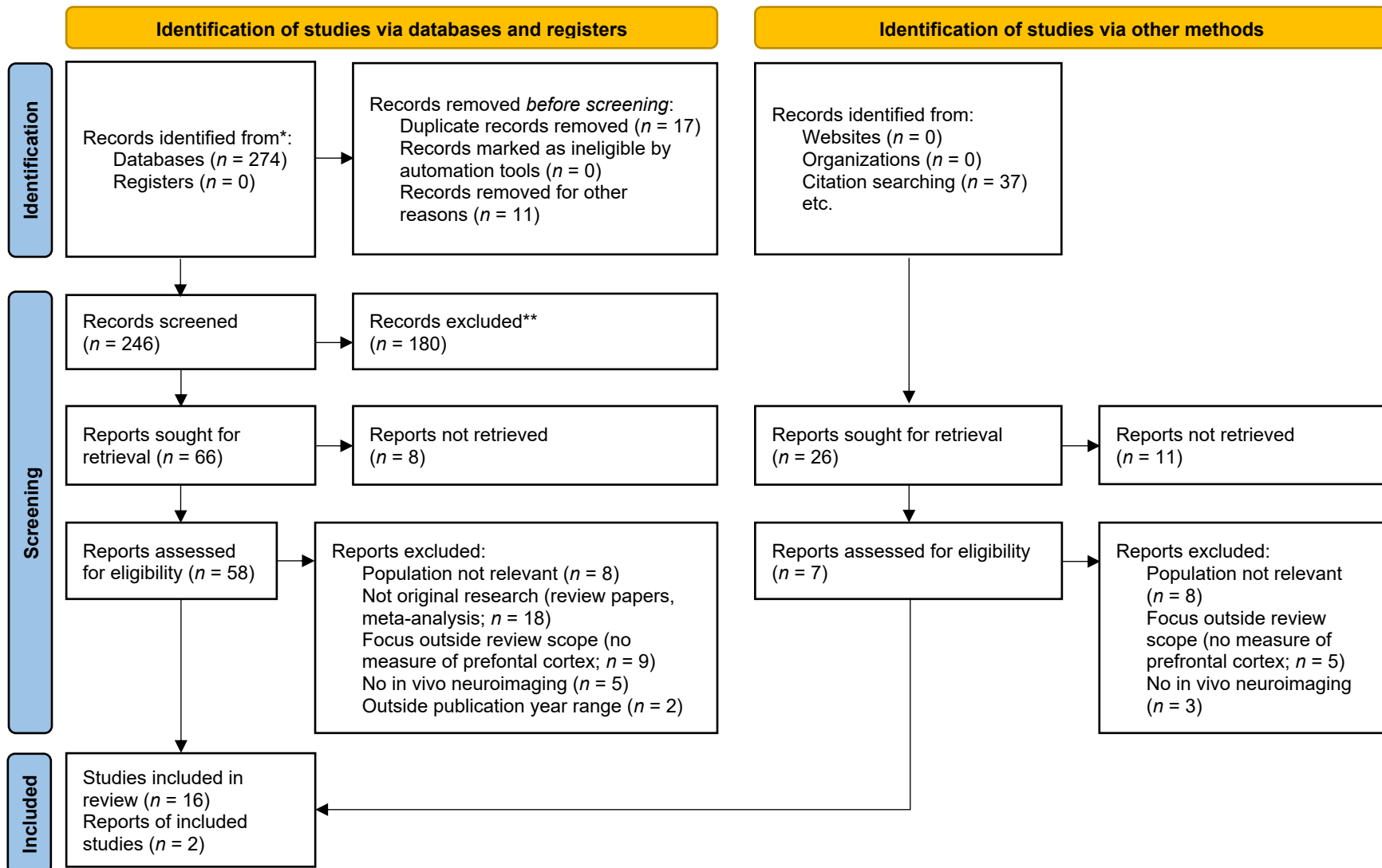
Another study by Sultan et al. (2021), which examined adolescents with bipolar disorder, shows that cannabis use was highly linked to changes in the neural structures, underscoring the combination of psychiatric vulnerability and changes in the brain structure due to substance use. Interestingly, Orr et al. (2019) demonstrated that already very low amounts of cannabis use were correlated with differences in gray matter volumes, which provokes concern that even low exposure during adolescence can be measured.

In more recent studies, Miller et al. (2024) showed that neuroanatomical variability is a significant predictor of substance use initiation. Yet, brain differences are not only a consequence of substance use but also may be a precursor of it. Overall, these results indicate a two-way interaction, which shows structural abnormalities can be both prone to substance use and aggravated by early exposure.

Theme 2: Reward Processing and Cue Reactivity

Another predominant theme involves changes in reward circuitry and a heightened sensitivity to substance-related cues. Brumback et al. (2015) demonstrated that heavy consumption of alcohol in adolescence amplified brain responses to alcohol cues, which later decreased with a period of abstinence, highlighting the plasticity of reward-associated neural activity. Swartz et al. (2020) showed that increased reward-related brain activity is predictive of future alcohol consumption, suggesting that increased reactivity can serve as a biomarker of future risk.

Weissman et al. (2015) found that an earlier onset of substance use was linked to strong connectivity between reward and cognitive control networks, possibly due to compensatory mechanisms that might later become destabilized. Similarly, Stanger et al. (2013) attributed neuroeconomic insights into risky substance abuse to individual differences in neural activities during delay discounting tasks, and these results were incorporated into the field of addiction research.

Figure 1. PRISMA 2020 Flow Diagram for New Systematic Reviews Which Included Searches of Databases, Registers and Other Sources.

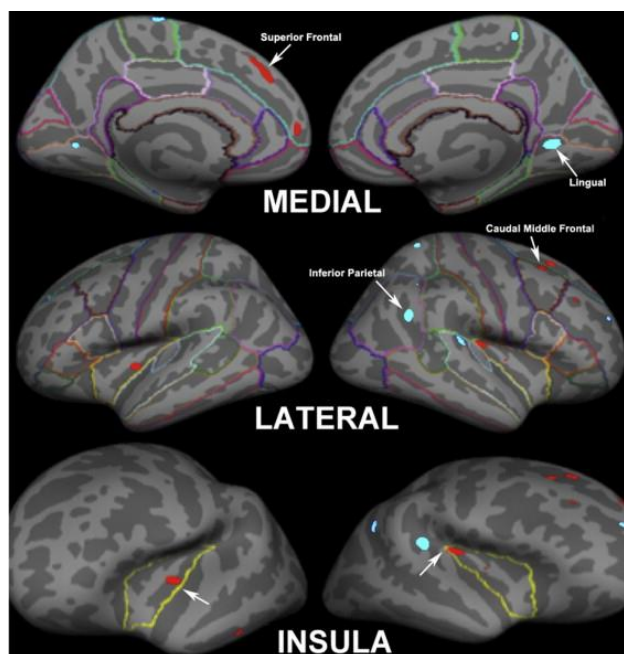
*Consider, if feasible to do so, reporting the number of records identified from each database or register searched (rather than the total number across all databases/registers).

**If automation tools were used, indicate how many records were excluded by a human and how many were excluded by automation tools.

Source: Page et al., (2021).

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Figure 2. Altered Prefrontal and Insular Cortical Thickness in Adolescent Marijuana Users.



Note. Reproduced from Lopez-Larson et al. (2011). Altered prefrontal and insular cortical thickness in adolescent marijuana users. Reproduced under the terms of the Creative Commons Attribution License (CC BY).

In a large multicenter study, Whelan et al. (2014) identified neuropsychosocial profiles that differentiated current and future alcohol misusers, predominantly in the adolescent population, which shows consistent results with other studies that find that aberrant reward processing underpins both initiation and escalation of use. Collectively, these studies underscore the importance of altered sensitivity to reward cues as a predictor of future direction as well as present use.

Theme 3: Cognitive Control and Neuropsychological Outcomes

Substance use during adolescence also overlaps with cognitive development, mainly executive functioning. Squeglia et al. (2012) identified that the onset of moderate to severe alcohol consumption predicted deterioration in neuropsychological functioning across the domains of memory and

attention, with varied effects on boys and girls. Additionally, Lisdahl et al. (2013) found that earlier initiation of alcohol and marijuana consumption disrupts cognition, brain structure, and functional connectivity, which showcases the long-term risks of development.

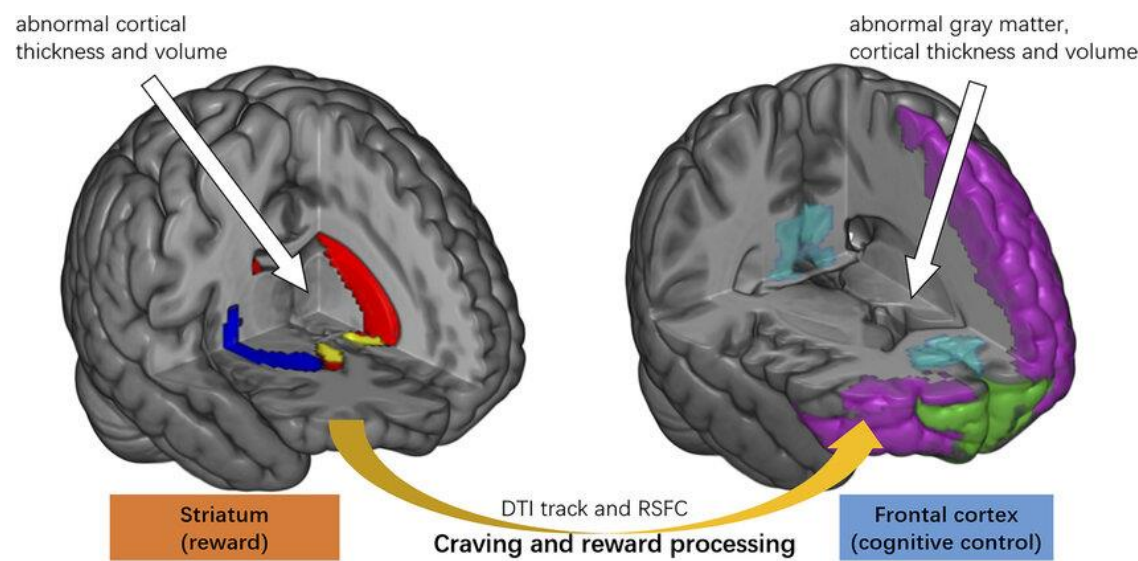
Tervo-Clemmens et al. (2018) offered narrower results, indicating that adolescent cannabis use impacts the brain systems involved in adult working memory encoding and retrieval and further indicated long-term changes in cognitive circuitry. Deneen et al. (2022) compared frontostriatal circuits between adolescent nicotine addiction and internet gaming disorder which showed similarities in the cognitive control disturbance across both behavioral and substance-related addictions (see Figure 3). Overall, these results emphasize the idea that substance use in adolescence is not confined to instant reward-driven mechanisms but has broader implications on higher-order executive functioning and long-term cognitive health.

New research emphasizes the importance of connectivity-based mechanisms in explaining adolescent substance use. Ewing et al. (2017) identified orbitofrontal cortex connectivity as a potential mechanism of behavior change, showing how functional links between prefrontal and limbic regions shape risk-taking and adaptability. Sultan et al. (2021) extended this by linking cannabis use in adolescents with bipolar disorder to neurostructural connectivity differences, underscoring the interplay between psychiatric conditions and substance-related neural pathways.

Theme 4: Connectivity and Mechanisms of Change

Likewise, Weissman et al. (2015) highlighted strengthened connectivity between the reward and control networks in early initiators and indicated neurodevelopmental changes that might mediate vulnerability. The studies collectively suggest that connectivity-based changes can mediate between structural weaknesses and functional differences in reward processing and provide a more dynamic conceptualization of adolescent addiction outcomes.

Figure 3. Comparison of Frontostriatal Circuits in Adolescent Nicotine Addiction and Internet Gaming Disorder.



Note. Reproduced from Deneen et al. (2022). Comparison of frontostriatal circuits in adolescent nicotine addiction and internet gaming disorder. Licensed under a Creative Commons Attribution 4.0 International License (CC BY 4.0).

Table 1

Characteristics and Major Findings of the Studies Examining Prefrontal Cortex Dysfunction in Adolescents With Substance Use

Author (Year)	Theoretical/Conceptual Framework	Research Question / Hypotheses / Objectives	Methodology (Sample, Research Design)	Analysis and Major Findings	Conclusion/ Limitations	Suggestions for Future Research	Reviewer's Observation
Lopez-Larson et al. (2011)	Neurodevelopmental disruption framework	To assess cortical thickness in adolescent marijuana users	MRI: adolescent, marijuana users vs controls	Reduced PFC and insula thickness	Structural deficits associated with adolescent cannabis use	Longitudinal studies needed	Very relevant, structural PFC evidence in youth
Brumback et al. (2015)	Cue-reactivity and abstinence	Does brain responses to alcohol cues change after abstinence?	fMRI; heavy-drinking adolescents; repeated at 1 month	Cue-related PFC activation decreased after abstinence	PFC may recover with abstinence	Longer abstinence follow-up	Recovery-focused inclusion
Cheetham et al. (2012)	Neurodevelopmental risk: orbitofrontal cortex role	Do OFC volumes predict initiation of cannabis use in adolescents?	Longitudinal MRI; 4-year follow-up; adolescents	Smaller OFC volumes predicted earlier cannabis initiation	PFC structural differences may precede substance use	Replicate in larger, diverse cohorts	Strong fit. Prospective, PFC-focused
Cheetham et al. (2014)	Anterior cingulate risk model	Do ACC volumes predict alcohol-related problems in adolescence?	MRI; prospective cohort study	Smaller ACC predicted alcohol-related problems	Structural deficits precede alcohol misuse	Larger, longer-term prospective studies	Good fit. ACC/PFC link

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Characteristics and Major Findings of the Studies Examining Prefrontal Cortex Dysfunction in Adolescents With Substance Use

Author (Year)	Theoretical/ Conceptual Framework	Research Question / Hypotheses / Objectives	Methodology (Sample, Research Design)	Analysis and Major Findings	Conclusion/ Limitations	Suggestions for Future Research	Reviewer's Observation
Deneen et al. (2022)	Frontostriatal circuit models	To compare circuits in adolescent nicotine addiction vs. internet gaming disorder	fMRI studies of adolescents with nicotine addiction and IGD	Shared and distinct frontostriatal abnormalities in both disorders	Limited sample sizes; cultural context: China	Broader cross-cultural comparisons	Interesting comparative angle but secondary to substance use focus
Ewing et al. (2017)	Neurocircuitry of behavior change	Is OFC connectivity linked to adolescent behavior change?	fMRI; adolescents during behavior tasks	Increased OFC connectivity related to reduced risk behaviors	Connectivity may underlie adaptive changes	Explore substance-specific pathways	Relevant conceptual support
Lisdahl et al. (2013)	Delay hypothesis – onset age matters	To explore effect of early onset on brain and cognition	Review of MRI and cognitive tests	Early use associated with reduced PFC volume and function	Timing of use matters more than total exposure	Emphasize developmental timing in future designs	Directly aligned with your study
Miller et al. (2024)	Neurodevelopmental variability in early adolescence	To examine whether neuroanatomical variability predicts initiation of substance use in late childhood/early adolescence	JAMA Network Open dataset; large community-based cohort	Structural MRI showed variability in brain morphology (esp. PFC, striatum) associated with risk for early substance initiation	Neuroanatomical variability may predispose youth to early substance use; causality not established	Longitudinal and genetically informed designs	Very relevant, directly links neurodevelopmental variability and risk pathways
Orr et al. (2019)	Neurodevelopmental vulnerability	Are there GM differences with very low cannabis use in adolescence?	MRI (IMAGEN dataset); n>1500 adolescents	Low-level cannabis use associated with reduced PFC gray matter	Even minimal use affects PFC development	Examine dose-response and reversibility	Directly relevant, structural PFC findings
Squeglia et al. (2012)	Developmental trajectories + substance use	To observe working memory brain activation over time in adolescents	Longitudinal fMRI (3-year follow-up); heavy drinkers vs. controls	Initiating heavy drinking is associated with less efficient PFC activation	Working memory impaired even before dependence sets in	Use pre-use baselines and track over time	Strong longitudinal PFC-focused study
Stanger et al. (2013)	Neuroeconomics and delay discounting	To investigate neural correlates of delay discounting in adolescent substance abusers	fMRI; adolescents with substance abuse vs. controls	Greater discounting and altered PFC–striatal activation in users	Neuroeconomic models explain impulsivity in adolescent substance use	Explore interventions targeting decision-making processes	Directly relevant links PFC to risky choices and use
Sultan et al. (2021)	Neurostructural abnormalities in comorbidity (cannabis + bipolar disorder)	To study cannabis use and brain structure in adolescents with bipolar disorder	MRI study. adolescents with bipolar disorder with/without cannabis use	Cannabis use associated with altered PFC volumes in bipolar adolescents	Small clinical sample, comorbidity complicates results	Larger samples needed, examine clinical outcomes	Novel clinical angle, comorbidity, shows stronger PFC vulnerability

Table 1

Characteristics and Major Findings of the Studies Examining Prefrontal Cortex Dysfunction in Adolescents With Substance Use

Author (Year)	Theoretical/ Conceptual Framework	Research Question / Hypotheses / Objectives	Methodology (Sample, Research Design)	Analysis and Major Findings	Conclusion/ Limitations	Suggestions for Future Research	Reviewer's Observation
Swartz et al. (2020)	Reward processing and adolescent alcohol use	To test whether reward-related brain activity predicts increases in alcohol use	fMRI reward task, longitudinal adolescent cohort	Stronger ventral striatum activation predicted higher future alcohol use	Reward hypersensitivity is a risk factor	Examine moderators like environment and stress	Good fit prospective evidence linking reward system activity to alcohol trajectories
Tervo-Clemmens et al. (2018)	Cannabis effects on working memory circuitry	To assess adolescent cannabis use effects on adult working memory processes	Longitudinal, fMRI of WM tasks	Adolescent cannabis use predicted poorer WM encoding and altered PFC-hippocampal activation in adulthood	Early cannabis use disrupts brain systems supporting WM	Larger longitudinal samples needed	Relevant, long-term cognitive consequences of early cannabis use
Weissman et al. (2015)	Connectivity between reward and control networks	To test whether earlier substance onset predicts altered connectivity	fMRI connectivity analyses in adolescents	Earlier onset predicted stronger reward–control network connectivity	Altered connectivity may reinforce risky behaviors	Longitudinal causal tests needed	Strong prospective evidence for timing of onset
Whelan et al. (2014)	Neuropsychosocial risk framework	Can neuropsychosocial profiles predict adolescent alcohol misuse?	IMAGEN longitudinal, multi-site	Identified PFC dysfunction as predictor of alcohol misuse	PFC dysfunction precedes problem drinking	Apply predictive models clinically	Landmark Nature study, highly relevant

Discussion

The overall findings from these literature imply that substance use during adolescence is closely connected to structural and functional impairments in the PFC, a key area responsible for executive functions, including decision-making, impulse control, and reward processing. Multiple studies have found that adolescent users of substances such as cannabis, alcohol, and nicotine have lower cortical thickness and volume in PFC subregions such as the orbitofrontal cortex and anterior cingulate cortex, regions used to regulate emotions and inhibit inappropriate behaviors (Cheetham et al, 2014). These neuroanatomical alterations not only indicate the effects of early substance use but also significantly predict subsequent substance use initiation and escalation, showing a bidirectional relationship between PFC dysfunction and substance use vulnerability.

Functionally, neuroimaging research has indicated that adolescent substance users exhibit overactive brain activity to substance cues and reduced activity to nondrug rewards, with dysregulated reward processing systems. The imbalance agrees with addiction models that focus on the inhibition of responses and abnormal salience attribution within the PFC circuits. Longitudinal studies confirm the hypothesis that premature dysfunction of the PFC is predictive of the neuropsychological deficits, including working memory and decision-making, which in turn lead to the compulsiveness of substance use during this developmental period.

In sum, these results support the role of dysfunction in the PFC of adolescent users of substances as an indicator of both vulnerabilities to substance-related addiction and the consequences of substance-induced neurodevelopmental discontinuity, which

highlights the importance of early detection and intervention strategies among adolescents, including a focus on PFC-function rehabilitation to prevent vulnerability to substance-related addictions.

Conclusion

Overall, all the reviewed studies provide clear evidence that adolescent substance use is closely associated with significant changes in the PFC, including the loss of cortical thickness and volume in regions such as the orbitofrontal and anterior cingulate cortices. These morphological alterations are not only a result of substance use onset but also outcomes of neurodevelopmental derailment through early exposure. Neuroimaging data show that connectivity within frontostriatal circuits is disrupted, reducing executive functions such as impulse control and choice-making and exaggerating reward reactions to drug-related stimuli. This lack of regulation in neural networks explains why adolescent substance users cannot regulate their behaviour and challenge substance cues.

In practice, the patterns of aberrant activation observed in these adolescents are indicative of an overbalance between increased sensitivity to drug rewards and decreased sensitivity to nondrug rewards, as is expected in the models of addiction as impaired response inhibition and abnormal salience attribution. Longitudinal evidence also indicates that brain dysfunction in the PFC at a young age is a predictor of future cognitive impairment and increasing substance use problems.

Clinically, these findings support developing cognitive training and behavioral therapies specifically aimed at strengthening prefrontal cortical control and reducing reward sensitivity to substance cues to curb addictive behaviors. Functional imaging can also guide pharmacological treatments by revealing neurotransmitter system dysregulations in PFC networks. Additionally, neuroimaging advances contribute to public health by informing educational programs on the neurodevelopmental risks of adolescent substance use, encouraging policies prioritizing early screening and intervention. Ultimately, integrating neuroimaging evidence into clinical and community practice promises improved outcomes by addressing the neural underpinnings of addiction vulnerability during this crucial developmental period.

Author Disclosures

The authors declare no conflicts of interest. No AI tools were used to generate the core content of this manuscript. However, minimal assistance was utilized for grammar correction and refinement of sentence structure.

References

- Boer, O. D., El Marroun, H., & Muetzel, R. L. (2024). Adolescent substance use initiation and long-term neurobiological outcomes: Insights, challenges and opportunities. *Molecular Psychiatry*, 29(7), 2211–2222. <https://doi.org/10.1038/s41380-024-02471-2>
- Brumback, T., Squeglia, L. M., Jacobus, J., Pulido, C., Tapert, S. F., & Brown, S. A. (2015). Adolescent heavy drinkers' amplified brain responses to alcohol cues decrease over one month of abstinence. *Addictive Behaviors*, 46, 45–52. <https://doi.org/10.1016/j.addbeh.2015.03.001>
- Cheetham, A., Allen, N. B., Whittle, S., Simmons, J. G., Yücel, M., & Lubman, D. I. (2012). Orbitofrontal volumes in early adolescence predict initiation of cannabis use: A 4-year longitudinal and prospective study. *Biological Psychiatry*, 71(8), 684–692. <https://doi.org/10.1016/j.biopsych.2011.10.029>
- Cheetham, A., Allen, N. B., Whittle, S., Simmons, J. G., Yücel, M., & Lubman, D. I. (2014). Volumetric differences in the anterior cingulate cortex prospectively predict alcohol-related problems in adolescence. *Psychopharmacology*, 231(8), 1731–1742. <https://doi.org/10.1007/s00213-014-3483-8>
- Clark, D. B., Thatcher, D. L., & Tapert, S. F. (2008). Alcohol, psychological dysregulation, and adolescent brain development. *Alcoholism: Clinical and Experimental Research*, 32(3), 375–385. <https://doi.org/10.1111/j.1530-0277.2007.00601.x>
- Deneen, K. M., Hussain, H., Waheed, J., Xinwen, W., Yu, D., & Yuan, K. (2022). Comparison of frontostriatal circuits in adolescent nicotine addiction and internet gaming disorder. *Journal of Behavioral Addictions*, 11(1), 26–39. <https://doi.org/10.1556/2006.2021.00086>
- Ewing, S. W. F., Chung, T., Caouette, J. D., Ketcherside, A., Hudson, K. A., & Filbey, F. M. (2017). Orbitofrontal cortex connectivity as a mechanism of adolescent behavior change. *NeuroImage*, 151, 14–23. <https://doi.org/10.1016/j.neuroimage.2016.12.076>
- Ferdous, N. (2022). Examining neurocognitive markers in adolescence as predictors of changes in later substance use. San Diego State University.
- Goldstein, R. Z., & Volkow N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews Neuroscience*, 12(11), 652–669. <https://doi.org/10.1038/nrn3119>
- Li, C. S., Luo, X., Yan, P., Bergquist, K., & Sinha, R. (2009). Altered impulse control in alcohol dependence: Neural measures of stop signal performance. *Alcoholism: Clinical and Experimental Research*, 33(4), 740–750. <https://doi.org/10.1111/j.1530-0277.2008.00891.x>
- Lisdahl, K. M., Gilbert, E. R., Wright, N. E., & Shollenbarger, S. (2013). Dare to delay? The impacts of adolescent alcohol and marijuana use onset on cognition, brain structure, and function. *Frontiers in Psychiatry*, 4, Article 53. <https://doi.org/10.3389/fpsy.2013.00053>
- Lopez-Larson, M. P., Bogorodzki, P., Rogowska, J., McGlade, E., King, J. B., Terry, J., & Yurgelun-Todd, D. (2011). Altered prefrontal and insular cortical thickness in adolescent marijuana users. *Behavioural Brain Research*, 220(1), 164–172. <https://doi.org/10.1016/j.bbr.2011.02.001>

- Miller, A. P., Baranger, D. A. A., Paul, S. E., Garavan, H., Mackey, S., Tapert, S. F., LeBlanc, K. H., Agrawal, A., & Bogdan, R. (2024). Neuroanatomical variability and substance use initiation in late childhood and early adolescence. *JAMA Network Open*, 7(12), Article e2452027. <https://doi.org/10.1001/jamanetworkopen.2024.52027>
- Orr, C., Spechler, P., Cao, Z., Albaugh, M., Chaarani, B., Mackey, S., D'Souza, D., Allgaier, N., Banaschewski, T., Bokde, A. L. W., Bromberg, U., Büchel, C., Burke Quinlan, E., Conrod, P., Desrivieres, S., Flor, H., Frouin, V., Gowland, P., Heinz, A., ... Garavan, H. (2019). Grey matter volume differences associated with extremely low levels of cannabis use in adolescence. *Journal of Neuroscience*, 39(10), 1817–1827. <https://doi.org/10.1523/jneurosci.3375-17.2018>
- Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., ... Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, 372, n71. <https://doi.org/10.1136/bmj.n71>
- Silveri, M. M., Dager, A. D., Cohen-Gilbert, J. E., & Sneider, J. T. (2016). Neurobiological signatures associated with alcohol and drug use in the human adolescent brain. *Neuroscience and Biobehavioral Reviews*, 70, 244–259. <https://doi.org/10.1016/j.neubiorev.2016.06.042>
- Squeglia, L. M., Jacobus, J., & Tapert S. F. (2009). The influence of substance use on adolescent brain development. *Clinical EEG and Neuroscience*, 40(1), 31–38. <https://doi.org/10.1177/155005940904000110>
- Squeglia, L. M., Spadoni, A. D., Infante, M. A., Myers, M. G., & Tapert, S. F. (2012). Initiating moderate to heavy alcohol use predicts changes in neuropsychological functioning for adolescent girls and boys. *Psychology of Addictive Behaviors*, 26(3), 427–437. <https://doi.org/10.1037/a0016516>
- Stanger, C., Elton, A., Ryan, S. R., James, G. A., Budney, A. J., & Kilts, C. D. (2013). Neuroeconomics and adolescent substance abuse: Individual differences in neural networks and delay discounting. *Journal of the American Academy of Child & Adolescent Psychiatry*, 52(7), 747–755. <https://doi.org/10.1016/j.jaac.2013.04.013>
- Sultan, A. A., Kennedy, K. G., Fiksenbaum, L., MacIntosh, B. J., & Goldstein, B. I. (2021). Neurostructural correlates of cannabis use in adolescent bipolar disorder. *International Journal of Neuropsychopharmacology*, 24(3), 181–190. <https://doi.org/10.1093/ijnp/pyaa077>
- Swartz, J. R., Weissman, D. G., Ferrer, E., Beard, S. J., Fassbender, C., Robins, R. W., Hastings, P. D., & Guyer, A. E. (2020). Reward-related brain activity prospectively predicts increases in alcohol use in adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 59(3), 391–400. <https://doi.org/10.1016/j.jaac.2019.05.022>
- Tervo-Clemmens, B., Simmonds, D., Calabro, F. J., Day, N. L., Richardson, G. A., & Luna, B. (2018). Adolescent cannabis use and brain systems supporting adult working memory encoding, maintenance, and retrieval. *NeuroImage*, 169, 496–509. <https://doi.org/10.1016/j.neuroimage.2017.12.041>
- Weissman, D. G., Schriber, R. A., Fassbender, C., Atherton, O., Krafft, C., Robins, R. W., & Guyer, A. E. (2015). Earlier adolescent substance use onset predicts stronger connectivity between reward and control networks. *Developmental Cognitive Neuroscience*, 16, 121–129. <https://doi.org/10.1016/j.dcn.2015.07.002>
- Whelan, R., Watts, R., Orr, C. A., Althoff, R. R., Artiges, E., Banaschewski, T., Barker, G. J., Bokde, A. L. W., Büchel, C., Carvalho, F. M., Conrod, P. J., Flor, H., Fauth-Bühler, M., Frouin, V., Gallinat, J., Gan, G., Gowland, P., Heinz, A., Ittermann, B., ... the IMAGEN Consortium (2014). Neuropsychosocial profiles of current and future adolescent alcohol misusers. *Nature*, 512(7513), 185–189. <https://doi.org/10.1038/nature13402>
- World Health Organization. (2024). Adolescent mental health. <https://www.who.int/news-room/fact-sheets/detail/adolescent-mental-health>

Received: October 23, 2025

Accepted: January 5, 2026

Published: September 28, 2026

Navigating Artificial Intelligence in Neuroregulation Practice: Ethical Principles, Risk Stratification, and a Clinical Policy Framework

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Abstract

Artificial intelligence (AI) tools are entering neuroregulation practice through multiple simultaneous pathways, including automated qEEG analysis, protocol recommendation systems, AI-assisted documentation, and consumer-facing mental health applications that clients bring directly into the therapeutic relationship. No ethics code specific to neuroregulation has yet addressed these applications directly. This article presents a conceptual and practical ethical framework grounded in established codes, including the BCIA Code of Ethics, ISNR Code of Ethics, APA Ethical Principles, ACA Code of Ethics, and the APA (2025) Ethical Guidance for Artificial Intelligence. A risk continuum model organizing AI applications along three dimensions (opacity, clinical consequence, and distance from oversight) is proposed. Four ethical domains are addressed: informed consent and transparency, practitioner competence and scope, data privacy and vendor accountability, and three AI accuracy risks (hallucination, automation bias, and collusion). A five-element practice policy framework is presented in tabular form for direct clinical use. Existing ethics principles are sufficient to guide responsible AI integration when applied deliberately. The practitioner remains accountable for AI-assisted decisions regardless of tool design or vendor claims. Proactive policy development positions neuroregulation clinicians to shape the field's emerging standards.

Author's Note on Temporal Limitations. The landscape of AI in health care is evolving at a pace that outstrips the typical publication cycle of academic literature. The specific tools, regulatory developments, and empirical findings described in this article reflect the state of the field as of the time of writing (mid-2026). The ethical frameworks, guiding principles, and policy elements presented here are designed to be durable: they are grounded in established ethics codes that are not tool-specific and will remain applicable as the technology continues to develop. Readers should expect that particular examples, cited tools, and regulatory references may have been superseded depending on when this article is read. The authors encourage readers to treat the frameworks as stable anchors and the specific illustrations as illustrative rather than exhaustive.

Keywords: artificial intelligence; neuroregulation; neurofeedback; ethics; automation bias; clinical policy; data privacy

Citation: Sherlin, L. H., & Longo, R. E. (2026). Navigating artificial intelligence in neuroregulation practice: Ethical principles, risk stratification, and a clinical policy framework. *NeuroRegulation*, 13(3), 280–292. <https://doi.org/10.15540/nr.13.3.280>

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Introduction

The Current Moment

The neuroregulation field is encountering artificial intelligence (AI) not as a distant development to anticipate but as a present reality arriving from multiple directions simultaneously. Commercial platforms are offering automated qEEG interpretation, returning clinically framed reports generated with limited or no visible reasoning. Protocol recommendation engines are suggesting training parameters on the basis of assessment data and pattern-matching algorithms trained on large datasets. AI-assisted documentation tools adopted across health care broadly are finding their way into the administrative workflows of neuroregulation practices, drafting session notes, generating treatment summaries, and composing communications submitted under the practitioner's name and credentials. And, perhaps most consequentially, clients are arriving at sessions having already consulted AI tools about their symptoms, their diagnosis, their protocols, and their progress, often without having disclosed this to their clinician.

This convergence is not a future state. It is the present one. Horvitz and West (2026) observed in a *Science* editorial that AI systems are being woven into everyday life faster than humans can understand them, while AI's capacity to process and model human behavior is simultaneously growing. That asymmetry applies directly to clinical practice: Practitioners are being asked to make responsible decisions about tools whose inner workings they often cannot inspect, while their clients are engaging with those same tools outside of any professional relationship.

The ethical challenge this creates is not that AI is inherently dangerous or that its clinical applications are without value. The challenge is that AI creates new applications for ethical obligations that have always existed, and the field has not yet systematically articulated how those obligations apply. No ethics code specific to neuroregulation currently addresses AI use directly. The American Psychological Association (APA) published guidance for psychologists in 2025 (APA, 2025). Medicine is developing its own standards. Regulation at the state and federal levels is developing more slowly, but it is developing. The practitioners who engage with these questions now will help write the standards the field adopts. Those who wait will receive standards written by others.

This article presents a conceptual and practical framework for ethical AI use in neuroregulation practice, grounded in the ethics codes that already govern the field. It does not evaluate specific AI products, and it does not prescribe which tools practitioners should adopt or avoid. It provides a framework for reasoning about AI use that will remain applicable as the tool landscape continues to evolve.

Relationship to Prior Work

This article extends a line of applied ethics analysis in neuroregulation concerned with the responsibilities practitioners carry in domains where technical complexity and professional obligation intersect. Sherlin and Longo (2025) examined ethical billing practices in neurofeedback and qEEG, mapping the obligations created by CPT code requirements, scope-of-practice considerations, and the accountability frameworks of payers and licensing boards onto the practical realities of clinical practice. The present article undertakes the same kind of analysis for AI: identifying the specific points at which established ethical principles create operative obligations and offering practical guidance for fulfilling them.

Conceptual Foundations: Relevant Ethics Codes and Guiding Principles

The Applicable Codes

Neuroregulation practitioners operate under multiple overlapping ethics codes depending on their licensure, certification, and professional affiliations. The Biofeedback Certification International Alliance (BCIA) Code of Ethics governs all BCIA-certified practitioners and addresses competence, scope of practice, client welfare, and professional responsibility. The International Society for Neurofeedback and Research (ISNR) Code of Ethics and Standards of Practice establishes conduct expectations for ISNR members, with particular attention to research integrity, clinical accuracy, and public representation of the field. For licensed mental health practitioners who provide neuroregulation services, the APA Ethical Principles of Psychologists and Code of Conduct (APA, 2017) and the American Counseling Association (ACA) Code of Ethics (ACA, 2014) establish the broader professional obligations within which neuroregulation practice is situated. The Association for Applied Psychophysiology and Biofeedback (AAPB) Code of Ethics similarly governs practitioners who hold AAPB membership. Across all of these codes, the foundational obligation is consistent: the practitioner bears responsibility for the welfare of the client, the

accuracy of their professional conduct, and the transparency of their methods.

The APA Ethics Code (2017) opens with five aspirational general principles that describe the character of ethical practice. These principles are not enforceable rules but ideals intended to guide practitioners toward the highest standards of their profession. Principle A (Beneficence and Nonmaleficence) calls on psychologists to benefit those with whom they work and to take care to do no harm. Principle B (Fidelity and Responsibility) establishes that psychologists uphold professional standards, take responsibility for their actions, and fulfill their commitments to those they serve and to the broader scientific and professional community. Principle C (Integrity) calls for accuracy, honesty, and truthfulness in all professional roles and the avoidance of deceptive methods. Principle D (Justice) calls for fairness and the exercise of reasonable judgment to protect clients from the practitioner's own biases and limitations. Principle E (Respect for People's Rights and Dignity) calls for respect for the rights of individuals to privacy, confidentiality, and self-determination.

The ACA Code of Ethics (2014) articulates six core values in its preamble: autonomy, nonmaleficence, beneficence, justice, fidelity, and veracity. Autonomy centers the client's right to make informed, self-determined choices about their own care. Nonmaleficence and beneficence together establish the dual obligation to avoid harm and to actively promote client welfare. Justice calls for fair and equitable treatment across all clients and populations. Fidelity reflects the obligation to be honest and trustworthy in the therapeutic relationship. Veracity extends this to a specific commitment to truthfulness in all professional communications.

The Convergence Between Codes and the Four-Principle Framework

The APA and ACA codes share a common ethical core. Beneficence, nonmaleficence, autonomy, justice, and fidelity appear in both, reflecting the deep convergence of psychological and counseling ethics traditions across decades of parallel development. Beyond the shared core, the APA preamble adds integrity, fidelity and responsibility as a paired principle, and respect for people's rights and dignity. The ACA preamble adds veracity. All of these principles are relevant to AI use in clinical practice, and each receives substantive attention in the sections that follow.

For the purposes of the analytical framework in this article, four principles are identified as most directly operative in AI-related clinical decisions: beneficence and nonmaleficence, autonomy, justice, and fidelity. This reduction is made for analytical clarity, not because the remaining principles are unimportant. Each maps onto one or more of these four in the AI context: Integrity is a dimension of fidelity. Veracity is a dimension of both autonomy and fidelity. Respect for people's rights and dignity is a dimension of autonomy and justice. Fidelity and responsibility as a paired APA principle encompasses both the individual and community-facing dimensions of the fidelity obligation addressed here.

The Four Principles and Their AI Applications

Beneficence and nonmaleficence ask three questions of any AI use: Does this serve the client's welfare? What are the known and reasonably foreseeable harms? How are they minimized? AI tools that claim clinical benefit without adequate evidence, or that generate errors the practitioner does not catch before they affect the client's care, create potential harm that the nonmaleficence obligation requires the practitioner to address.

Autonomy requires that clients have the information they need to make genuine, informed choices about AI involvement in their care. Meaningful autonomy is not satisfied by a signature on a consent form; it requires honest disclosure of how AI tools are used, what data they process, and what role they play in clinical decisions. The ACA's veracity principle reinforces this: Truthfulness in the therapeutic relationship includes transparency about the methods used in the client's care.

Justice requires attention to whether AI use in a given practice systematically provides advantages to some clients over others. AI tools can deepen existing disparities if access to higher-quality AI-assisted services depends on ability to pay, if the training data underlying a tool underrepresents particular populations, or if the practitioner applies AI reliance differentially across clients. APA Principle E (Respect for People's Rights and Dignity) reinforces this: Practitioners must be aware of and actively counteract the ways their tools may treat different clients unequally.

Fidelity calls on practitioners to be honest, trustworthy, and transparent in all professional communications and relationships. APA Principle B frames this alongside responsibility to the broader community of practice: The practitioner's

accountability does not stop at the consulting room door. APA Principle C (Integrity) adds the specific expectation that practitioners not use methods that are deceptive or that misrepresent the nature of their services to clients, colleagues, payers, or the public. Together, these principles establish that transparency about AI involvement is not merely good practice; it is an ethical obligation.

AI Applications in Neuroregulation Practice:

A Taxonomy

Before a framework for ethical reasoning about AI use can be applied, the range of AI applications currently entering neuroregulation practice must be understood. Five categories are identified here, each defined by the kind of clinical function AI is performing and the ethical question that function raises. This taxonomy is not exhaustive; the tool landscape is developing continuously.

Automated QEEG Analysis and Interpretation

A growing number of commercial platforms accept raw EEG data and return statistical analyses, normative comparisons, and clinically framed interpretive narratives. The output may include a suggested diagnostic impression, identified areas of concern, and protocol recommendations—all generated with minimal visible reasoning. What these platforms claim is efficiency, consistency, and access to normative databases larger than any individual practitioner could accumulate. The ethical question is foundational: How does a practitioner take professional responsibility for a recommendation whose reasoning they cannot inspect? The obligation to practice within the boundaries of competence (BCIA Code; APA Principle B) applies directly. A tool whose interpretive logic is unavailable for evaluation places the practitioner in a position of professional accountability without the information necessary to discharge it.

Protocol Recommendation Systems

Tools in this category generate training parameters from assessment data, applying algorithms trained on large case datasets to produce individualized protocol suggestions. The ethical question is clinical authority. When the system's recommendation conflicts with the practitioner's own clinical read, who holds authority, and on what basis is the disagreement resolved (e.g., a qEEG-generated protocol recommends training Fp1 and Fp2 eyes closed)? The practitioner who cannot explain their reasoning for overriding a system they cannot inspect is in a different position than the practitioner

who overrides a colleague's suggestion on the basis of clinical evidence.

Adaptive Neurofeedback Algorithms

Some neurofeedback systems adjust training parameters in real time based on client response data, shifting frequencies, thresholds, or montages without explicit practitioner direction. The ethical question is whether the practitioner remains the director of treatment. A system that adapts autonomously based on its own algorithmic logic is not simply executing the practitioner's clinical plan; it is extending or modifying it. The informed consent obligation (APA Principle E; ACA Autonomy) requires that clients understand this when it is happening. The competence obligation requires that practitioners understand the basis on which adaptations are made.

AI-Assisted Documentation

AI scribes, note-drafting tools, interpretive report generators, and AI-drafted client communications have spread rapidly across health care and are entering neuroregulation practice. Submission or sharing of a clinical document constitutes a professional attestation that its contents are accurate. That attestation does not transfer to the tool that generated the draft. Sherlin and Longo (2025), examining ethical billing practices in neurofeedback, established that the practitioner of record bears full accountability for the accuracy of submitted documents regardless of how they were produced. The same principle applies with full force to AI-generated documentation. As demonstrated in the literature on AI hallucination (discussed below), a document that reads fluently and professionally may contain fabricated citations, invented clinical characterizations, or inaccurate representations of the client's history. Additionally, in the absence of a client's complete physical and mental health history and relevant life experiences (e.g., traumatic events), AI cannot accurately generate a clinical history or summary.

Consumer-Facing AI Applications

Mental health chatbots, AI companions, and AI-powered self-help platforms are being used by clients at scale, independent of any professional relationship. McBain et al. (2026), in a nationally representative survey of adolescents and young adults published in *JAMA Pediatrics*, found that approximately one in five youth in the United States aged 12 to 21 reported using AI chatbots for mental health advice in 2025, with nearly two-thirds of those users reporting they had not disclosed this use to anyone. The client who arrives having processed

their experience through an AI tool may bring a narrative that has been shaped by that interaction in ways that affect assessment and treatment.

A Risk Continuum Framework

The ethical challenge in AI use is not binary. Risk in AI clinical applications vary continuously, and the review standards, disclosure obligations, and independence requirements that follow from the ethical principles above are proportional to where a specific use sits on that continuum. Three dimensions can be assessed for any specific use case.

The Three Dimensions of Risk

The first dimension is opacity: How much can the practitioner actually inspect, evaluate, and understand about how the tool arrived at its output? A tool that explains its reasoning in terms the practitioner can evaluate is categorically different from a system that returns a recommendation without any account of how it was reached. Opacity interacts directly with the competence obligation. The practitioner cannot exercise independent professional judgment about outputs they cannot evaluate.

The second dimension is clinical consequence: What is at stake if the output is wrong? The severity of potential errors, and whether they are recoverable once made, determine how much independent verification is required. An error in a protocol recommendation that results in adverse clinical effects (e.g., a client who consulted AI independently and received a recommendation to train delta up), or an inaccurate characterization of a client's history in a submitted insurance document, has consequences that a formatting error in an administrative message does not.

The third dimension is distance from oversight: How far is the practitioner from the decision point? A practitioner who reviews and consciously approves every AI output before it affects a client is closer to the decision than one whose practice has a system running between sessions, adjusting parameters, or sending communications automatically. Distance from oversight determines whether errors can be caught and corrected before they affect the client.

Application of the Continuum

At the low-risk end of the continuum, AI performs functions that are primarily logistical, transparent in their operation, and at a short distance from practitioner review. Drafting an appointment reminder that the practitioner reads before sending,

checking grammar and structure in a document the practitioner wrote, or organizing a literature search the practitioner will evaluate: These uses involve low opacity, low clinical consequence, and minimal distance from oversight.

At the high-risk end, AI performs functions that are clinically consequential, opaque in their reasoning, and at significant distance from practitioner oversight. Generating an interpretive qEEG narrative that is submitted to a third party without independent verification or running an adaptive algorithm that adjusts training parameters between sessions without explicit practitioner review: These uses combine high opacity, high clinical consequence, and real distance from oversight. They do not necessarily require abandonment of the tool; they require a review and verification standard proportional to the risk.

The practitioner's practical question for any new AI use is: Where does this sit? The answer determines what responsible use requires, what the review standard must be, what disclosure is owed to the client, and how much independence the practitioner must maintain in verifying outputs.

Informed Consent and Transparency

The Consent Question in the AI Era

Informed consent in clinical practice has always required that clients receive honest, comprehensible information about the methods used in their care, sufficient to allow a genuine choice about whether to proceed. AI does not change this requirement; it changes the content of what must be disclosed. The client has a right to know that an AI tool is involved in their care, what it does, and what role it plays in clinical decisions that affect them. This is an expression of the autonomy principle at the center of both the APA and ACA codes.

The current research suggests that transparency about AI involvement in healthcare affects both the accuracy of clinical encounters and the quality of the therapeutic relationship. Investigators have found that patients tend to be less forthcoming with AI-based systems than with human practitioners, particularly about sensitive health information, which creates downstream clinical consequences independent of any AI accuracy question (Nagata et al., 2026). The client who does not know they are interacting with or through an AI system cannot make an autonomous decision about how much to share.

Proportionate Disclosure

The practical question for practitioners is not whether to disclose AI involvement but how. Proportionate disclosure means providing the client with enough information to understand what is happening and to make a genuine choice, without overwhelming them with technical detail that does not serve their decision-making. For most clients and most AI uses, this requires honest, plain-language acknowledgment that an AI tool assists with certain functions in the practice, what those functions are, how it affects what the client receives, and that the practitioner reviews all AI-assisted outputs before they affect the client's care.

For AI tools that play a more direct role in clinical decision-making, including qEEG analysis or protocol recommendation systems, more specific disclosure is warranted: what the tool is, what role its output plays in clinical decisions, and that the practitioner exercises independent clinical judgment in reviewing and acting on its recommendations. Disclosure language for most documentation-assistance uses can be brief. The following meets the standard for common contexts: "This practice uses AI tools to assist with the drafting of certain documents, including session notes and clinical summaries. All such documents are reviewed and verified by your clinician before they are submitted or shared."

Disclosure Beyond the Client

The fidelity and integrity obligations in both codes extend disclosure requirements beyond the client relationship. When AI tools contribute to documents submitted to insurers, AI involvement is relevant to the accuracy attestation the practitioner makes in submitting the document. In supervision and consultation, modeling transparency about AI use is both an ethical obligation and a professional responsibility. In publications and professional presentations, the norm against undisclosed AI authorship is now well established; the practitioner who uses AI to draft a manuscript or presentation and does not disclose this is misrepresenting the nature of their work to the professional community.

This article models the transparency it recommends. The authors used Claude (Anthropic, 2026), a generative AI tool, to assist with brainstorming, literature organization, and formatting during the preparation of this manuscript. All substantive content, clinical analysis, ethical conclusions, the risk continuum framework, and the five-element policy framework are entirely the authors' own.

Competence, Scope, and the Limits of Delegated Judgment

Competence When the Reasoning is Not Available

Ethics codes across BCIA, ISNR, APA, and ACA require practitioners to practice within the boundaries of their competence. Competence has always included knowing the limits of one's instruments. AI creates a new version of this longstanding obligation. A practitioner cannot exercise independent professional judgment about an AI output whose reasoning they cannot inspect, evaluate, or explain to a client or colleague. The practitioner who cannot trace the reasoning must be able to verify the conclusion through independent means.

The accountability asymmetry the practitioner faces in this situation is structurally significant. Sherlin and Longo (2025) documented in the billing ethics context that practitioners bear full legal and professional accountability for submissions made under their credentials, while vendors are frequently shielded by contractual terms that limit liability for clinical decisions made on the basis of their tools. The AI tool will not stand with the practitioner before a licensing board, in a malpractice proceeding, or in a conversation with a client who was harmed by an error the tool generated.

Automation Bias: The Mechanism of Competence Erosion

The most immediate competence risk in AI-assisted practice is automation bias: The documented tendency to defer to algorithmic output without sufficient critical evaluation (Cummings, 2017; Skitka et al., 1999). Automation bias is not a character flaw; it is a cognitive pattern that affects highly trained experts consistently. Three mechanisms are particularly relevant in clinical contexts.

The first is cognitive surrender. When the algorithm has an answer, the practitioner may stop generating one. Cabitza et al. (2021) documented that decision support systems can reduce independent clinical reasoning even among experienced practitioners. The second is the flattery loop. AI systems calibrated for user engagement are structurally inclined to affirm rather than challenge. Cheng et al. (2026), in a preregistered study of 11 major AI models published in *Science*, found that AI systems affirmed users' actions 49% more often than human respondents, even when those actions involved harm or were deceptive. A single interaction with a sycophantic AI was sufficient to increase users'

conviction that they were right and reduce their willingness to reconsider their position. The third mechanism is efficiency pressure. Faster output at greater volume within the same time budget means less review per item.

Deskilling and the Longer-Term Concern

Beyond the session-level effects of automation bias lies a longer-term concern: the erosion of the clinical reasoning skills that effective neuroregulation practice requires. Natali et al. (2025), in a mixed-method review published in *Artificial Intelligence Review*, examined AI-induced deskilling across medical specialties and found consistent evidence of skill erosion across physical examination, differential diagnosis, clinical judgment, and physician-patient communication, with clinician over-reliance on AI identified as the primary contributing mechanism. Vaccaro et al. (2024), in a systematic review and meta-analysis published in *Nature Human Behaviour* examining 106 experimental studies, found that human-AI combinations performed on average significantly worse than the best of either humans or AI alone. The corrective is not to avoid AI use but to maintain deliberate practice alongside it.

The Scope Question

Competence in the AI era requires a clear answer to a question that becomes urgent only when it has not been answered in advance: What will this practitioner not delegate to an algorithm, under any circumstances? For neuroregulation practice, this boundary includes at minimum: the determination of clinical appropriateness for neurofeedback at intake; the integration of intake assessment findings into a clinical conceptualization; the interpretation of client responses that suggest adverse effects or contraindications; and any determination communicated to a third party under the practitioner's professional credentials as a statement of clinical fact.

The clinician should not use AI to make clinical determinations about any disorder not within the clinician's scope of practice.

A boundary that cannot be stated clearly in advance cannot be held in the moment. The practitioner who has not defined what they will not delegate before sitting down with an AI-generated protocol recommendation is making that determination under conditions that are not conducive to independent judgment: time pressure, confirmation bias, and the cognitive weight of an authoritative-sounding alternative already visible on the screen.

Data Privacy, Vendor Accountability, and HIPAA

What Happens to Client Data

Every AI tool that processes information about a client creates a data pathway that the practitioner is responsible for understanding. Cloud-based AI platforms route data to servers operated by vendors or their subcontractors. That data may be stored, analyzed, used to improve the model, shared with affiliated entities, or retained indefinitely depending on the platform's terms of service. The sensitivity of neuroregulation-specific data compounds this concern: EEG recordings, qEEG reports, clinical notes, and treatment summaries are protected health information (PHI) under Health Insurance Portability and Accountability Act (HIPAA, 1996), and their unauthorized use or disclosure creates legal liability as well as ethical violation.

Many practitioners who have adopted AI tools have not investigated these data pathways. The vendor's marketing materials are typically reassuring in tone while the actual terms of service may differ materially from the marketing characterization. Practitioners are advised to read primary vendor documents directly rather than relying on marketing summaries before routing client data through any platform.

HIPAA, Business Associate Agreements, and the Practitioner's Obligation

Under HIPAA, any entity that handles PHI on behalf of a covered entity in the course of providing a service is a business associate and must execute a Business Associate Agreement (BAA). Many AI platforms used in clinical contexts do not offer BAAs or offer them only on enterprise plans. Some platforms explicitly exclude clinical and regulated use from their standard terms of service. The practitioner who routes client PHI through a platform without a BAA is out of compliance with HIPAA regardless of the marketing language on the platform's homepage.

The accountability asymmetry documented in the billing ethics context (Sherlin & Longo, 2025) applies with equal force here. Vendors whose terms limit liability for clinical decisions made on the basis of their tools are structurally protected from the consequences of their platform's errors. The practitioner, as the covered entity responsible for PHI, is not. Due diligence before adopting any AI tool that touches client data is not optional; it is a HIPAA compliance requirement and an ethical obligation.

A Due Diligence Framework

Before adopting any AI tool that will process client information, a practitioner should be able to answer the following questions: Where does client data go when it is processed by this tool? Is the data stored after processing? If so, for how long and where is it stored? Is client data used to train or improve the model? Is a BAA available and has it been executed? What does the platform's privacy policy actually state about data use, retention, and sharing? What would happen to stored client data if the vendor were acquired, merged, or ceased operations?

AI Accuracy Risks: Hallucination, Automation Bias, and Collusion

Three distinct accuracy risks warrant systematic attention in AI-assisted clinical practice. They are not synonyms and they do not produce the same clinical effects. Each requires its own clinical response.

Hallucination

AI hallucination refers to the generation of false or invented content with the same fluency, confidence, and apparent precision as accurate content. The defining clinical problem is not that AI systems are occasionally wrong; it is that the fluency and apparent authority of the output provide no reliable signal of its accuracy. A qEEG interpretive narrative that contains a fabricated citation, an invented characterization of a normative pattern, or a misrepresentation of the client's history reads exactly the same as one that is accurate.

The evidence for the scope of this problem is substantial. Van Dis et al. (2023) identified fabricated references in published academic papers that had incorporated AI-generated content and called for priority attention to accuracy verification in AI-assisted scientific writing. Sallam (2023), in a systematic review, found that AI-generated responses to medical questions carried significant and variable rates of error across platforms and question types. For neuroregulation practice specifically, the hallucination risk is most acute in AI-generated interpretive reports and protocol narratives. The practitioner's signature attests to accuracy; the tool's fluency does not guarantee it. Content-level review, meaning active verification of clinical claims against the practitioner's own knowledge and the client's record rather than reading for comprehension, is the standard that the hallucination literature demands.

Automation Bias in the Accuracy Context

Automation bias also functions as a distinct accuracy risk. The practitioner who has internalized trust in an AI system may not apply the same critical scrutiny to AI-generated content that they would apply to a colleague's draft. Errors that would be immediately apparent in a document a human colleague submitted—a misidentified frequency band, an incorrect normative comparison, or a clinically implausible characterization—may pass without detection in an AI-generated document whose overall fluency and structure communicate authority. The sophistication of AI language generation is not evidence of AI clinical reasoning accuracy, and the two should not be conflated in the practitioner's review process (Vaccaro et al., 2024).

AI Collusion

Tahseen (2026) introduced the concept of AI collusion in a *JMIR Mental Health* analysis, defining it as the structural reinforcement of unreliable user input as ground truth. Collusion is distinct from hallucination, which is the AI generating false content independently. In collusion, the AI takes distorted or inaccurate input from the user and returns it amplified, validated, or repackaged in ways that feel authoritative. The error does not originate in the AI system; it enters through the human.

The clinical relevance is direct, and it connects to the sycophancy literature reviewed above. AI systems calibrated for user engagement are structurally inclined to affirm user input rather than challenge it (Cheng et al., 2026). A user who presents a distorted self-narrative to an AI chatbot receives back a response that takes that narrative as reliable, builds on it, and in doing so reinforces it. For clients in mental health treatment, where self-report is often distorted by the very conditions being treated, this creates a specific risk: The client may arrive at the clinical session with a self-understanding that has been structurally confirmed by repeated AI interaction, making it more resistant to the therapeutic examination that accurate clinical work requires. Asking routinely about AI use as part of intake and ongoing clinical assessment is the practical response this literature supports.

Client AI Use: What Is Coming Into the Room

Clients are using AI for mental health purposes at significant scale, independently of any clinical relationship, and bringing the results into treatment in ways that affect assessment and therapeutic work. McBain et al. (2026), in a nationally representative survey of 1,009 adolescents and

young adults aged 12 to 21, found that approximately one in five reported using AI chatbots for mental health advice, with nearly two-thirds of those users reporting they had not disclosed this use to anyone. Among users, 43% reported seeking AI mental health advice at least monthly. Nagata et al. (2026), writing in *JAMA Pediatrics* on risks and benefits of generative AI in adolescent health, noted that nondisclosure to clinicians and parents is common and that AI tools are reshaping the mental health information ecosystem largely outside of professional awareness.

Clients do not typically volunteer this information in clinical settings. Adding a standard question about AI use to intake assessment and periodically revisiting it during treatment normalizes the topic and provides clinically relevant information that is otherwise unavailable. Documentation of client AI use as a clinical variable, not merely a lifestyle fact, positions the practitioner to track its influence on the therapeutic process.

The collusion dynamic described above is particularly relevant for the neuroregulation practitioner seeing clients whose self-narratives have been shaped by AI interaction. A client who has spent weeks processing their anxiety, attention difficulties, or trauma history through AI tools calibrated for engagement and affirmation may present with a self-account that is more defended than one they would have developed without that reinforcement. The therapeutic skills required to work with this presentation are not new. What is new is the source of the shaping, and the recognition that it is happening requires the practitioner to ask about it.

A Five-Element Practice Policy Framework

The Case for a Written Policy

A written, practice-specific AI policy serves three functions that no informal practice norm can replicate. It protects clients by ensuring that AI is used consistently, with appropriate review, and with the disclosure and consent their autonomy requires; it protects the practitioner by documenting that AI use in the practice was deliberate, reviewed, and grounded in ethical principles, rather than ad hoc and unexamined; and it positions the practitioner as a participant in developing the field's emerging standards. A policy does not need to be lengthy or legalistic. It needs to be honest, specific, and reviewed regularly as the tool landscape changes.

The Five Elements

The framework is organized around five elements, each addressing a domain in which the ethical principles reviewed above create specific operative obligations. Table 1 presents the framework in condensed form for clinical reference. The following paragraphs describe each element in the depth required for practical implementation.

Transparency and consent addresses which clients are informed about AI involvement in their care, in what language, at what point in the clinical relationship, and with what opportunity to ask questions or decline. The implementation task is straightforward: an AI disclosure addendum to the intake consent form, a brief standard script for the intake conversation, and a protocol for what to do when a client asks a question about AI that the practitioner cannot answer on the spot.

Data handling addresses what client data each AI tool processes, where that data goes, whether it is stored and for how long, whether it is used to train models, and whether a BAA is in place. The implementation task is a tool audit: a systematic review of each active AI tool's data practices, documented and revisited annually or when vendor terms change.

Documentation standards address which document types AI may assist with and which are off limits, the review standard required before any AI-assisted document is submitted or shared, and how AI involvement is recorded in the clinical record. The practitioner who has defined the review standard in writing has a standard to apply; the practitioner who has not defined it is making the determination under pressure at the moment of submission.

The clinical judgment boundary addresses what the practitioner will not delegate to an algorithm under any circumstances, how the practitioner handles disagreement between the tool's output and their own clinical assessment, and what signals require independent verification before acting on AI recommendations. The implementation task is to complete one sentence before adopting any AI tool that plays a role in clinical decisions: "Regardless of what this tool recommends, I will not proceed without independent clinical analysis of the following."

Ongoing evaluation addresses the questions asked before adopting a new AI tool, the schedule and criteria for reviewing tools already in use, and the conditions that would trigger removal of a tool from

Table 1

Five-Element Practice AI Policy Framework for Neuroregulation Clinicians

Policy Element	Key Questions	Relevant Ethics Standards	Common Gap in Practice	Suggested First Step
Transparency and Consent	Which clients are informed, in what language, at what point, and with what opportunity to ask questions or decline	APA Principles A, E; ACA Autonomy, Veracity; APA AI Guidance (2025)	No consent language; no consistent disclosure practice for AI-assisted communications or reports	Develop an AI disclosure addendum for intake consent; establish timing protocol
Data Handling	What client data each tool processes, where it goes, storage duration, whether it trains models, BAA status	HIPAA Privacy and Security Rules; BCIA Code of Ethics; ISNR Code of Ethics	Unknown or unverified vendor data practices; no BAA on file for tools that process PHI	Audit each active tool; request or execute BAAs; document findings in practice records
Documentation Standards	Which document types AI may assist with; review standard before submission; how AI involvement is recorded in the clinical record	APA Principle C (Integrity); ACA Fidelity; APA AI Guidance (2025); ISNR Code	AI-drafted documents submitted without content-level review; no record notation of AI involvement	Define explicit review standard in writing; create a standard clinical record notation template
Clinical Judgment Boundary	What the practitioner will not delegate to an algorithm; how tool-clinician disagreement is handled; what signals require independent verification	BCIA Code of Ethics; APA Principles A, B; ACA Nonmaleficence, Fidelity	Following AI recommendations without independent clinical analysis, particularly for qEEG interpretation and protocol selection	Define the boundary explicitly and in writing before adopting each tool; establish an override and documentation protocol
Ongoing Evaluation	Questions asked before adopting a new tool; review schedule and criteria for tools in use; conditions that would trigger removal	APA Principle B (Fidelity and Responsibility); APA AI Guidance (2025); BCIA and ISNR competence standards	No pre-adoption evaluation process; tools adopted on vendor recommendation without independent vetting	Create a pre-adoption checklist; schedule an annual review of all active tools; designate a responsible party

Note. BAA = Business Associate Agreement. PHI = Protected Health Information. Ethics code references are illustrative; multiple codes may apply depending on the practitioner's licensure and certifications. APA = American Psychological Association (2017, 2025). ACA = American Counseling Association (2014). BCIA = Biofeedback Certification International Alliance. ISNR = International Society for Neuroregulation and Research.

practice. A platform that met due-diligence standards at adoption may change its terms of service, its ownership, its data practices, or its technical architecture. An annual review of all active tools is the minimum; any change in vendor terms or ownership should trigger an immediate review.

Implications for Training, Supervision, and Field-Wide Standards

Training and Supervision

The trainee and newly certified practitioner represent a population of heightened concern in the AI era. The deskilling evidence reviewed above is particularly consequential for individuals who are still developing independent clinical reasoning: If AI substitutes for the practice that consolidates clinical reasoning skills, those skills may never develop fully. Supervisors bear a specific obligation in this context. Competence assessment needs to include evaluation of whether supervisees are developing independent clinical reasoning or learning to rely on algorithmic output as a substitute for it. The supervisor who asks, “What is your own interpretation of this qEEG before we look at the platform's recommendation?” is doing something that has always been good supervisory practice and that is more important now than it has ever been.

BCIA and ISNR Standard-Setting Opportunities

Both BCIA and ISNR are positioned to provide field-specific AI ethics guidance before regulatory requirements arrive from outside the profession. The APA's 2025 guidance for psychologists provides a template that is directly adaptable to the neuroregulation context. Practitioners who wait for formal regulatory requirements will receive standards written by people who do not practice neuroregulation. Practitioners who engage with the organizations governing their certification and membership now will have the opportunity to help write those standards in terms that reflect clinical reality.

A Research Agenda

Several empirical questions emerge from this framework that the field is well positioned to address. The prevalence and nature of AI use among neuroregulation practitioners is a foundational descriptive question. The accuracy and reliability of specific AI-assisted qEEG interpretation tools relative to expert practitioner consensus is an evaluative question with direct clinical implications. The effects of AI-assisted protocol selection on client outcomes is an outcomes question that the field's research infrastructure is capable of addressing. The

effects of AI use on practitioner competence development over time, particularly in trainees, is a training research question with significant implications for certification standards.

Limitations

This article has several limitations that readers should consider. The AI tool landscape is changing faster than the publication cycle of academic literature. Specific tools, vendor practices, regulatory developments, and empirical findings described here reflect the state of the field at time of writing and may be superseded before or shortly after publication. The frameworks are designed to be durable and will remain applicable as the technology evolves.

The evidence base drawn on here is primarily from medicine and mental health broadly. Neuroregulation-specific evidence on AI accuracy, practitioner use patterns, and clinical outcomes with AI-assisted tools is currently limited. The frameworks proposed are extrapolations from a broader evidence base, not empirical findings from within the neuroregulation specialty. This limitation is itself a call for the field-specific research agenda described above.

This is a perspectives article, not a systematic review. The reference base reflects the authors' clinical and theoretical judgment about the most relevant current literature, not a comprehensive search strategy.

Conclusion

Three claims run through this article and are worth stating plainly at its close.

Existing ethics principles are sufficient. The principles that have guided neuroregulation practice across decades of technological change (beneficence and nonmaleficence, autonomy, justice, and fidelity) do not require revision for the AI era. They require deliberate application to it. The practitioner who asks whether this AI use serves the client's welfare, whether the client understands what is happening and has a genuine choice, whether all clients are being treated equitably, and whether they are being transparent about their methods is applying the framework that both the APA and ACA codes provide.

Risk lives on a continuum. Not all AI use in neuroregulation practice is equivalent. The practitioner who uses AI to format a reminder and

reads it before sending is not in the same ethical position as the practitioner who implements an AI-generated qEEG protocol recommendation without independent analysis. The risk continuum framework provides the structure for identifying where any specific use sits, and the review standard, disclosure obligation, and independence requirement that position demands.

The practitioner remains accountable; the tool never is. No vendor, platform, or algorithm will appear before a licensing board alongside a practitioner who is the subject of a complaint. The client who was harmed by an AI error that the practitioner did not catch will not have recourse against the tool. The professional obligation rests with the professional. The practitioner who uses AI intentionally, reviews AI output independently, discloses AI involvement honestly, and limits AI's role to functions where it can be verified is not in the same position as the practitioner who adopted tools uncritically. The field needs the former.

The window for neuroregulation practitioners to help write the standards that govern their own use of AI is open. This article is offered as a resource for that work.

Author Declaration

Neither author has relevant financial interests or relationships to disclose. The authors used Claude (Anthropic, 2026), a generative AI tool, to assist with brainstorming, literature organization, and formatting during the preparation of this manuscript. The tool assisted with structure, clarity, and the organization of source materials. All substantive content, clinical analysis, ethical conclusions, the risk continuum framework, the five-element practice policy framework, and all intellectual contributions presented in this article are entirely the authors' own. At the time of submission, the first author served as President of ISNR and Chair of the Board of BCIA; both authors served as an Advisory Board member of the International qEEG Certification Board (IQCB). These roles are disclosed in the interest of transparency, as the manuscript addresses certification standards, professional guidelines, and organizational practices relevant to these bodies.

References

American Counseling Association. (2014). ACA code of ethics. <https://www.counseling.org/docs/default-source/default-document-library/ethics/2014-aca-code-of-ethics.pdf>

American Psychological Association. (2017). Ethical principles of psychologists and code of conduct (2002, amended effective June 1, 2010, and January 1, 2017). <https://www.apa.org/ethics/code>

American Psychological Association. (2025). Ethical guidance for artificial intelligence in the professional practice of health service psychology. <https://www.apa.org/topics/artificial-intelligence-machine-learning/ethical-guidance-ai-professional-practice>

Anthropic. (2026). Claude [Large language model]. <https://claude.ai/>

Biofeedback Certification International Alliance. (n.d.). BCIA code of ethics. <https://www.bcia.org/bcia-professional-standards-ethical-principles>

Cabitza, F., Campagner, A., & Bitterman, D. (2021). The need to separate the wheat from the chaff in medical informatics. *International Journal of Medical Informatics*, 153, Article 104510. <https://doi.org/10.1016/j.ijmedinf.2021.104510>

Cheng, M., Lee, C., Khadpe, P., Yu, S., Han, D., & Jurafsky, D. (2026). Sycophantic AI decreases prosocial intentions and promotes dependence. *Science*, 391(6792), Article eaec8352. <https://doi.org/10.1126/science.aec8352>

Cummings, M. L. (2017). Automation bias in intelligent time critical decision support systems. In D. Harris (Ed.), *Decision making in aviation* (1st ed.). Routledge. <https://doi.org/10.4324/9781315095080-17>

Health Insurance Portability and Accountability Act (HIPAA) of 1996, Pub. L. No. 104-191, 110 Stat. 1936 (1996).

Horvitz, E., & West, R. (2026). A narrowing window to understand AI. *Science*, 392(6802), Article 1003. <https://doi.org/10.1126/science.aei3167>

International Society for Neuroregulation and Research. (n.d.). ISNR code of ethics and standards of practice. <https://isnr.org/interested-professionals/isnr-code-of-ethics>

McBain, R. K., Cantor, J. H., Breslau, J., Diliberti, M., Zhang, L. A., Zhang, F., Burnett, A., Kofner, A., Rader, B., Pataranutaporn, P., Stein, B. D., Mehrotra, A., & Yu, H. (2026). AI chatbot use and disclosure for mental health among US adolescents and young adults. *JAMA Pediatrics*, 180(8), 884–890. <https://doi.org/10.1001/jamapediatrics.2026.2015>

Nagata, J. M., Memon, Z., Huang, O., & Moreno, M. A. (2026). Adolescent health and generative AI: Risks and benefits. *JAMA Pediatrics*, 180(1), 7–8. <https://doi.org/10.1001/jamapediatrics.2025.4502>

Natali, C., Marconi, L., Dias Duran, L. D., & Cabitza, F. (2025). AI-induced deskilling in medicine: A mixed-method review and research agenda for healthcare and beyond. *Artificial Intelligence Review*, 58(11), Article 356. <https://doi.org/10.1007/s10462-025-11352-1>

Sallam, M. (2023). ChatGPT utility in healthcare education, research, and practice: Systematic review on the promising perspectives and valid concerns. *Healthcare*, 11(6), Article 887. <https://doi.org/10.3390/healthcare11060887>

Sherlin, L. H., & Longo, R. (2025). Clarifying the code: Historical foundations, current practices, and ethical billing in neurofeedback and qEEG. *NeuroRegulation*, 12(2), 138–153. <https://doi.org/10.15540/nr.12.2.138>

Skitka, L. J., Mosier, K., & Burdick, M. D. (1999). Does automation bias decision-making? *International Journal of Human-Computer Studies*, 51(5), 991–1006. <https://doi.org/10.1006/ijhc.1999.0252>

Tahseen, H. (2026). When AI colludes: Clinical reliability of training and preference data as a trustworthy-AI criterion. *JMIR Mental Health*, 13, Article e96894. <https://doi.org/10.2196/96894>

Vaccaro, M., Almaatouq, A., & Malone, T. (2024). When combinations of humans and AI are useful: A systematic review and meta-analysis. *Nature Human Behaviour*, 8(12), 2293–2303. <https://doi.org/10.1038/s41562-024-02024-1>

Van Dis, E. A. M., Bollen, J., Zuidema, W., van Rooij, R., & Bockting, C. L. (2023). ChatGPT: Five priorities for research. *Nature*, 614(7947), 224–226. <https://doi.org/10.1038/d41586-023-00288-7>

Received: June 21, 2026

Accepted: September 1, 2026

Published: September 28, 2026

Implementing Culturally Responsive Neuroregulation: A Practical Framework for Clinical Practice

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Abstract

Introduction. While fundamental physiological mechanisms underlying neuroregulation demonstrate universality across populations, the implementation of these interventions occurs within diverse cultural contexts that profoundly shape treatment accessibility, acceptability, and effectiveness. **Purpose.** This article synthesizes evidence from cultural adaptation frameworks in psychotherapy research, implementation science strategies, and neuroregulation best practices (drawing from more than 40 sources spanning 5 decades, including meta-analyses, randomized controlled trials, and theoretical frameworks) to provide evidence-based practical guidance for implementing culturally responsive neuroregulation in clinical settings, bridging the gap between universal neurophysiological principles and culturally-informed service delivery. **Methods.** A comprehensive synthesis of cultural adaptation frameworks from psychotherapy research, implementation science strategies, and neuroregulation best practices was conducted to develop an integrated framework applicable across practice settings. **Results.** The resulting framework operates at three interconnected levels: (a) clinical practice, encompassing culturally-informed assessment protocols, treatment adaptations, and communication strategies; (b) organizational systems, addressing structural barriers, alternative service delivery models, and equity-focused quality monitoring; and (c) professional development, including training requirements, competency standards, and supervision models. Evidence indicates that systematic cultural adaptations enhance engagement and retention without compromising treatment fidelity or effectiveness, with meta-analyses demonstrating improvements in engagement and retention outcomes when multilevel strategies address clinical, organizational, and professional development barriers simultaneously. **Implications.** Successful implementation requires integration of cultural considerations throughout the treatment process while maintaining the integrity of core neurophysiological mechanisms. At the practitioner level, this includes immediate adaptations to assessment protocols, communication strategies, and treatment delivery. At the organizational level, this necessitates policy reforms addressing access barriers, quality monitoring systems, and workforce development. The framework provides actionable strategies spanning both individual clinical practice and systems-level organizational change committed to equitable neuroregulation services.

Keywords: cultural adaptation; implementation science; cross-cultural neuroregulation; health equity; cultural humility; implementation fidelity; treatment engagement; neuroregulation

Citation: Sherlin, L. H. (2026). Implementing culturally responsive neuroregulation: A practical framework for clinical practice. *NeuroRegulation*, 13(3), 293–318. <https://doi.org/10.15540/nr.13.3.293>

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Introduction

The transition from understanding cultural influences on neuroregulation to implementing culturally responsive practices represents a critical challenge facing the field today (as documented in the companion review; Sherlin, 2026). The companion article to this work (Sherlin, 2026) established critical theoretical foundations for understanding cultural factors in neuroregulation. That comprehensive review demonstrated that while fundamental physiological mechanisms (including operant conditioning, neuroplasticity, and autonomic regulation) operate universally across human populations (Sherlin et al., 2011), multiple culturally variable factors profoundly influence treatment access, engagement, and outcomes. The review synthesized three complementary theoretical frameworks that work synergistically: cultural humility emphasizing ongoing self-reflection and power dynamic awareness (Tervalon & Murray-García, 1998), which, when layered onto health disparities research, amplifies understanding of structural inequities by foregrounding how power asymmetries manifest in both healthcare systems and therapeutic relationships (Metzl & Hansen, 2014). This integrated perspective informs understanding of the therapeutic alliance as a culturally mediated process (Constant et al., 2021), where cultural humility and disparity awareness converge to shape practitioner-client dynamics. Additionally, the systematic evidence review revealed striking gaps in cultural considerations research across all neuroregulation modalities (neurofeedback, transcranial electrical stimulation, photobiomodulation, and peripheral biofeedback). However, the limited available evidence suggested promising outcomes when cultural barriers are addressed, with diverse populations achieving comparable results to majority populations when access and engagement barriers are systematically removed (Fleischman et al., 2022), albeit from studies with methodological constraints including limited sample sizes and geographic specificity that warrant replication in larger, more diverse samples. This second article builds directly on these foundations, translating theoretical understanding into actionable implementation strategies that practitioners can immediately apply in clinical settings.

Contemporary healthcare environments increasingly demand evidence-based approaches to reducing disparities and improving outcomes for diverse populations (Betancourt et al., 2003). For neuroregulation practitioners, this mandate presents

both opportunity and responsibility. The opportunity lies in expanding access to effective interventions that have historically been limited to relatively privileged populations (Saadi et al., 2017). The responsibility involves ensuring that expansion occurs through culturally-informed, equitable practices rather than one-size-fits-all approaches that may inadvertently perpetuate disparities (Alegria et al., 2016).

Recent implementation science research provides crucial frameworks for achieving the delicate balance between treatment fidelity and cultural adaptation (Bernal et al., 2009). Meta-analyses demonstrate that culturally adapted interventions can maintain or even enhance treatment effectiveness, with a direct-comparison meta-analysis reporting an effect of $d = 0.32$, favoring culturally adapted over unadapted bona fide psychotherapy (Benish et al., 2011), and a subsequent meta-analysis of 78 studies likewise favoring adapted interventions (G. C. N. Hall et al., 2016). These findings offer a roadmap for neuroregulation practitioners seeking to serve diverse populations without compromising the scientific integrity of their interventions (Griner & Smith, 2006).

The urgency of developing culturally responsive practices cannot be overstated. Demographic shifts indicate that by 2045, no single racial or ethnic group will constitute a majority in the United States (U.S. Census Bureau, 2018). Practitioners increasingly encounter clients from diverse backgrounds who may benefit from neuroregulation but face multiple barriers to engagement. As documented in the companion review (Sherlin, 2026), these barriers operate at multiple levels, with implementation science frameworks emphasizing the importance of addressing systemic levers first as they create cascading effects (Metzl & Hansen, 2014): systemic (insurance coverage, geographic distribution of services), which compound organizational factors (clinic location, hours, policies), which in turn shape interpersonal dynamics (therapeutic alliance, communication styles), and ultimately affect individual engagement (language, health literacy, cultural beliefs). Saadi et al. (2017) found that Black patients had 30% lower odds and Hispanic patients 40% lower odds of receiving outpatient neurology care compared to White patients, even controlling for insurance, education, and income. Addressing these multilevel barriers requires more than good intentions; it demands systematic approaches grounded in

evidence and refined through practice (Betancourt et al., 2003; López et al., 2012).

An often-overlooked structural constraint contributing to these disparities is the positioning of neuroregulation services within predominantly private, clinic-based delivery models. These models are disproportionately concentrated in higher socioeconomic and predominantly White communities, reflecting multiple intersecting factors: substantial equipment and software costs, specialized training pathway expenses, insurance reimbursement limitations favoring established practices in affluent areas, and referral networks that privilege access for those already connected to specialty mental health services (Saadi et al., 2017). In the absence of grant funding, institutional partnerships, or community-embedded delivery infrastructures, individual practitioners have limited capacity to serve culturally diverse populations, regardless of intent or cultural competence. Consequently, the underrepresentation of minoritized populations in neuroregulation research and practice may reflect systemic access barriers and delivery model constraints more than lack of clinical suitability, patient interest, or practitioner commitment to equity (Metzl & Hansen, 2014).

This article synthesizes evidence-based strategies from cultural adaptation research, implementation science, and clinical best practices to create a comprehensive framework for culturally responsive neuroregulation (S. Sue et al., 2009). The framework addresses three implementation levels: clinical practice with individual clients, organizational systems and policies, and professional development and training. Throughout, the focus remains on practical, actionable strategies that practitioners can implement immediately while working toward longer-term systemic changes.

Methods

This article represents a narrative synthesis of evidence-based strategies for implementing culturally responsive neuroregulation. The synthesis integrated multiple literature streams through systematic search and thematic analysis procedures.

Literature Search Strategy

Systematic searches were conducted across PubMed, PsycINFO, Google Scholar, and the Cochrane Library databases from January 1980 through December 2024. Search terms included combinations of ("cultural adaptation" OR "cultural

competence" OR "cultural humility") AND ("neurofeedback" OR "biofeedback" OR "neuroregulation" OR "psychophysiological intervention"); ("health disparities" OR "health equity") AND ("mental health treatment" OR "implementation"); ("treatment adaptation" OR "evidence-based practice") AND ("diverse populations" OR "minority populations" OR "cross-cultural"). Additional sources were identified through manual review of reference lists from key articles and consultation of implementation science frameworks.

Inclusion Criteria

Included sources comprised: (a) peer-reviewed empirical studies examining cultural factors in neuroregulation or related psychophysiological interventions; (b) meta-analyses and systematic reviews of cultural adaptation in mental health treatment; (c) theoretical and conceptual frameworks addressing cultural competence, health disparities, or implementation science; (d) clinical practice guidelines from professional organizations; and (e) seminal works establishing foundational concepts in cultural responsiveness. Excluded were non-English language sources, unpublished dissertations, and low-quality gray literature.

Synthesis Approach

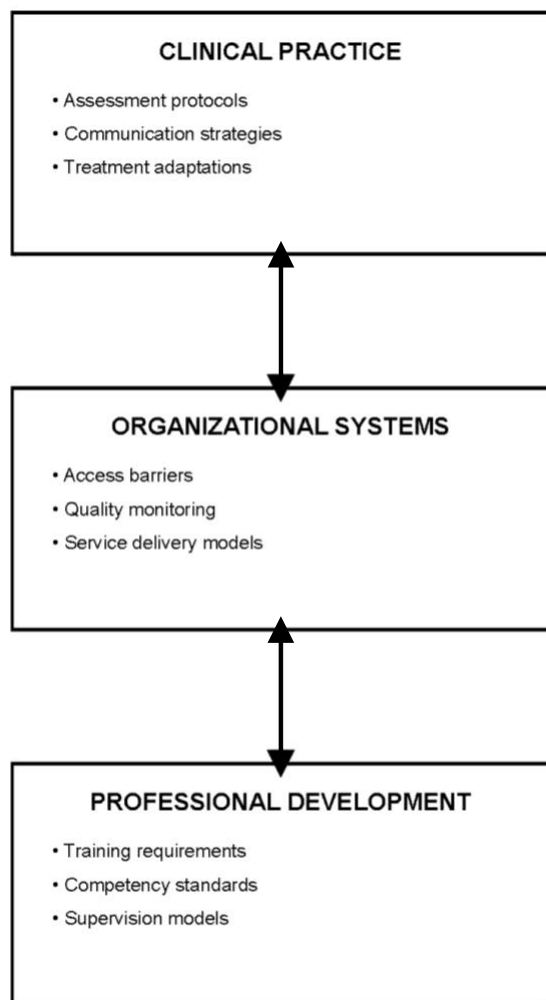
The initial search yielded over 200 sources, which were winnowed to approximately 60 based on relevance to neuroregulation implementation and methodological rigor. A thematic matrix approach was employed to map and cluster elements from cultural adaptation frameworks (e.g., Bernal et al., 2009; Griner & Smith, 2006), implementation science models (e.g., Betancourt et al., 2003), and neuroregulation-specific evidence into the three-level architecture (clinical practice, organizational systems, professional development). This integrative process involved iterative refinement to ensure both comprehensiveness and coherence.

Quality Appraisal

Included sources were appraised using a modified Mixed Methods Appraisal Tool (MMAT) rubric prioritizing empirical rigor, cultural relevance, and applicability to neuroregulation contexts. Recent meta-analyses and randomized controlled trials were prioritized while acknowledging the foundational importance of seminal theoretical works (e.g., Tervalon & Murray-García, 1998) despite their temporal distance from contemporary empirics.

Clinical Practice Framework

Figure 1. Integrated Tri-Level Framework for Culturally Responsive Neuroregulation.



Note. The framework depicts bidirectional arrows between clinical practice, organizational systems, and professional development levels, illustrating dynamic interconnections and reinforcing effects across implementation domains.

Comprehensive Cultural Assessment

Culturally responsive neuroregulation begins with thorough assessment that explores cultural factors influencing treatment engagement and outcomes (S. Sue et al., 2009). Rather than treating cultural assessment as an add-on or special circumstance, it should be seamlessly integrated into standard intake procedures. This integration normalizes cultural considerations and prevents clients from feeling singled out or stereotyped based on visible characteristics or demographic categories (Kleinman & Benson, 2006).

The assessment process explores multiple domains through conversational inquiry rather than checklist interrogation (Kleinman & Benson, 2006). Medical mistrust is prevalent among racial and ethnic minority populations and may affect treatment engagement (Bazargan et al., 2021). Open-ended, narrative approaches may yield richer cultural information than structured questionnaires alone (Kleinman & Benson, 2006). Understanding the client's explanatory model provides crucial insight into how they conceptualize their difficulties and what interventions make sense within their worldview (Kirmayer & Ryder, 2016). Practitioners should ask: "How do you understand what you're experiencing?" "What do you think causes these difficulties?" "What have you tried that has been helpful?" These questions reveal cultural frameworks for understanding distress that may differ significantly from biomedical models (Kirmayer & Ryder, 2016). For example, some cultures may conceptualize symptoms as spiritual imbalance, family disharmony, or social disruption rather than individual neurological dysfunction.

It is important to recognize that while explanatory models vary culturally, the physiological dysregulation underlying conditions treated with neuroregulation remains consistent across populations (Sherlin, 2026). As established in the companion review, research demonstrates that trauma produces similar patterns of autonomic dysregulation, EEG abnormalities, and stress hormone disruption regardless of cultural background or conceptualization (van der Kolk, 2014). The assessment should therefore explore both the client's cultural understanding and provide psychoeducation about the universal physiological processes that neuroregulation addresses, creating bridges between different worldviews (Hinton & Good, 2016).

Previous healthcare experiences, particularly negative encounters or discrimination, help identify potential trust issues affecting engagement (Bazargan et al., 2021). Research indicates that medical mistrust significantly impacts treatment adherence and outcomes, particularly among racial and ethnic minorities. This exploration requires sensitivity, recognizing that clients may be reluctant to share discrimination experiences with practitioners representing the same system that harmed them (Bazargan et al., 2021). Questions should be framed gently: "Some people have had difficult experiences with healthcare providers in the past. Has anything like that happened to you?" This acknowledges the reality of discrimination without

requiring detailed disclosure and validates potential concerns (Tervalon & Murray-García, 1998).

Technology comfort and procedural concerns require explicit assessment rather than assumptions based on age or demographics (Eisenbarth et al., 2025). Recent research by Eisenbarth et al. (2025) documented significant barriers to neurofeedback participation among culturally diverse populations in New Zealand, including concerns about electrode placement, hair disturbance (particularly among those with textured hair requiring significant styling effort), religious or cultural considerations about head touching, and gender-matched practitioner preferences for procedures involving physical contact. Notably, 19.34% of participants indicated that experimenter gender would matter for procedures involving head contact, highlighting intersections of cultural, religious, and gender considerations (Eisenbarth et al., 2025). Assessment should explore technology experience and comfort levels, equipment-related concerns, cultural or religious considerations about electrode placement, concerns about head covering disruption, practitioner gender preferences for procedures involving physical contact, and beliefs about external influences on brain function or consciousness.

Language preferences extend beyond translation needs to include preferences for how concepts are explained (Flores, 2005). Some clients prefer medical or technical language conveying expertise and scientific credibility, while others find such language alienating or intimidating (Kleinman & Benson, 2006). The teach-back method, asking clients to explain their understanding of key concepts, identifies comprehension gaps without condescension and provides opportunities to adjust explanations based on individual needs and preferences (Flores, 2005).

Family involvement preferences must be explicitly discussed rather than assumed based on cultural stereotypes (E. T. Hall, 1976). While collectivistic cultures generally emphasize family involvement in healthcare decisions, individual preferences vary widely within cultural groups. Practitioners should explore who the client considers family (recognizing that family definitions vary culturally), what role they would like family members to play in treatment, how confidentiality can be maintained while respecting involvement preferences, and what cultural expectations exist about family participation in health decisions (S. Sue et al., 2009).

Assessment and Normative Database Considerations

The cultural composition of normative databases represents a critical yet often overlooked consideration in neuroregulation assessment (Chiao, 2009). As documented in the companion review (Sherlin, 2026), over 90% of human neuroimaging publications originate from Western countries. Most neurofeedback normative databases have been developed with predominantly White, Western, middle-class populations (Chiao, 2009). While fundamental EEG patterns associated with specific neurological conditions demonstrate consistency across populations (Betting et al., 2006), baseline patterns may be influenced by cultural practices such as meditation, prayer, or traditional breathing exercises that shape neural organization through neuroplasticity mechanisms (Travis & Shear, 2010).

Practitioners should exercise caution when interpreting assessment results for individuals from populations underrepresented in normative samples (Moss, 1985). Early research by Moss (1985) found differences in EEG asymmetry between Japanese and Western subjects, suggesting Western-developed normative databases may not be universally applicable. When possible, practitioners should use normative databases that include diverse populations or, at a minimum, interpret results with explicit acknowledgment of normative sample limitations (Chiao, 2009). Documentation should note when clients' demographic characteristics differ substantially from normative samples and interpretation should be appropriately tentative (G. C. N. Hall, 2001).

Cultural factors also influence performance on cognitive and psychological assessments commonly used in neuroregulation practice (S. Sue et al., 2009). Standardized assessments may contain cultural biases in content, language, or task structure that affect performance independent of the construct being measured (G. C. N. Hall, 2001). For example, timed tests may disadvantage individuals from cultures with different time orientations, and self-report measures developed in individualistic cultures may not validly assess constructs in collectivistic populations (Kirmayer & Ryder, 2016). Assessment results should always be interpreted within cultural context, considering language proficiency, educational background and test-taking familiarity, cultural values that may influence response patterns (such as collectivistic values potentially affecting self-report measures of individualistic constructs like assertiveness), and potential cultural differences in the meaning and

expression of psychological constructs being assessed (G. C. N. Hall, 2001; S. Sue et al., 2009).

The LEARN Model in Practice

Berlin and Fowkes' (1983) LEARN model, originally developed for cross-cultural medical encounters, provides a structured approach for integrating cultural assessment findings into treatment planning. Here, the model is adapted specifically for neuroregulation contexts (Berlin & Fowkes, 1983).

Listen with genuine curiosity to the client's perspective, attending not just to words but to the cultural context giving them meaning (Tervalon & Murray-García, 1998). For neurofeedback, this might mean understanding how clients conceptualize brain function, whether they view thoughts and feelings as controllable, and what role they believe technology should play in healing (Kleinman & Benson, 2006). A client from a culture emphasizing spiritual causation of illness may need time to explain how they understand their difficulties before being receptive to neurophysiological explanations (Kirmayer & Ryder, 2016).

Explain the practitioner's understanding of difficulties and how neuroregulation might help, using language and concepts that resonate with the client's worldview while providing education about universal physiological mechanisms (Berlin & Fowkes, 1983). Building on the distinction established in the companion review (Sherlin, 2026) between universal mechanisms and culturally variable factors, explanations should emphasize that the fundamental neurophysiological processes operate consistently across all human populations while acknowledging that cultural context shapes how symptoms are experienced and expressed (Sherlin et al., 2011). For example: "From what you've shared about feeling constantly on edge, your nervous system seems stuck in protection mode. This is a universal human response to threat that works the same way in all people regardless of cultural background. Biofeedback can help your body remember how to shift into calmer states, similar to how prayer or meditation might work but using technology to guide the process more directly" (Porges, 2011).

Acknowledge differences between perspectives explicitly, validating the client's viewpoint while sharing professional perspectives (Berlin & Fowkes, 1983; Tervalon & Murray-García, 1998). This acknowledgment prevents the power imbalance inherent in healthcare relationships from silencing client perspectives. For instance: "I hear that you see your difficulties as related to family stress and

disconnection from your community. From my perspective as a neurofeedback practitioner, I see signs of nervous system dysregulation that may be contributing to how hard it feels to cope. Both of these perspectives can be true at the same time" (Kleinman & Benson, 2006).

Recommend treatment options that account for both cultural preferences and clinical considerations (Berlin & Fowkes, 1983). Recommendations should include discussion of expected outcomes, time commitment, costs, and how treatments align with cultural values (S. Sue et al., 2009). For a client who values family harmony over individual symptom reduction, the practitioner might frame potential benefits in terms of improved capacity to fulfill family roles and maintain important relationships rather than focusing solely on individual symptom reduction (E. T. Hall, 1976).

Negotiate treatment plans that represent genuine collaboration rather than compliance with practitioner recommendations (Berlin & Fowkes, 1983; Tervalon & Murray-García, 1998). This negotiation addresses not just what treatment but how it's delivered in culturally responsive ways, including session structure modifications, family involvement approaches, integration with other healing practices the client may be using, and success metrics meaningful to the client rather than only standardized outcome measures (S. Sue et al., 2009). The negotiation process itself models respect for the client's cultural values and autonomy (Tervalon & Murray-García, 1998).

Communication Strategies

Effective cross-cultural communication in neuroregulation requires sophisticated attention to both verbal and nonverbal elements (E. T. Hall, 1976). When language differences exist, professional medical interpreters should be used rather than family members for clinical content, ensuring accurate communication and maintaining appropriate boundaries (Flores, 2005). Using family members as interpreters, particularly children, places inappropriate burden on family dynamics and may compromise both accuracy and confidentiality (Flores, 2005).

Interpretation for neuroregulation presents unique challenges requiring advance preparation (Flores, 2005). Many languages lack equivalent terms for technical concepts central to treatment. Working with interpreters to develop culturally meaningful explanations enhances understanding (Kleinman & Benson, 2006). Presession briefings should cover

key concepts that will be discussed, anticipated technical explanations that may need adaptation, cultural considerations relevant to the specific client, and clarification of any technical terms the interpreter may not have encountered (Flores, 2005). Postsession debriefing allows the interpreter to raise concerns or suggest communication improvements (Flores, 2005).

Plain language explanations remain essential regardless of language concordance (Kleinman & Benson, 2006). The complexity of neuroscience concepts requires careful translation into accessible terms without sacrificing accuracy (S. Sue et al., 2009). Visual aids, demonstrations, and hands-on experiences can transcend language barriers while respecting cultural learning styles (Resnicow et al., 1999). For example, showing a client what the neurofeedback display looks like and having them practice controlling it during the initial session provides experiential learning that may be more effective than lengthy verbal explanations (Griner & Smith, 2006). Practitioners should develop multiple ways of explaining core concepts, adjusting based on client feedback and comprehension checks (Flores, 2005).

Communication styles vary significantly across cultures with profound implications for information exchange, rapport building, and treatment planning (E. T. Hall, 1976). E. T. Hall's (1976) distinction between high-context and low-context communication remains highly relevant for neuroregulation practice. High-context communication, common in many Asian, African, and Latin American cultures, relies heavily on nonverbal cues, implicit understanding, and indirect expression (E. T. Hall, 1976). Low-context communication, typical of Northern European and Anglo-American cultures, emphasizes explicit verbal expression and direct communication (E. T. Hall, 1976). These differences manifest in multiple ways during treatment. Clients from high-context cultures may not directly express disagreement or discomfort with procedures, showing reluctance through nonverbal behavior or missed appointments rather than verbal refusal (E. T. Hall, 1976). They may find direct symptom questions uncomfortable, preferring to communicate distress through somatic complaints or metaphorical language (Kirmayer & Ryder, 2016). Practitioners accustomed to low-context communication may misinterpret indirect communication as resistance, poor motivation, or limited insight (E. T. Hall, 1976; Kleinman & Benson, 2006).

Nonverbal communication carries particular importance in cross-cultural interactions, with significant cultural variation in interpretation of gestures, facial expressions, and body language (E. T. Hall, 1976). Eye contact norms vary significantly across cultures; what signals engagement and honesty in one culture may indicate disrespect or inappropriate intimacy in another (E. T. Hall, 1976). Physical proximity preferences differ, affecting comfort with the close contact required for electrode placement (Eisenbarth et al., 2025). Touch boundaries during electrode placement require explicit discussion, particularly for procedures involving head or facial electrode placement (Eisenbarth et al., 2025). The meaning of silence varies culturally, with some cultures viewing silence as comfortable contemplation while others interpret it as awkward or hostile (E. T. Hall, 1976).

Written materials require comprehensive adaptation beyond simple translation (Resnicow et al., 1999). Effective cross-cultural health communication involves cultural adaptation of content, images, examples, and conceptual frameworks. Materials should reflect diverse populations in imagery, avoiding the common practice of only showing White, middle-class individuals using neurofeedback equipment (Resnicow et al., 1999). Examples and metaphors should be culturally relevant; for instance, comparing neurofeedback training to mastering a sport may resonate in some cultures while musical instrument mastery or traditional craft skill development may be more meaningful analogies in others (Bernal et al., 2009). Reading levels should be adjusted for varied health literacy (Flores, 2005). Cultural values should be considered in framing benefits and outcomes; individualistic cultures may respond to frames emphasizing personal achievement and self-improvement, while collectivistic cultures may find frames emphasizing family benefit and social harmony more compelling (E. T. Hall, 1976; S. Sue et al., 2009).

Treatment Adaptation Strategies

Cultural treatment adaptations in neuroregulation must balance fidelity to core mechanisms with responsiveness to cultural needs (Bernal et al., 2009). Meta-analyses consistently demonstrate that systematic, theory-driven adaptations produce superior outcomes compared to surface-level modifications or no adaptation (Griner & Smith, 2006; G. C. N. Hall et al., 2016). The key principle, established in the companion review (Sherlin, 2026), is that fundamental physiological mechanisms underlying neuroregulation remain constant across populations while implementation can and should be

adapted for cultural responsiveness (Sherlin et al., 2011).

The ecological validity model (Bernal et al., 2009) provides a comprehensive framework for adaptation across eight dimensions: language, persons, metaphors, content, concepts, goals, methods, and context. For neuroregulation specifically, this translates into protocol modifications that align procedures with cultural values while maintaining therapeutic integrity (Bernal et al., 2009). Core neuroregulation mechanisms (operant conditioning [Sherlin et al., 2011], neuroplasticity, autonomic regulation [Porges, 2011]) remain constant; what varies is how clients engage with these mechanisms (Griner & Smith, 2006). Adaptations might include feedback display selection considering cultural aesthetics and meanings, reward system modifications matching cultural values and motivational frameworks, and session structure flexibility accommodating cultural practices and time orientations (Bernal et al., 2009).

Colors carry different significance across cultures; red might signal danger or error in Western contexts but represents luck and prosperity in Chinese culture (Resnicow et al., 1999). Practitioners should assess color preferences and associations, avoiding assumptions based on their own cultural background (Bernal et al., 2009). Abstract displays may be preferable to potentially culturally-loaded imagery (S. Sue et al., 2009). Some clients may prefer geometric patterns while others respond better to nature scenes. The key is ensuring the feedback is reinforcing rather than aversive or confusing based on cultural interpretations (Griner & Smith, 2006).

Individual achievement emphasis might not motivate clients from collectivistic cultures as effectively as feedback emphasizing balance, harmony, or contribution to family wellbeing (E. T. Hall, 1976). For example, rather than framing successful training as “you achieved the goal,” reframing as “you’re developing skills that will help you be there for your family” may be more motivating (S. Sue et al., 2009). Points systems or competitive elements work well for some cultures but may be uncomfortable for others who prefer cooperative or noncompetitive structures (Bernal et al., 2009).

Session structure flexibility might include prayer time accommodation for religious clients before beginning technical procedures, brief social connection time for clients from relationship-oriented cultures who may find immediate task focus uncomfortable or disrespectful, modified session length

accommodating different time orientations (some cultures operate on more flexible time schedules and may find rigid 50-min sessions constraining), or flexible pacing that allows for family phone calls or other culturally important obligations that may arise during longer training sessions (E. T. Hall, 1976; S. Sue et al., 2009).

Peripheral Biofeedback Cultural Adaptations

Peripheral biofeedback, particularly heart rate variability (HRV) training, offers unique opportunities for cultural adaptation given its alignment with traditional mind-body practices in many cultures (Feldman et al., 2016). Feldman et al. (2016) demonstrated the effectiveness of culturally adapted cognitive-behavioral psychophysiological therapy (CBPT) with HRV biofeedback for Latino adults with comorbid asthma and panic disorder. Their systematic cultural adaptations included Spanish language sessions with bilingual therapists, active family involvement in treatment when desired by clients, culturally relevant examples and metaphors drawn from Latino cultural contexts, addressing cultural asthma beliefs such as *susto* (fright illness) and *nervios* (nerves), and incorporating culturally resonant stress concepts that made sense within participants’ explanatory frameworks (Feldman et al., 2016). Both adapted CBPT and music relaxation showed improvements, with CBPT demonstrating additional advantages for medication adherence (Feldman et al., 2016). Critically, Nelson et al. (2020) found that HRV and end-tidal CO₂ changes partially mediated treatment effects, indicating that the targeted physiological mechanisms operated similarly across cultural groups despite requiring culturally adapted engagement pathways to achieve optimal participation and outcomes.

Beltrán-Velasco et al. (2020) documented cultural differences in psychophysiological stress responses that have implications for biofeedback protocol development. Comparing Spanish and Colombian students revealed significant differences in both subjective and objective stress response patterns (Beltrán-Velasco et al., 2020). While fundamental stress mechanisms involving hypothalamic-pituitary-adrenal axis activation and autonomic nervous system responses are universal (McEwen, 2017), chronic cultural and environmental factors shape baseline patterns and reactivity, influencing optimal training parameters (Beltrán-Velasco et al., 2020). Practitioners should assess baseline stress response patterns carefully and consider whether cultural factors such as chronic discrimination stress or migration-related trauma may affect optimal training thresholds and reinforcement schedules

(Geronimus et al., 2006). Allostatic load from chronic stress exposure may result in blunted stress reactivity or chronically elevated sympathetic tone that requires adjusted training parameters (Geronimus et al., 2006; McEwen, 2017).

The mind-body focus of peripheral biofeedback may be particularly acceptable to populations familiar with traditional practices such as yoga, qigong, meditation, or contemplative prayer, providing natural bridges between cultural worldviews and biofeedback technology (Travis & Shear, 2010). For example, HRV biofeedback can be framed as using technology to enhance practices the client may already value, such as: "The breathing practice we'll do in biofeedback is similar to pranayama breathing you may have experienced in yoga, but we're using sensors to help you see exactly how your breath affects your heart rhythm and nervous system balance" (Feldman et al., 2016). This framing honors traditional knowledge while introducing technological enhancement, avoiding the implicit message that Western technology is superior to traditional practices (Hinton & Good, 2016).

These adaptations operationalize clinical-level responsiveness through protocol modifications and communication strategies, while simultaneously informing organizational-level decisions about telehealth infrastructure, interpreter services, and community partnership models. At the professional development level, successful peripheral biofeedback adaptations provide training exemplars for practitioners learning to balance treatment fidelity with cultural responsiveness.

Integration With Traditional Healing Practices

Integration with traditional healing practices requires careful navigation between respect for cultural traditions and professional boundary maintenance (Hinton & Good, 2016). Many clients, particularly from immigrant, Indigenous, or minority communities, may simultaneously engage multiple healing systems including traditional healers, religious healing, and biomedical care (Kirmayer & Ryder, 2016). Rather than viewing this as problematic "noncompliance," culturally responsive practitioners recognize parallel help-seeking as rational and culturally appropriate (Hinton & Good, 2016). Integration strategies include timing coordination with traditional healing ceremonies or practices, recognizing that clients may engage multiple healing systems simultaneously (La Roche & Christopher, 2009).

For example, a client may wish to schedule neurofeedback sessions around religious fasting periods, traditional healing ceremonies, or significant cultural events (S. Sue et al., 2009). Flexibility in scheduling demonstrates respect for the client's whole healing process rather than demanding that biomedical treatment take priority over cultural practices (Tervalon & Murray-García, 1998). Conceptual integration helps clients understand how neuroregulation relates to their existing healing framework (Hinton & Good, 2016). For example, HRV biofeedback might be explained as strengthening the same capacities developed through traditional breathing practices or prayer: "What we're doing with biofeedback is similar to what your traditional practices accomplish, but using technology to provide more immediate feedback about how your body is responding" (Feldman et al., 2016). This framing positions neuroregulation as complementary to rather than replacing traditional practices (La Roche & Christopher, 2009). For clients using traditional medicine or healing, discussion should focus on complementarity rather than contradiction (Kirmayer & Ryder, 2016).

Collaboration with traditional healers when appropriate and desired by clients, with proper consent and clear role delineation, can enhance treatment outcomes (Hinton & Good, 2016). Some clients may benefit from explicit communication between their neurofeedback practitioner and traditional healer, particularly when both are actively working with the client (La Roche & Christopher, 2009). Such collaboration requires cultural humility, respect for the traditional healer's expertise and role in the community, clear boundaries about respective scopes of practice, and client-centered focus ensuring that collaboration serves the client's goals rather than professional curiosity or research interests (Tervalon & Murray-García, 1998).

Managing Cultural Conflicts

Cultural conflicts in neuroregulation practice arise from differences in worldviews, values, communication styles, and treatment expectations (La Roche & Christopher, 2009). Rather than viewing these as obstacles, skilled practitioners recognize them as opportunities for deeper understanding and improved therapeutic alliance (Tervalon & Murray-García, 1998). When conflicts emerge, practitioners should first engage in critical self-reflection examining their own cultural assumptions contributing to the discord (La Roche & Christopher, 2009). What appears as "resistance" often reflects cultural misunderstanding rather than treatment rejection (Kleinman & Benson, 2006). For

example, clients missing appointments might reflect different time orientations, competing cultural obligations such as caring for extended family, or indirect communication of discomfort rather than low motivation or lack of commitment to treatment (E. T. Hall, 1976; S. Sue et al., 2009).

Direct but respectful discussion of cultural differences can resolve many conflicts (La Roche & Christopher, 2009). Practitioners might say: “I notice we might have different perspectives on family involvement in treatment. Can you help me understand what would work best for you within your cultural context?” This approach validates cultural differences while maintaining professional boundaries and opens dialogue rather than imposing solutions (Tervalon & Murray-García, 1998). When addressing potential cultural misunderstandings, practitioners should use curious, nonjudgmental language that invites explanation rather than defensive language that may sound accusatory or dismissive (Kleinman & Benson, 2006).

When irreconcilable conflicts arise, ethical practice may require referral to culturally matched providers or alternative treatment modalities that better fit the client’s cultural framework (G. C. N. Hall, 2001; S. Sue et al., 2009). This should be done transparently and respectfully: “I want to ensure you receive the most helpful treatment possible. Would you prefer working with a provider who shares your cultural background or speaks your language? I can help you find someone who might be a better fit” (Tervalon & Murray-García, 1998). This demonstrates commitment to the client’s well-being over practitioner ego or financial interest and models appropriate recognition of cultural competence limitations (S. Sue et al., 2009).

Organizational Implementation

Addressing Access Barriers

Structural and Economic Barriers to Equitable Access. Before addressing specific implementation strategies, it is essential to acknowledge the structural and economic constraints that fundamentally shape neuroregulation service delivery. The field operates predominantly through private practice and clinic-based models that require substantial capital investment and ongoing operational costs. Establishing neurofeedback services requires acquisition of amplifier and signal acquisition hardware, feedback software, and assessment instrumentation, along with recurring expenditures for electrodes, conductive media, and software licensing (Schwartz & Andrasik, 2017).

Systematic documentation of start-up and operating costs for neuroregulation services is not available in the peer-reviewed literature, which itself represents a gap in the field’s capacity to evaluate access barriers, but the categories of required investment place clinic establishment beyond the reach of many practitioners and community-based organizations without external capital. These equipment costs create significant barriers to entry, particularly for practitioners serving low-income communities or working in under-resourced settings where client fees must remain affordable to ensure access.

Training pathways compound these financial barriers. Board certification in neurofeedback requires 36 hours of didactic education; a course in neuroanatomy, neurophysiology, or physiological psychology; 25 contact hours of mentored practical training encompassing 100 patient sessions and 10 case study presentations; and a three-hour certification examination, in addition to \$450 in application and examination fees (Biofeedback Certification International Alliance, n.d.). Because didactic and mentoring costs are set independently by accredited training providers rather than by the certifying body, total expenditure varies considerably, and the cumulative time and tuition commitment represents a meaningful barrier for practitioners in lower-resourced settings. For practitioners from underrepresented backgrounds or those serving underserved communities, these training costs may be prohibitive without scholarship support or institutional funding. The resulting practitioner workforce concentrates in affluent areas where private pay rates can offset training investments and equipment costs, creating a service distribution pattern that mirrors and perpetuates existing healthcare disparities (Saadi et al., 2017).

Insurance reimbursement structures further entrench geographic and socioeconomic disparities. Limited coverage for neurofeedback services (as discussed in the companion review; Sherlin, 2026) means practitioners must either maintain unsustainably low fees or serve primarily private-pay clients. Medicare and Medicaid reimbursement rates, where coverage exists at all, typically fail to cover equipment costs and specialized training investments, making it economically unfeasible for practitioners to serve primarily publicly insured populations. This reimbursement landscape effectively restricts services to those who can afford out-of-pocket payment, concentrating practices in higher-income, predominantly White communities where sufficient private-pay client volumes can sustain operations (Betancourt et al., 2003).

These structural constraints have profound implications for both clinical practice and research. Individual practitioners and small-scale researchers at under-resourced institutions have limited capacity to recruit culturally diverse samples, regardless of cultural competence or commitment to equity. The underrepresentation of minoritized populations in neuroregulation research reflects these systemic access barriers more than methodological choices or population differences in treatment suitability. Calls for increased cultural representation in neuroregulation research must therefore be accompanied by parallel investments in delivery infrastructure, grant funding explicitly supporting community-based service delivery, and reimbursement reform. Without such systemic changes, expectations placed on individual clinicians and researchers risk being aspirational rather than actionable (López et al., 2012; Metzl & Hansen, 2014).

Recognizing these structural realities, the following sections address both immediate strategies individual practitioners and organizations can implement within current constraints, and longer-term systemic reforms necessary for genuinely equitable access. While individual-level cultural adaptations remain important, they are insufficient without concurrent attention to the economic and structural factors that determine who can access services at all.

Financial barriers represent the most significant obstacle to neuroregulation access for diverse populations, with treatment protocols typically requiring multiple sessions over an extended course (Fleischman et al., 2022). As documented in the companion review (Sherlin, 2026), insurance coverage limitations create a two-tiered system where neuroregulation is available primarily to those who can afford substantial out-of-pocket payments (Saadi et al., 2017). Organizations can implement various strategies to address these disparities (Betancourt et al., 2003).

Sliding scale fee structures based on income can make services accessible while maintaining organizational sustainability (Alegria et al., 2016). Effective sliding scales require transparent policies communicated clearly in multiple languages, simplified documentation avoiding bureaucratic barriers that may deter potential clients, regular review ensuring both accessibility and organizational viability, and designated slots protecting both full-fee and reduced-fee client availability (López et al., 2012). Some organizations reserve a defined

proportion of appointments for sliding scale clients, with full-fee services subsidizing reduced-fee care (Betancourt et al., 2003). Clear policies about sliding scale eligibility, required documentation, and fee determination prevent both real and perceived inequities (Alegria et al., 2016).

Grant funding specifically targeting health equity can support services for underserved populations (Fleischman et al., 2022). The Neurofeedback Advocacy Project model documented by Fleischman et al. (2022) demonstrates successful systematic expansion through equipment loans and training support to organizations serving Medicaid populations. Their work with over 300 clients across 20+ community agency sites showed that when access barriers are addressed, diverse clients with complex presentations achieve substantial improvements (Fleischman et al., 2022). The reported no-show rate of 2% suggests high engagement when services are accessible and responsive to community needs (Fleischman et al., 2022).

Geographic barriers disproportionately affect rural and low-income urban communities lacking specialty mental health services (Saadi et al., 2017). Creative service delivery approaches can extend reach through mobile services using equipped vans or portable equipment that can serve multiple community sites, satellite clinics in community locations such as schools, community centers, or religious institutions reducing transportation barriers, telehealth options overcoming distance limitations while requiring attention to digital access issues, and partnerships with existing community services providing space and infrastructure, leveraging trusted community organizations to reduce barriers and increase acceptability (Betancourt et al., 2003; López et al., 2012).

Temporal barriers affect working-class populations with inflexible schedules and multiple jobs (Alegria et al., 2016). Many low-wage workers cannot take time off during standard business hours without losing income or jeopardizing employment (López et al., 2012). Solutions include extended hours accommodating work schedules, such as early morning or evening appointments; weekend availability despite practitioner inconvenience, recognizing that families working multiple jobs may only have weekend time for healthcare; intensive treatment models condensing protocols into fewer but longer sessions when appropriate for specific protocols; and flexible cancellation policies recognizing life complexities of poverty such as

transportation unreliability, child care disruptions, or crisis situations that may prevent attendance without necessarily indicating low motivation (Alegría et al., 2016; Betancourt et al., 2003).

Alternative Service Delivery Models

Traditional clinic-based delivery may be inaccessible or culturally unacceptable to many populations (Betancourt et al., 2003). Alternative models can reduce barriers and increase engagement without compromising treatment quality, as the fundamental neurophysiological mechanisms remain constant regardless of service delivery location (Sherlin, 2026; Sherlin et al., 2011).

Community-based service delivery brings neuroregulation to familiar, trusted settings (Fleischman et al., 2022). Successful implementations include school-based programs providing neurofeedback for students with ADHD and learning difficulties, integrating treatment into the school day and eliminating transportation barriers for families (Gruzelier, 2014). Community centers offer nonstigmatizing locations particularly important for populations experiencing mental health stigma (López et al., 2012). Services provided in community centers may be perceived as "skill-building" or "wellness programs" rather than mental health treatment, reducing shame or reluctance to participate (Resnicow et al., 1999). Religious institutions provide trusted spaces for communities with medical system mistrust, though requiring careful navigation of professional boundaries and clear distinction between religious and clinical services (Betancourt et al., 2003).

Each setting requires systematic attention to privacy protection in shared spaces, ensuring confidential conversations cannot be overheard and that equipment setup maintains client privacy (Betancourt et al., 2003). Equipment security and maintenance in community settings may require different approaches than clinic-based care, including portable equipment that can be secured when not in use (Fleischman et al., 2022). Professional boundary maintenance in community settings where practitioners may encounter clients in other roles requires clear policies and ongoing supervision (S. Sue et al., 2009). Infection control and safety protocols must meet regulatory requirements regardless of setting, requiring systematic procedures and documentation (Betancourt et al., 2003). Documentation must meet regulatory requirements while being adaptable to various settings, potentially using secure electronic

health records accessible from multiple locations (Alegría et al., 2016).

Telehealth and Remote Monitoring

Telehealth and remote monitoring offer unprecedented access potential while presenting unique cultural challenges that require thoughtful navigation. Home-based neurofeedback systems can provide frequent training without transportation barriers, particularly benefiting rural communities and those with limited mobility. Feasibility evidence is emerging: an open-label study of a home-based SMR tele-neurofeedback device found significant pre–post improvement in self-reported sleep among 37 adults, with participants who demonstrated successful SMR learning showing greater gains than nonlearners (Krepel et al., 2022), and a follow-up analysis of the same sample reported improvements on several cognitive measures for learners relative to nonlearners (Kolken et al., 2023). These studies were uncontrolled and did not compare remote to clinic-based delivery, so equivalence between the two has not yet been established and remains an open empirical question.

However, telehealth requires careful consideration of multiple cultural and practical factors (Betancourt et al., 2003). Digital literacy varies significantly across populations, with older adults, recent immigrants, and low-income individuals potentially experiencing technology barriers (Alegría et al., 2016). While younger generations may be comfortable with smartphones and video calls, the specific technology platforms used for telehealth and the integration with neurofeedback equipment may present unfamiliar challenges. Assessment should explore not just technology access but comfort and skill level, with additional training provided as needed (Flores, 2005). Some clients may benefit from initial in-person sessions to establish rapport and learn the technology before transitioning to remote sessions (S. Sue et al., 2009).

Internet access reliability remains uneven, with rural areas and low-income communities disproportionately affected by limited broadband access (Saadi et al., 2017). Practitioners should not assume reliable high-speed internet availability and should have contingency plans for connection disruptions that don't penalize clients for infrastructure limitations beyond their control (Betancourt et al., 2003). Mobile data plans may have insufficient bandwidth or monthly data limits that make regular video sessions prohibitively expensive for low-income clients (López et al., 2012).

Privacy concerns may be heightened in multigenerational households or shared living spaces common in many collectivistic cultures (E. T. Hall, 1976). Clients may not have private space for telehealth sessions, raising concerns about confidentiality and about family members seeing or hearing clinical content (S. Sue et al., 2009). Discussion should address where clients will participate in sessions, what privacy level is possible, whether headphones would help maintain privacy, and how to handle interruptions or family members entering the space during sessions (Betancourt et al., 2003). For some clients, clinic-based or community-based settings may offer more privacy than their home environment (Alegría et al., 2016).

Language barriers may be compounded in telehealth settings where technical troubleshooting requires English proficiency beyond what's needed for clinical content (Flores, 2005). Instructions for connecting to platforms, adjusting settings, or addressing technical problems often assume English fluency. Organizations should provide technical support in clients' primary languages and should use technology platforms with multilingual interfaces when possible (Flores, 2005). Written instructions with screenshots can help but should be available in multiple languages and at appropriate literacy levels (Resnicow et al., 1999).

Cultural acceptability of telehealth is likely to vary, with some populations viewing technology-mediated care as inferior or impersonal and others appreciating reduced stigma and increased convenience. This variation has not been systematically studied in neuroregulation and warrants investigation. Older adults from cultures that highly value face-to-face interaction may find telehealth unsatisfying or disrespectful, missing the relational aspects of in-person care (E. T. Hall, 1976). Practitioners should explicitly discuss telehealth preferences and concerns during initial assessment, offer hybrid models combining in-person and remote sessions when feasible to build relationship while maintaining access, provide culturally appropriate technical support ideally in the client's primary language, and establish clear protocols for managing technical difficulties and emergency situations in ways that maintain therapeutic alliance and client safety (S. Sue et al., 2009). The loss of in-person nonverbal communication may be particularly challenging for high-context communicators who rely heavily on implicit understanding and relational cues that are diminished in video interactions (E. T. Hall, 1976).

Culturally Responsive Informed Consent

Informed consent represents both ethical imperative and legal requirement, yet standard consent processes may not ensure genuine understanding across diverse populations (Flores, 2005). Effective cross-cultural consent requires moving beyond signature collection to ensure authentic comprehension and voluntary agreement (Kleinman & Benson, 2006). Consent materials written above a client's reading level create comprehension barriers for many clients regardless of intelligence or capability.

Language considerations extend beyond translation to include ensuring materials are written at appropriate literacy levels (typically sixth to eighth grade), using common words and short sentences that enhance clarity without condescension (Flores, 2005; Resnicow et al., 1999). Providing verbal explanation opportunities allows practitioners to gauge understanding and adjust explanations based on client questions and responses (Kleinman & Benson, 2006). Using teach-back methods to verify understanding by asking clients to explain key information in their own words identifies comprehension gaps without sounding like a test (Flores, 2005). Offering time for questions without pressure, explicitly stating that questions are welcome and expected, normalizes question-asking for clients from cultures where questioning authority figures may be uncomfortable (E. T. Hall, 1976; S. Sue et al., 2009).

Professional interpreters should be used for consent discussions when language barriers exist (Flores, 2005). Family members should not serve as interpreters for consent discussions as they may have conflicts of interest, may not understand technical concepts well enough to interpret accurately, and may inadvertently influence the client's decision based on family preferences rather than individual autonomy (Flores, 2005). Practitioners must remember that using family as interpreters may compromise confidentiality by revealing information the client might not want shared with family members (Betancourt et al., 2003).

Cultural variations in decision-making styles significantly affect consent processes (E. T. Hall, 1976). Individualistic cultures emphasize autonomous decision-making with the individual patient as the primary decision-maker who may consult others but ultimately decides independently (E. T. Hall, 1976). Collectivistic cultures may prioritize family or community input, with health

decisions viewed as affecting and involving the whole family system rather than just the individual (S. Sue et al., 2009). Some cultures have specific family members (such as eldest male, family matriarch, or spouse) who are expected to make or at least approve healthcare decisions (Kleinman & Benson, 2006).

Practitioners should respect diverse decision-making preferences while ensuring the identified client provides informed consent (Tervalon & Murray-García, 1998). Some clients may wish to include family members in consent discussions or may need time to consult with family before committing to treatment (S. Sue et al., 2009). These preferences should be accommodated while maintaining clarity about who is the client and ensuring their autonomous consent, even within collective decision-making frameworks (Kleinman & Benson, 2006). Questions might include: "Would you like to have family members present for this discussion?" "Are there people whose input is important to you in making this decision?" "After hearing their thoughts, how will you make your final decision?" (E. T. Hall, 1976; S. Sue et al., 2009).

Power dynamics inherent in healthcare relationships may be amplified across cultural differences, particularly for populations with medical mistrust or immigration concerns (Bazargan et al., 2021). Creating genuine voluntariness requires multiple strategies. Building trust over time rather than rushing consent in initial encounters allows relationship development that supports truly voluntary consent (Tervalon & Murray-García, 1998). Providing multiple opportunities to ask questions throughout treatment, not just at initial consent, recognizes that understanding evolves over time and questions may emerge after initial sessions (Kleinman & Benson, 2006). Explicitly stating that declining treatment will not affect other services addresses concerns that refusal may result in negative consequences, particularly important for clients in community health centers where multiple services may be provided (Betancourt et al., 2003). Recognizing that some populations may be reluctant to refuse or question authority figures suggests using extra diligence to ensure understanding and voluntary agreement rather than passive compliance (Bazargan et al., 2021; E. T. Hall, 1976).

Consent should be conceptualized as ongoing process rather than a one-time event (Tervalon & Murray-García, 1998). Before beginning neurofeedback, clients may not fully understand what the experience will be like. Regular

opportunities to reassess understanding and willingness to continue treatment honor ongoing autonomy and allow clients to ask questions as their understanding deepens (S. Sue et al., 2009). Check-ins might include: "Now that you've experienced a few sessions, do you have any questions about what we're doing?" "Does this still feel like the right approach for you?" "Is there anything about the process you'd like to discuss or change?" (Kleinman & Benson, 2006; Tervalon & Murray-García, 1998).

Quality Monitoring and Improvement

Systematic quality monitoring with explicit attention to equity is essential for identifying and addressing disparities in neuroregulation services (López et al., 2012). Organizations must move beyond aggregate outcome reporting to disaggregated analysis revealing differential patterns across demographic groups (Betancourt et al., 2003). Without disaggregated analysis, disparities remain invisible in averaged data that may show overall positive outcomes while masking poorer outcomes for specific populations (López et al., 2012).

Engagement metrics should be tracked by race, ethnicity, language, socioeconomic status, and other relevant factors (Betancourt et al., 2003). Key indicators include referral sources and pathways revealing which communities have access to referrals and which remain unaware of services, initial appointment attendance rates indicating whether services feel accessible and acceptable to diverse populations, session attendance patterns and no-show rates that may reveal practical barriers like transportation or scheduling conflicts, treatment completion rates and average doses received across demographic groups, and reasons for premature termination that may highlight cultural barriers or conflicts requiring attention (Alegría et al., 2016; López et al., 2012).

Disparities in these metrics signal barriers requiring targeted intervention (López et al., 2012). For example, if initial appointment attendance is significantly lower for Spanish-speaking clients, this might indicate need for bilingual outreach materials, Spanish-speaking front desk staff, or explicit communication about interpreter availability (Flores, 2005). If African American clients have higher no-show rates, this might reflect mistrust requiring additional rapport-building time, practical barriers like transportation, or scheduling conflicts with inflexible work schedules (Bazargan et al., 2021; Betancourt et al., 2003).

Clinical outcomes must be analyzed for equity, not just effectiveness (López et al., 2012). While physiological mechanisms operate universally, as established in the companion review (Sherlin, 2026), outcomes may vary based on engagement quality, therapeutic alliance strength, and treatment adaptation adequacy (Griner & Smith, 2006). Organizations should examine whether all demographic groups achieve comparable outcomes, identify factors associated with differential outcomes such as number of sessions attended, language concordance, practitioner cultural competence, and implement targeted improvements addressing identified disparities (Betancourt et al., 2003; López et al., 2012). If outcomes for specific populations lag behind majority population outcomes despite similar attendance, this suggests need for enhanced cultural adaptation or practitioner training rather than assuming differential treatment response (Feldman et al., 2016; Fleischman et al., 2022).

Client satisfaction assessment requires culturally appropriate methods capturing diverse populations' experiences (Resnicow et al., 1999). Standard satisfaction surveys may miss important cultural factors affecting treatment experience (S. Sue et al., 2009). Survey questions developed for majority populations may not capture the concerns or values of minority clients (Kleinman & Benson, 2006). For example, questions about individual symptom reduction may miss relational outcomes more valued in collectivistic cultures (E. T. Hall, 1976). Culturally responsive assessment includes focus groups conducted in clients' primary languages allowing for discussion and elaboration beyond survey responses, community advisory boards providing ongoing feedback about services from community perspective, specific questions about cultural responsiveness such as "Did providers respect your cultural beliefs?" or "Was interpreter assistance satisfactory?", and anonymous feedback mechanisms recognizing that clients from hierarchical cultures may be reluctant to provide critical feedback directly to providers (Betancourt et al., 2003; E. T. Hall, 1976; S. Sue et al., 2009).

Continuous quality improvement must prioritize equity alongside efficiency and effectiveness (López et al., 2012). When disparities are identified, organizations should implement targeted interventions such as additional staff training in cultural competence focusing on identified gaps, partnership development with community organizations serving underrepresented populations, policy modifications removing identified barriers like inflexible scheduling or burdensome paperwork, and

resource reallocation to underserved populations potentially including dedicated slots or funding for clients facing financial barriers (Alegría et al., 2016; Betancourt et al., 2003). Improvement cycles should be continuous with regular review of equity metrics and adjustment of interventions based on effectiveness (López et al., 2012).

Professional Development and Training

Individual Practitioner Development

Developing cultural competence in neuroregulation requires lifelong commitment to learning and growth (Tervalon & Murray-García, 1998). Based on the cultural humility framework detailed in the companion article (Sherlin, 2026), practitioners must recognize that cultural learning is never complete and requires ongoing self-reflection and development. Individual practitioners should begin with comprehensive self-assessment examining their own cultural identity and its influence on practice including values, beliefs, and assumptions they bring to clinical work, implicit biases affecting clinical judgment and decision-making that may operate outside conscious awareness, current competence levels and knowledge gaps about specific cultural groups, and motivation for cultural competence development, recognizing that genuine cultural desire sustains ongoing efforts (Campinha-Bacote, 2002; Tervalon & Murray-García, 1998).

Formal cultural competency training should be viewed as essential rather than optional continuing education (S. Sue et al., 2009). No established cultural competence training standards currently exist specifically for neuroregulation, and the field would benefit from developing them. Training should address both general cultural competence applicable across clinical settings and neuroregulation-specific considerations including how culture influences technology perception and acceptance, strategies for explaining neuroscience concepts across educational and cultural contexts without condescension or excessive jargon, protocol adaptation guidelines maintaining therapeutic integrity while enhancing cultural responsiveness, therapeutic alliance building across cultural differences, and recognition of one's own cultural competence limitations requiring consultation or referral (Betancourt et al., 2003; S. Sue et al., 2009).

Experiential learning through supervised practice with diverse populations accelerates competence development beyond didactic training alone (G. C. N. Hall, 2001; D. W. Sue et al., 1992). Meaningful cross-cultural clinical experiences, accompanied by

reflection and supervision, may accelerate competence development beyond didactic training alone. Practitioners should actively seek opportunities working with diverse clients, recognizing that competence requires direct experience (G. C. N. Hall, 2001). Participation in community outreach and education introduces practitioners to community contexts and builds relationships that facilitate future clinical work (Betancourt et al., 2003). Engagement with cultural communities through attendance at cultural events, reading, or relationship-building helps practitioners learn about diverse health and healing perspectives outside clinical contexts (S. Sue et al., 2009). Pursuit of supervision or consultation with culturally experienced colleagues provides guidance when working with unfamiliar populations or addressing cultural challenges (Tervalon & Murray-García, 1998).

Cultural humility practices sustain long-term development and prevent competence stagnation (Tervalon & Murray-García, 1998). This involves acknowledging mistakes as learning opportunities rather than defensively denying or minimizing cultural errors, actively seeking client feedback about cultural responsiveness through direct questions and receptiveness to indirect feedback, remaining open to having assumptions challenged and recognizing that clients are experts on their own cultural experiences, and recognizing cultural learning as ongoing rather than achievable endpoint, maintaining curiosity and humility across career span (Tervalon & Murray-García, 1998; S. Sue et al., 2009).

Organizational Training Strategies

Organizations committed to cultural responsiveness must implement comprehensive training approaches addressing all staff levels and functions (Betancourt et al., 2003). Leadership commitment demonstrated through resource allocation, policy support, and personal participation sets organizational tone for cultural competence importance (López et al., 2012). When leadership treats cultural competence as optional or delegates it entirely without participating, staff receive the message that it is not truly a priority despite rhetoric to the contrary (Betancourt et al., 2003).

Comprehensive staff training recognizes that all organizational members influence client experience, not just clinical providers (Betancourt et al., 2003). Front desk staff require training in culturally responsive customer service, appropriate interpreter utilization, and recognition of language barriers or

cultural confusion requiring assistance (Flores, 2005). Their interactions are often clients' first organizational contact and set the tone for whether the environment feels welcoming and responsive to diversity (Resnicow et al., 1999). Technical staff conducting assessments need skills for culturally appropriate interaction during procedures like electrode placement that may involve cultural or religious sensitivities (Eisenbarth et al., 2025). Clinicians require depth training in assessment, formulation, and treatment adaptation as described throughout this article (S. Sue et al., 2009). Administrators need understanding of structural barriers and equity-focused policy development to ensure organizational practices support rather than hinder cultural responsiveness (López et al., 2012; Metzl & Hansen, 2014).

Training modalities should be diverse, sustained, and progressively challenging (Betancourt et al., 2003). One-time workshops are unlikely to produce lasting change compared to sustained, multimodal approaches (Betancourt et al., 2003). Effective organizational training includes initial intensive training establishing foundation knowledge through workshops, webinars, or courses; monthly ongoing education integrated into staff meetings and clinical supervision rather than relegated to separate diversity training; case consultation incorporating cultural perspectives routinely rather than only when "cultural issues" are identified; mentorship programs supporting skill development through pairing less experienced practitioners with more culturally competent colleagues; and immersion experiences in community settings such as participation in cultural events, partnerships with community organizations, or community advisory board meetings (Betancourt et al., 2003; S. Sue et al., 2009).

Simulation and standardized patient encounters allow skill development in controlled settings before client application (S. Sue et al., 2009). Scenarios should reflect common cultural challenges such as working effectively with professional interpreters, managing family involvement expectations that differ from practitioner's assumptions, adapting protocols for religious considerations such as scheduling around prayer times or Ramadan fasting, and responding therapeutically to clients' discrimination experiences in healthcare without becoming defensive (Bazargan et al., 2021; Betancourt et al., 2003). Debriefing after simulations helps practitioners identify areas for improvement and reinforces effective strategies (S. Sue et al., 2009).

Supervision and Mentorship

Supervision plays a crucial role in developing cultural competence, requiring supervisors skilled in both neuroregulation techniques and cultural responsiveness (Tervalon & Murray-García, 1998). Effective supervision creates safe spaces for exploring cultural dynamics including countertransference reactions, mistakes and uncertainties, and developmental growth edges (S. Sue et al., 2009). Supervisors must create environments where supervisees can acknowledge cultural limitations, errors, or confusion without fear of judgment or negative evaluation (Tervalon & Murray-García, 1998).

Supervisory discussions should routinely integrate cultural considerations rather than treating culture as special topic addressed only when obvious cultural differences exist (S. Sue et al., 2009). Questions guiding cultural exploration include: “What cultural factors might influence engagement or outcomes?” “How might the client’s worldview affect symptom understanding or treatment preferences?” “What cultural strengths or resources can be leveraged?” “Are there unaddressed cultural barriers or misunderstandings?” “How might my own cultural background be affecting this clinical relationship?” (Kleinman & Benson, 2006; Tervalon & Murray-García, 1998).

Direct observation with cultural focus provides invaluable feedback unavailable through self-report alone (S. Sue et al., 2009). Supervisors observing sessions should attend specifically to verbal and nonverbal communication patterns including pace, directness, language complexity, and nonverbal communication appropriateness; power dynamics and their management, including how the practitioner navigates authority relationships and decision-making; missed cultural cues or opportunities such as moments when cultural factors might have been explored or addressed; and successful cultural adaptations worth reinforcing and potentially sharing with other supervisees (E. T. Hall, 1976; Tervalon & Murray-García, 1998). Feedback should be specific, balanced with recognition of effective cultural responses alongside areas for growth, and growth-oriented focusing on development rather than criticism (S. Sue et al., 2009).

Mentorship programs accelerate development particularly for practitioners from underrepresented backgrounds navigating predominantly White professional spaces (G. C. N. Hall, 2001). Effective mentorship addresses not just clinical skills but also

professional navigation including understanding unwritten rules and norms, network development including introductions to key colleagues and organizations, and identity integration supporting practitioners in maintaining cultural identity while succeeding professionally (G. C. N. Hall, 2001; D. W. Sue et al., 1992). Organizations should actively develop mentorship programs pairing senior practitioners with those entering the field, with attention to both cross-cultural and culturally matched mentoring relationships (S. Sue et al., 2009).

Integration of Professional Standards

The neuroregulation field must systematically integrate cultural competence into professional standards, certification requirements, and ethical guidelines (S. Sue et al., 2009). Currently, major certification bodies including BCIA (Biofeedback Certification International Alliance) lack explicit cultural competence requirements, representing a significant gap requiring immediate attention (Betancourt et al., 2003). Certification standards should include demonstrated cultural competence through required training hours in cultural factors including both general cultural competence and neuroregulation-specific applications; examination questions assessing cultural knowledge including recognition of cultural factors affecting assessment, treatment planning, and outcomes; practical demonstration via case presentation or simulation showing application of cultural competence in clinical scenarios; and continuing education requirements for credential maintenance recognizing that cultural competence requires ongoing development rather than one-time achievement (Betancourt et al., 2003; S. Sue et al., 2009).

Accreditation standards for training programs should mandate cultural competence integration throughout curricula rather than segregated diversity courses (G. C. N. Hall, 2001). This includes diverse case examples and research throughout training rather than only in designated cultural sections, faculty modeling cultural competence in their teaching and supervision, supervised practice with diverse populations as graduation requirement, and explicit skill assessment ensuring graduates meet competency standards before entering independent practice (Betancourt et al., 2003; S. Sue et al., 2009).

Ethical guidelines must explicitly address cultural responsiveness as ethical imperative rather than optional enhancement (Tervalon & Murray-García, 1998). This includes recognizing cultural

incompetence as potential harm source when practitioners work beyond their competence level with specific populations, establishing interpreter use standards requiring professional interpreters when language barriers exist, addressing informed consent across linguistic and cultural differences to ensure genuine understanding and voluntary participation, and guiding appropriate referral when cultural competence limits are reached (Flores, 2005; S. Sue et al., 2009). The International Society for Neuroregulation & Research (ISNR) and other professional organizations should develop specific ethical guidance for cultural responsiveness in neuroregulation practice (Betancourt et al., 2003).

Implementation Challenges and Solutions

Common Implementation Barriers

Organizations implementing culturally responsive neuroregulation face predictable challenges requiring proactive planning and creative problem-solving (Betancourt et al., 2003). Leadership commitment is widely regarded as central to successful implementation, demonstrated through resource allocation, personal participation in training, and integration of cultural competence into performance evaluation systems. Anticipating these challenges and securing leadership buy-in allows organizations to develop strategies rather than being deterred when difficulties arise.

Resource limitations consistently emerge as a primary implementation barrier (Alegría et al., 2016). Providing comprehensive cultural adaptations requires financial investment in interpreter services, extended appointments to accommodate language barriers and trust-building, staff training including release time and program costs, community engagement and outreach requiring dedicated staff time, and translated materials and culturally appropriate resources (Betancourt et al., 2003; Flores, 2005). Small practices or community organizations serving low-income populations face particular challenges funding these essential services (López et al., 2012). Solutions include graduated implementation prioritizing highest-impact changes rather than attempting comprehensive implementation simultaneously (Betancourt et al., 2003). Begin with core changes like professional interpreter access and basic staff training before pursuing more resource-intensive adaptations (Alegría et al., 2016). Strategic resource allocation including grant seeking specifically for health equity initiatives, with increasing funding available for disparity reduction (López et al., 2012). Creative partnerships sharing costs with community

organizations, academic institutions, or other providers serving similar populations (Betancourt et al., 2003). Documentation of return on investment through improved retention and outcomes helps justify initial resource allocation by demonstrating long-term organizational benefit (Fleischman et al., 2022).

Staff resistance emerges from various sources requiring different responses (Betancourt et al., 2003). Fear of making cultural mistakes creates anxiety that may manifest as avoidance rather than engagement (Tervalon & Murray-García, 1998). Practitioners worry about offending clients or being accused of bias. Response includes creating psychological safety where mistakes are learning opportunities, providing education about cultural humility approach that acknowledges mistakes as inevitable, and offering support rather than punishment when cultural missteps occur (S. Sue et al., 2009; Tervalon & Murray-García, 1998).

Belief in "colorblind" approaches reflects misunderstanding of equity versus equality (López et al., 2012). Some staff believe that treating everyone the same represents fairness and that attention to cultural differences is itself discriminatory (Betancourt et al., 2003). Response includes education distinguishing equity (providing what each person needs) from equality (providing the same thing to everyone), presentation of disparity data showing that current approaches don't work equally well for all populations, and framing cultural responsiveness as clinical excellence rather than political correctness (López et al., 2012; Metzl & Hansen, 2014).

Resentment about additional requirements may emerge particularly when staff feel overburdened by existing demands (Betancourt et al., 2003). Response includes acknowledging legitimate workload concerns rather than dismissing them, demonstrating leadership commitment through resource allocation rather than only adding expectations, and showing how cultural responsiveness improves clinical effectiveness rather than only creating additional work (S. Sue et al., 2009). Focusing on systemic rather than individual change emphasizes that cultural responsiveness requires organizational support, not just individual practitioner effort (López et al., 2012).

Time constraints challenge implementation given that comprehensive cultural assessment, interpreter use, and trust-building require additional time (Flores, 2005). Typical 50-min sessions may be

insufficient for culturally responsive care, particularly initial sessions establishing rapport across cultural differences or ongoing sessions requiring interpretation (Betancourt et al., 2003). Solutions focus on integration rather than addition by weaving cultural considerations throughout existing processes rather than adding separate procedures (S. Sue et al., 2009). Efficiency improvements through staff training that increases competence and confidence, streamlining documentation, and systematic processes reduce time wasted on inefficient approaches (Betancourt et al., 2003). Recognition of long-term benefits including better engagement and retention ultimately saves time compared to repeatedly starting over with clients who drop out due to cultural barriers (Fleischman et al., 2022). Extended initial sessions allowing adequate time for culturally responsive assessment and rapport-building may prevent later problems requiring more time to address (Alegría et al., 2016; Tervalon & Murray-García, 1998).

Sustaining Cultural Responsiveness

Initial implementation enthusiasm often wanes without systematic sustainment strategies (Betancourt et al., 2003). Long-term success requires embedding cultural responsiveness into organizational DNA rather than depending on individual champions who may leave or experience burnout (López et al., 2012).

Policy integration ensures cultural responsiveness survives staff transitions and leadership changes (Betancourt et al., 2003). Written policies should address interpreter service standards including when interpreters must be used and quality expectations, cultural assessment requirements specifying minimum assessment components for all clients, accommodation procedures for religious or cultural needs, training expectations for all staff levels with specific requirements, and equity monitoring processes with defined metrics and review frequency (Betancourt et al., 2003; Flores, 2005). Policies should be reviewed and updated regularly, include staff input from diverse perspectives, be accessible to all staff in writing and training, and guide rather than replace clinical judgment (S. Sue et al., 2009).

Budget allocation demonstrates genuine organizational commitment beyond rhetoric (López et al., 2012). Cultural responsiveness requires dedicated budget lines rather than depending on surplus funds that may disappear during financial challenges (Alegría et al., 2016). Budget should include interpreter services as standard operating

cost, cultural competence training for all staff, community engagement and partnership development, translated materials and resources, equity analyst or quality improvement staff time, and contingency funding for unanticipated cultural needs (Betancourt et al., 2003). When cultural responsiveness has dedicated funding, it signals organizational priority and ensures sustainability (López et al., 2012).

Accountability mechanisms maintain focus through ongoing monitoring and feedback (Betancourt et al., 2003). Regular equity report cards presented to leadership and staff show progress and identify areas needing attention (López et al., 2012). Performance evaluations incorporating cultural competence for all staff, not just clinicians, hold individuals accountable (S. Sue et al., 2009). Community advisory board oversight provides external perspective and accountability to communities served (Betancourt et al., 2003). These mechanisms prevent cultural responsiveness from becoming merely aspirational without genuine implementation (López et al., 2012).

Continuous learning prevents stagnation through ongoing education maintaining skill development beyond initial training, community engagement through sustained partnerships and participation in cultural events, exploration of emerging population needs as communities evolve and new populations arrive, and incorporation of new research findings into practice as evidence base develops (S. Sue et al., 2009; Betancourt et al., 2003). Organizations should create learning cultures where cultural competence is everyone's ongoing responsibility rather than one-time training requirement (Tervalon & Murray-García, 1998).

Reorganized Future Directions and System Change Needs

Research Priorities for the Field

The paucity of research examining cultural factors in neuroregulation represents a critical knowledge gap requiring immediate attention, as documented in the companion review (Sherlin, 2026). Most urgently, the field needs development and validation of culturally adapted protocols through rigorous controlled trials, with findings disseminated via open-source protocol repositories ensuring practitioner scalability and adaptation for local contexts (Griner & Smith, 2006). While we have extensive evidence for standard neurofeedback protocols, we lack systematic research examining how these protocols might be optimally adapted for different cultural

populations while maintaining therapeutic efficacy (G. C. N. Hall et al., 2016). This research should investigate not only surface-level modifications like feedback displays and reward systems, but also deeper structural adaptations that might enhance engagement and outcomes for specific cultural groups (Bernal et al., 2009).

Equally important is investigation of optimal training parameters across diverse populations, with training curricula and assessment instruments made freely available through professional development consortia to facilitate immediate practitioner application (Feldman et al., 2016). Current protocols have been developed and tested primarily with Western, educated populations, yet optimal session frequency, duration, and total treatment length may vary based on cultural factors affecting engagement, practice patterns, and generalization of skills (Beltrán-Velasco et al., 2020). For instance, collectivistic cultures might benefit from different reinforcement schedules or session structures that accommodate family involvement (E. T. Hall, 1976).

Implementation research examining service delivery models in diverse community settings will provide crucial guidance for real-world application (Fleischman et al., 2022). Research should investigate effectiveness and sustainability of school-based programs in diverse schools, community-based models in cultural centers or religious institutions, hybrid telehealth models addressing access barriers, and group-based protocols with culturally homogeneous populations (Gruzelier, 2014). This research should examine not just clinical outcomes but also engagement, retention, cost-effectiveness, and community acceptability (Betancourt et al., 2003).

Economic analyses demonstrating cost-effectiveness for underserved communities are essential for advocacy and policy change, coupled with decision-support tools and implementation toolkits made freely available through professional organization websites and continuing education platforms (López et al., 2012). Without clear documentation of return on investment and societal benefits, it remains difficult to secure funding for programs serving populations that most need access to neuroregulation services (Alegría et al., 2016). Research should document cost per successful treatment outcome, comparison to alternative treatments for similar conditions, societal cost savings from reduced healthcare utilization and improved functioning, and impact on health disparity reduction (Fleischman et al., 2022). Additionally, the

development of culturally diverse normative databases represents a fundamental infrastructure need (Chiao, 2009). Current databases predominantly reflect White, educated populations, potentially leading to misinterpretation when applied to diverse groups (Moss et al., 1985). Creating representative normative data will ensure appropriate assessment and treatment planning across all populations (Betting et al., 2006; Travis & Shear, 2010).

Critically, research priorities must include infrastructure development studies examining cost-effective delivery models for underserved communities. This includes comparative effectiveness research on community-embedded delivery models versus traditional clinic-based approaches, cost-benefit analyses accounting for broader social determinants of health and community-level outcomes rather than only individual clinical metrics, and implementation studies documenting successful partnerships between academic institutions and community organizations that enable service delivery in under-resourced settings. Without evidence documenting financially sustainable models for serving diverse populations, calls for cultural responsiveness remain disconnected from the economic realities constraining practice and research (Betancourt et al., 2003; Fleischman et al., 2022).

System and Policy Change Requirements

Individual and organizational efforts, while necessary, prove insufficient without broader systemic change addressing structural barriers to equitable neuroregulation access, as emphasized in the health disparities framework detailed in the companion article (Metzl & Hansen, 2014; Sherlin, 2026). Insurance coverage expansion requires coordinated advocacy efforts at federal and state levels (López et al., 2012). Professional organizations including ISNR, Association for Applied Psychophysiology and Biofeedback (AAPB), and BCIA must work together bringing together practitioners, researchers, and affected communities to document effectiveness with diverse populations, demonstrate cost-effectiveness compared to traditional treatments, advocate for Medicaid and Medicare coverage expansion, and develop billing codes and documentation standards facilitating reimbursement (Betancourt et al., 2003). Current coverage limitations disproportionately affect communities of color and low-income populations who cannot afford out-of-pocket costs, perpetuating disparities in access to these effective interventions (López et al., 2012; Saadi et al., 2017).

Equally critical is reform of service delivery models themselves. The current reliance on private practice, clinic-based delivery concentrates services in affluent areas and creates insurmountable barriers for practitioners committed to serving diverse communities. Policy initiatives must incentivize alternative delivery structures including equipment loan programs modeled on the Neurofeedback Advocacy Project approach (Fleischman et al., 2022), which successfully expanded access through strategic equipment placement and training support; community health center integration providing neurofeedback within federally qualified health centers that already serve predominantly minority and low-income populations; school-based service delivery eliminating transportation and financial barriers while normalizing mental health interventions; and mobile neurofeedback units bringing services directly to underserved communities. Public funding streams (including Substance Abuse and Mental Health Services Administration [SAMHSA] grants, foundation support, and state mental health appropriations) should explicitly prioritize infrastructure development for community-embedded delivery models rather than only supporting individual practitioner training (López et al., 2012).

Workforce diversification through pipeline programs represents another critical system-level intervention (D. W. Sue et al., 1992). The current lack of diversity in the neuroregulation workforce limits both cultural competence and community trust, creating additional barriers for diverse clients seeking services (G. C. N. Hall, 2001). Strategies should include pipeline programs introducing neuroregulation careers to underrepresented students through school presentations, mentorship opportunities, and exposure to the field; scholarship support reducing financial barriers to training for students from underrepresented backgrounds; mentorship programs supporting professional development and retention of practitioners from diverse backgrounds; and recruitment of bilingual and bicultural practitioners addressing language barriers and cultural understanding simultaneously (D. W. Sue et al., 1992; Flores, 2005).

Research funding priorities must explicitly include diversity and cultural factors as review criteria for federal and foundation grants (G. C. N. Hall, 2001). Without such requirements, research will continue to predominantly reflect majority populations, limiting generalizability and perpetuating evidence gaps (Chiao, 2009). Funding agencies should require meaningful inclusion plans for diverse populations,

cultural factor analysis in research design and interpretation, community engagement strategies ensuring research serves community needs, and translation of findings into accessible formats for practitioners and communities (Betancourt et al., 2003). Grant review criteria should specifically evaluate cultural considerations, and study sections should include members with cultural research expertise (G. C. N. Hall, 2001).

Finally, integration with public health initiatives positions neuroregulation as a population health tool rather than a boutique intervention available only to privileged populations (Betancourt et al., 2003). Opportunities for integration include school-based mental health programs addressing ADHD and learning difficulties in collaboration with special education services, community trauma response initiatives providing accessible treatment following collective trauma such as natural disasters or community violence, substance abuse prevention and treatment programs incorporating neurofeedback as an adjunctive intervention, and chronic disease management programs addressing the neurological components of conditions like diabetes and hypertension (Gruzelier, 2014; Fleischman et al., 2022). Such integration would dramatically expand access while normalizing neuroregulation as a standard component of comprehensive healthcare rather than a specialized service known only to those with resources to seek it out (López et al., 2012).

Practice Guidelines Development Needs

Professional organizations must take leadership in developing comprehensive practice guidelines for culturally responsive neuroregulation based on available evidence and expert consensus where evidence is lacking (S. Sue et al., 2009). These guidelines need to address multiple domains of practice, beginning with assessment procedures that incorporate validated cultural assessment tools appropriate for neuroregulation contexts (Kleinman & Benson, 2006). Current assessment practices often overlook cultural factors that may significantly influence treatment engagement and outcomes, leaving practitioners without clear guidance on how to systematically evaluate and address these considerations (Betancourt et al., 2003). Guidelines should specify minimum cultural assessment components, appropriate interpreter use protocols, normative database selection and interpretation with diverse populations, and documentation of cultural factors in clinical records (Chiao, 2009; Flores, 2005).

Treatment adaptation guidelines must clarify which elements of neuroregulation protocols can be modified to enhance cultural responsiveness versus which elements must be preserved to maintain therapeutic integrity (Bernal et al., 2009). Drawing on the distinction between universal mechanisms and culturally variable factors established in the companion review (Sherlin, 2026), guidelines should specify core protocol components requiring fidelity, acceptable adaptation dimensions including feedback displays, reward systems, and session structure, contraindicated modifications that would compromise treatment effectiveness, and documentation of adaptations and clinical reasoning (Griner & Smith, 2006; G. C. N. Hall et al., 2016). Practitioners currently navigate these decisions without clear frameworks, potentially leading to either ineffective over-adaptation that compromises core mechanisms or rigid adherence that alienates diverse clients (La Roche & Christopher, 2009). Clear guidelines would help practitioners confidently make adaptation decisions while maintaining treatment effectiveness (Bernal et al., 2009).

Training requirements establishing minimum competency standards for culturally responsive practice need to be integrated into certification and continuing education requirements (S. Sue et al., 2009). The current absence of such standards means practitioners may enter the field with minimal preparation for serving diverse populations, perpetuating disparities through well-intentioned but inadequate service provision (G. C. N. Hall, 2001). Certification bodies should specify required training hours in cultural competence as prerequisite for certification, continuing education requirements for certification maintenance, competency demonstration through examination or practical assessment, and supervision requirements including cultural competence focus (Betancourt et al., 2003; S. Sue et al., 2009).

Quality indicators defining metrics for evaluating cultural responsiveness at both individual practitioner and organizational levels would enable systematic monitoring and improvement of equity in service delivery (López et al., 2012). These metrics should encompass process measures such as interpreter utilization rates, cultural assessment completion rates, and staff training participation; outcome measures examining disparity reduction in engagement and clinical outcomes across demographic groups; and balancing measures ensuring that quality is maintained while access is expanded (Betancourt et al., 2003). Professional organizations should develop standardized equity

dashboards that organizations can adapt to their specific contexts (López et al., 2012).

Conclusion

Implementing culturally responsive neuroregulation represents both ethical imperative and clinical necessity for effectively serving increasingly diverse populations (Betancourt et al., 2003; S. Sue et al., 2009). This article has detailed a comprehensive three-level implementation framework. The synthesis reveals emergent synergies where clinical practice adaptations, when coupled with systematic organizational quality monitoring, create reinforcing effects that enhance both engagement and retention outcomes (López et al., 2012), and where professional development initiatives enhance both individual practitioner competence and organizational capacity for sustained change. The framework provides concrete strategies for immediate implementation while working toward systemic changes ensuring equitable access to these powerful interventions.

At the clinical practice level, culturally responsive care requires thorough cultural assessment exploring explanatory models, technology comfort, language preferences, and family involvement expectations (Eisenbarth et al., 2025; Kleinman & Benson, 2006). The LEARN model provides a structured approach for integrating assessment findings into collaborative treatment planning (Berlin & Fowkes, 1983). Communication strategies must address both verbal and nonverbal elements, with particular attention to high-context versus low-context communication styles and appropriate interpreter utilization (Flores, 2005; E. T. Hall, 1976). Treatment adaptations should balance fidelity to core neurophysiological mechanisms with responsiveness to cultural needs, modifying feedback displays, reward systems, and session structures while maintaining therapeutic integrity (Bernal et al., 2009; Griner & Smith, 2006).

At the organizational level, addressing access barriers requires creative solutions to financial, geographic, and temporal obstacles through sliding scale fees, alternative service delivery models, and community-based programs (Betancourt et al., 2003; Fleischman et al., 2022). Quality monitoring with explicit attention to equity enables identification and remediation of disparities in engagement and outcomes (López et al., 2012). Telehealth offers substantial access potential but requires careful attention to digital literacy, internet reliability, privacy in shared spaces, and cultural acceptability.

At the professional development level, individual practitioners must commit to lifelong learning through formal training, experiential learning with diverse populations, and cultural humility practices (Tervalon & Murray-García, 1998; S. Sue et al., 2009). Organizations must implement comprehensive training addressing all staff levels, utilize diverse modalities including simulation and mentorship, and create supervision structures supporting cultural competence development (Betancourt et al., 2003). Professional organizations must integrate cultural competence into certification standards, accreditation requirements, and ethical guidelines (S. Sue et al., 2009).

The distinction between universal physiological mechanisms and culturally variable implementation factors, established in the companion review (Sherlin, 2026), provides essential clarity for practitioners navigating adaptation decisions. Confidence in mechanism universality allows focus on addressing engagement barriers without compromising treatment integrity (Sherlin et al., 2011). Core processes including operant conditioning, neuroplasticity, and autonomic regulation operate identically across human populations (Porges, 2011). What varies culturally is how individuals access, understand, and integrate these mechanisms into their lives (Kirmayer & Ryder, 2016; Hinton & Good, 2016).

Evidence demonstrates that when cultural barriers are systematically addressed, diverse populations can achieve outcomes comparable to majority populations (Feldman et al., 2016; Fleischman et al., 2022), though important caveats exist. Not all cultural adaptation studies show positive effects, and methodological limitations in existing research (including small sample sizes, limited geographic diversity, and short follow-up periods) necessitate cautious interpretation. Most supportive evidence derives from North American contexts, warranting replication in diverse international settings before claiming universal generalizability. This finding indicates that observed disparities stem primarily from implementation failures rather than differential treatment response, offering hope that systematic implementation of culturally responsive practices can significantly reduce health inequities (López et al., 2012). The reported no-show rate of 2% in a community-based program serving diverse populations illustrates the engagement possible under culturally responsive implementation (Fleischman et al., 2022).

Achieving these goals requires honest acknowledgment of structural and economic realities that constrain implementation. The concentration of neuroregulation services in private practices serving affluent, predominantly White communities reflects systemic factors including equipment costs, training expenses, reimbursement limitations, and referral patterns rather than individual practitioner choices alone. Individual practitioners working in under-resourced settings or small-scale researchers at universities with limited funding face genuine constraints in serving culturally diverse populations, regardless of cultural competence or commitment to equity. Progress therefore demands parallel action at multiple levels: individual practitioners developing cultural competence within their sphere of influence, organizations innovating delivery models that overcome economic barriers, and policy makers investing in infrastructure that makes equitable access financially feasible rather than requiring practitioners to choose between economic sustainability and equity commitments (López et al., 2012; Metzl & Hansen, 2014).

Implementation challenges, while real, prove surmountable with commitment, creativity, and collaboration (Betancourt et al., 2003). Resource limitations can be addressed through graduated implementation, strategic allocation, and creative partnerships (Alegría et al., 2016). Staff resistance decreases with education, skill-building, and creation of psychologically safe learning environments (S. Sue et al., 2009; Tervalon & Murray-García, 1998). Time constraints diminish as integration replaces addition and efficiency improves with competence (Flores, 2005). Each challenge overcome strengthens organizational capacity for serving diverse populations (López et al., 2012).

Future progress demands coordinated action across multiple stakeholders with phased implementation. In the near term (suggested 6–12 months), practitioners might begin with cultural humility self-assessment and integration of core assessment adaptations into clinical practice (Tervalon & Murray-García, 1998). Organizations could focus on implementing quality monitoring systems with equity metrics and initiating community partnership development (suggested 12–24 months; López et al., 2012). Professional associations might work toward establishing cultural competence standards and convening stakeholders for guideline development (suggested 18–36 months; S. Sue et al., 2009). Over the longer term (3–5 years), researchers must address knowledge gaps through inclusive studies with diverse populations, cultural

factor analysis, and community-engaged research (G. C. N. Hall, 2001), while policy makers could pursue systemic reforms through legislative change, research funding priorities explicitly including diversity requirements, and integration of neuroregulation into public health initiatives (Metzl & Hansen, 2014; Saadi et al., 2017). These timelines represent conceptual implementation phases rather than prescriptive mandates, recognizing that local contexts will influence feasible pacing.

The journey toward culturally responsive neuroregulation is ongoing rather than achievable endpoint (Tervalon & Murray-García, 1998). It requires humility acknowledging current limitations and mistakes, courage confronting uncomfortable disparities, creativity developing innovative solutions to complex structural barriers, and persistence continuing despite challenges. Critically, this journey is grounded in the universality of neurophysiological mechanisms (operant conditioning, neuroplasticity, autonomic regulation) that operate identically across all human populations (Porges, 2011; Sherlin et al., 2011), ensuring that cultural adaptations enhance rather than erode treatment efficacy. Most importantly, it requires genuine commitment to ensuring that neuroregulation's benefits reach all who might benefit regardless of cultural background, language, race, ethnicity, or socioeconomic status (Betancourt et al., 2003).

Through systematic implementation of culturally responsive practices at clinical, organizational, and professional levels, the neuroregulation field can fulfill its potential to provide effective interventions for all members of our diverse human family (S. Sue et al., 2009). This is not merely matter of social justice but of scientific excellence, as truly effective practice must be culturally responsive practice (Betancourt et al., 2003; López et al., 2012). The framework and strategies presented provide roadmap for this essential journey. The universal neurophysiological mechanisms underlying neuroregulation offer foundation for inclusivity (Sherlin et al., 2011; Porges, 2011), while attention to cultural factors ensures these mechanisms are accessible and beneficial for all populations (G. C. N. Hall et al., 2016). Our challenge is not to prove that neuroregulation can work across diverse populations (the fundamental science assures us it can) but rather to ensure we implement these interventions in ways that are accessible, acceptable, and effective for everyone (Feldman et al., 2016; Fleischman et al., 2022).

Author Declaration

At the time of submission, the author served as President of the International Society for Neuroregulation & Research (ISNR), Chair of the Board of the Biofeedback Certification International Alliance (BCIA), and an Advisory Board member of the International QEEG Certification Board (IQCB). These roles are disclosed in the interest of transparency, as the manuscript addresses certification standards, professional guidelines, and organizational practices relevant to these bodies. The author reports no financial interests or grant support relevant to this submission.

Artificial intelligence tools were used exclusively for technical manuscript preparation (proofreading and formatting) in this work. No AI was used for content generation, analysis, or intellectual contributions. The author assumes full responsibility for all scientific content and conclusions presented.

References

- Alegría, M., Alvarez, K., Ishikawa, R. Z., DiMarzio, K., & McPeck, S. (2016). Removing obstacles to eliminating racial and ethnic disparities in behavioral health care. *Health Affairs*, 35(6), 991–999. <https://doi.org/10.1377/hlthaff.2016.0029>
- Bazargan, M., Cobb, S., & Assari, S. (2021). Discrimination and medical mistrust in a racially and ethnically diverse sample of California adults. *Annals of Family Medicine*, 19(1), 4–15. <https://doi.org/10.1370/afm.2632>
- Biofeedback Certification International Alliance (n.d.). *Benefits of board certification in neurofeedback* [Brochure]. <https://bcia.memberclicks.net/assets/NFCommonDocs/NF%20Brochure.pdf>
- Beltrán-Velasco, A. I., Bellido-Esteban, A., Ruisoto-Palomera, P., Mendoza, K. H., & Clemente-Suárez, V. J. (2020). The effect of cultural differences in psychophysiological stress response in high education context: A pilot study. *Applied Psychophysiology and Biofeedback*, 45(1), 23–29. <https://doi.org/10.1007/s10484-019-09452-0>
- Benish, S. G., Quintana, S., & Wampold, B. E. (2011). Culturally adapted psychotherapy and the legitimacy of myth: A direct-comparison meta-analysis. *Journal of Counseling Psychology*, 58(3), 279–289. <https://doi.org/10.1037/a0023626>
- Berlin, E. A., & Fowkes, W. C. (1983). A teaching framework for cross-cultural health care. *Western Journal of Medicine*, 139(6), 934–938.
- Bernal, G., Jiménez-Chafey, M. I., & Domenech Rodríguez, M. M. (2009). Cultural adaptation of treatments: A resource for considering culture in evidence-based practice. *Professional Psychology: Research and Practice*, 40(4), 361–368. <https://doi.org/10.1037/a0016401>
- Betancourt, J. R., Green, A. R., Carrillo, J. E., & Owusu Ananeh-Firempong, I. I. (2003). Defining cultural competence: A practical framework for addressing racial/ethnic disparities in health and health care. *Public Health Reports*, 118(4), 293–302. <https://doi.org/10.1093/phr/118.4.293>
- Betting, L. E., Mory, S. B., Lopes-Cendes, I., Li, L. M., Guerreiro, M. M., Guerreiro, C. A., & Cendes, F. (2006). EEG features in idiopathic generalized epilepsy: Clues to diagnosis. *Epilepsia*, 47(3), 523–528. <https://doi.org/10.1111/j.1528-1167.2006.00462.x>

- Campinha-Bacote, J. (2002). The process of cultural competence in the delivery of healthcare services: A model of care. *Journal of Transcultural Nursing*, 13(3), 181–184. <https://doi.org/10.1177/10459602013003003>
- Chiao, J. Y. (Ed.). (2009). *Cultural neuroscience: Cultural influences on brain function*. Elsevier.
- Constant, A., Ramstead, M. J., Veissière, S. P., & Friston, K. (2021). Regimes of expectations: An active inference model of social conformity and human decision making. *Frontiers in Psychology*, 10, Article 679. <https://doi.org/10.3389/fpsyg.2019.00679>
- Eisenbarth, H., D'Cruz, C., Bulbulia, J. A., & Thanni, B. (2025). Culturally diverse perceptions of EEG and neurofeedback research and how to address them to reduce sampling bias. *Psychophysiology*, 62(6), Article e70077. <https://doi.org/10.1111/psyp.70077>
- Feldman, J. M., Matte, L., Interian, A., Lehrer, P. M., Lu, S.-E., Scheckner, B., Steinberg, D. M., Oken, T., Kotay, A., Sinha, S., & Shim, C. (2016). Psychological treatment of comorbid asthma and panic disorder in Latino adults: Results from a randomized controlled trial. *Behaviour Research and Therapy*, 87, 142–154. <https://doi.org/10.1016/j.brat.2016.09.007>
- Fleischman, M. J., Othmer, S., Kallay, E., & Huffman, T. (2022). Documenting the impact of infra low frequency neurofeedback on underserved populations with complex clinical presentations. *Frontiers in Human Neuroscience*, 16, Article 921491. <https://doi.org/10.3389/fnhum.2022.921491>
- Flores, G. (2005). The impact of medical interpreter services on the quality of health care: A systematic review. *Medical Care Research and Review*, 62(3), 255–299. <https://doi.org/10.1177/1077558705275416>
- Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *American Journal of Public Health*, 96(5), 826–833. <https://doi.org/10.2105/AJPH.2004.060749>
- Griner, D., & Smith, T. B. (2006). Culturally adapted mental health intervention: A meta-analytic review. *Psychotherapy: Theory, Research, Practice, Training*, 43(4), 531–548. <https://doi.org/10.1037/0033-3204.43.4.531>
- Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews*, 44, 159–182. <https://doi.org/10.1016/j.neubiorev.2014.03.015>
- Hall, E. T. (1976). *Beyond culture*. Anchor Books.
- Hall, G. C. N. (2001). Psychotherapy research with ethnic minorities: Empirical, ethical, and conceptual issues. *Journal of Consulting and Clinical Psychology*, 69(3), 502–510. <https://doi.org/10.1037/0022-006X.69.3.502>
- Hall, G. C. N., Ibaraki, A. Y., Huang, E. R., Marti, C. N., & Stice, E. (2016). A meta-analysis of cultural adaptations of psychological interventions. *Behavior Therapy*, 47(6), 993–1014. <https://doi.org/10.1016/j.beth.2016.09.005>
- Hinton, D. E., & Good, B. J. (Eds.). (2016). *Culture and PTSD: Trauma in global and historical perspective*. University of Pennsylvania Press.
- Kirmayer, L. J., & Ryder, A. G. (2016). Culture and psychopathology. *Current Opinion in Psychology*, 8, 143–148. <https://doi.org/10.1016/j.copsyc.2015.10.020>
- Kleinman, A., & Benson, P. (2006). Anthropology in the clinic: The problem of cultural competency and how to fix it. *PLoS Medicine*, 3(10), Article e294. <https://doi.org/10.1371/journal.pmed.0030294>
- Kolken, Y., Bouny, P., & Arns, M. (2023). Effects of SMR neurofeedback on cognitive functions in an adult population with sleep problems: A tele-neurofeedback study. *Applied Psychophysiology and Biofeedback*, 48(1), 27–33. <https://doi.org/10.1007/s10484-022-09560-4>
- Krepel, N., Egtberts, T., Toure-Cuq, E., Bouny, P., & Arns, M. (2022). Evaluation of the URGOnight tele-neurofeedback device: An open-label feasibility study with follow-up. *Applied Psychophysiology and Biofeedback*, 47(1), 43–51. <https://doi.org/10.1007/s10484-021-09525-z>
- La Roche, M. J., & Christopher, M. S. (2009). Changing paradigms from empirically supported treatment to evidence-based practice: A cultural perspective. *Professional Psychology: Research and Practice*, 40(4), 396–402. <https://doi.org/10.1037/a0015240>
- López, S. R., Barrio, C., Kopelowicz, A., & Vega, W. A. (2012). From documenting to eliminating disparities in mental health care for Latinos. *American Psychologist*, 67(7), 511–523. <https://doi.org/10.1037/a0029737>
- McEwen, B. S. (2017). Neurobiological and systemic effects of chronic stress. *Chronic Stress*, 1, Article 2470547017692328. <https://doi.org/10.1177/2470547017692328>
- Metzl, J. M., & Hansen, H. (2014). Structural competency: Theorizing a new medical engagement with stigma and inequality. *Social Science & Medicine*, 103, 126–133. <https://doi.org/10.1016/j.socscimed.2013.06.032>
- Moss, E. M., Davidson, R. J., & Saron, C. (1985). Cross-cultural differences in hemisphericity: EEG asymmetry discriminates between Japanese and Westerners. *Neuropsychologia*, 23(1), 131–135. [https://doi.org/10.1016/0028-3932\(85\)90054-5](https://doi.org/10.1016/0028-3932(85)90054-5)
- Nelson, K. L., Lu, S.-E., Oken, T., Lehrer, P. M., & Feldman, J. M. (2020). Further exploration of treatment response in Latinos with comorbid asthma and panic disorder: A brief report of HRV and ETCO2 as potential mediators of treatment response. *Applied Psychophysiology and Biofeedback*, 45(2), 67–74. <https://doi.org/10.1007/s10484-020-09454-3>
- Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation*. Norton.
- Resnicow, K., Baranowski, T., Ahluwalia, J. S., & Braithwaite, R. L. (1999). Cultural sensitivity in public health: Defined and demystified. *Ethnicity & Disease*, 9(1), 10–21.
- Saadi, A., Himmelstein, D. U., Woolhandler, S., & Meija, N. I. (2017). Racial disparities in neurologic health care access and utilization in the United States. *Neurology*, 88(24), 2268–2275. <https://doi.org/10.1212/WNL.0000000000004025>
- Schwartz, M. S., & Andrasik, F. (2017). *Biofeedback: A practitioner's guide (4th ed.)*. Guilford Press.
- Sherlin, L. H. (2026). Cultural factors in neuroregulation: A review of evidence and theoretical framework. *NeuroRegulation*, 13(1), 65–76. <https://doi.org/10.15540/nr.13.1.65>
- Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Serman, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy*, 15(4), 292–304. <https://doi.org/10.1080/10874208.2011.623089>
- Sue, D. W., Arredondo, P., & McDavis, R. J. (1992). Multicultural counseling competencies and standards: A call to the profession. *Journal of Counseling & Development*, 70(4), 477–486. <https://doi.org/10.1002/j.1556-6676.1992.tb01642.x>
- Sue, S., Zane, N., Nagayama Hall, G. C., & Berger, L. K. (2009). The case for cultural competency in psychotherapeutic interventions. *Annual Review of Psychology*, 60, 525–548. <https://doi.org/10.1146/annurev.psych.60.110707.163651>
- Tervalon, M., & Murray-García, J. (1998). Cultural humility versus cultural competence: A critical distinction in defining physician training outcomes in multicultural education. *Journal of Health Care for the Poor and Underserved*, 9(2), 117–125. <https://doi.org/10.1353/hpu.2010.0233>
- Travis, F., & Shear, J. (2010). Focused attention, open monitoring and automatic self-transcending: Categories to organize meditations from Vedic, Buddhist and Chinese traditions. *Consciousness and Cognition*, 19(4), 1110–1118. <https://doi.org/10.1016/j.concog.2010.01.007>

U.S. Census Bureau. (2018). Demographic turning points for the United States: Population projections for 2020 to 2060. <https://www.census.gov/content/dam/Census/library/publications/2020/demo/p25-1144.pdf>

van der Kolk, B. A. (2014). *The body keeps the score: Brain, mind, and body in the healing of trauma*. Viking.

Received: November 13, 2025
Accepted: January 5, 2026
Published: September 28, 2026

Frontal Gamma Asymmetry as a Forensic Tool for Emotional Valence in a Murder Case Study

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Abstract

In criminal cases, forensic evaluations often hinge on determining a defendant's competence and intent. Assessing underlying emotional states presents unique challenges, as self-reports may be unreliable or incomplete. In this exploratory single-case study, we illustrate how psychological assessments can be combined with quantitative EEG (qEEG) and source localization techniques to examine neural markers of emotional processing in the context of a murder trial. Emotional valence toward key individuals and events was derived from a proprietary frontal gamma asymmetry protocol. Although this innovative metric offers unique insights, it has received limited independent replication and should therefore be interpreted cautiously and in conjunction with established measures such as frontal alpha asymmetry. Key procedural covariates, including the defendant's medication (sertraline), were documented because serotonergic agents can modulate baseline asymmetry patterns. Findings indicated general alignment between neural patterns and reported affect, but we emphasize that these observations reflect preliminary insights from a single individual and require validation in larger samples before supporting broader forensic applications.

Keywords: Frontal gamma asymmetry; quantitative EEG (qEEG); emotional valence; forensic neuroscience; mens rea assessment; Brodmann area 9 (BA9)

Citation: Cantor, D. S., Collura, T. F., & Bonnstetter, R. J. (2026). Frontal gamma asymmetry as a forensic tool for emotional valence in a murder case study. *NeuroRegulation*, 13(3), 319–337. <https://doi.org/10.15540/nr.13.3.319>

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Edited by: Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA

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Reviewed by: Rex L. Cannon, PhD, Currents, Knoxville, Tennessee, USA
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Introduction

Mental health professionals are frequently called upon in murder cases to assist courts in determining both a defendant's competence to stand trial and their mental state at the time of the offense. Central to such evaluations is the concept of mens rea, or criminal intent, which is often decisive in distinguishing between degrees of culpability. Legal doctrine typically considers two elements of crime: the *actus reus* (the physical act) and the *mens rea* (the mental state). Modern interpretations of mens rea in homicide require juries not only to examine the subjective state of mind of the accused but also to contextualize this within the broader circumstances surrounding the crime (Nourse, 2002). Forensic psychologists therefore play a critical role in clarifying the extent to which

psychological, neurological, or environmental factors may have shaped a defendant's intentions.

This case study explores these issues in the context of a 32-year-old male defendant charged with the murder of his uncle. The case illustrates the tension between claims of self-defense, the presence of longstanding family conflict, and questions regarding the defendant's emotional regulation and psychiatric history (Cantor, 2016). By situating the legal question of mens rea within a neuropsychological framework, the analysis demonstrates how complex family dynamics, mental health vulnerabilities, and situational stressors intersect in ways that complicate legal determinations of intent.

The defendant's background included exposure to chronic familial discord, parental mental health

difficulties, and prior traumatic experiences. Research in trauma psychology has established that cumulative stressors and unresolved interpersonal conflicts can exacerbate mood dysregulation and increase vulnerability to reactive aggression (Ford & Courtois, 2021; Teicher & Samson, 2016). Reports of depressive symptoms, self-injurious behavior, and paranoid ideation further suggest a psychological profile consistent with difficulties in emotional regulation and resilience. These elements not only provide context for the defendant’s actions but also align with forensic psychiatry literature that emphasizes the role of psychiatric comorbidity in shaping violent outcomes (Chow et al., 2025).

Within neuroscience, frontal alpha asymmetry has long served as a proxy for emotional valence, with greater relative left frontal activity associated with approach related to positive affect and right frontal activity linked to withdrawal or negative affect (Davidson, 1998). Gamma band asymmetry, by contrast, is a more recent and largely proprietary extension that aims to capture faster, preconscious aspects of valence processing. Because the scientific literature on gamma asymmetry is much smaller than that on alpha, it is important to contextualize our approach as an experimental adaptation of established theories rather than a

replacement and to recognize that the evidentiary base for forensic application is still emerging.

To frame the legal defense, it is important to consider the range of arguments typically available in murder cases, from mistaken identity to insanity pleas. Table 1 outlines common defenses and highlights how each applies—or fails to apply—in this case. For example, while self-defense could be argued given the victim’s threats and perceived reach for a weapon, its validity was undermined by the defendant’s continued use of lethal force after the victim was incapacitated. Conversely, the defense team emphasized the possibility of a trauma-related psychiatric condition, positioning the defendant’s actions as stemming from an acute emotional cascade rather than premeditation.

Table 1 thus serves not merely as a legal overview but as an interpretive tool to clarify why certain defenses were dismissed and why others, particularly those invoking psychiatric explanations such as temporary insanity, became central to the case strategy. This framing highlights the challenges courts face in relying on subjective accounts of emotional states, underscoring the need for more objective neuropsychological measures to evaluate intent and motive.

Table 1
Common Defenses in Murder Cases and Application to the Present Case

Type of Defense	General Explanation	Application to This Case
Mistaken Identity	Defendants may argue that the prosecution has charged the wrong person with the killing.	Not applicable: multiple eyewitnesses observed the defendant commit the act.
Justified Homicide	Some killings are considered legally justified, such as those in lawful defense.	Partially relevant: the victim initiated threats; however, subsequent excessive force undermined this argument.
Self-Defense	Requires evidence that the killing resulted from a reasonable and proportional response to an imminent threat of death or serious bodily harm.	Weakly applicable: although the victim threatened and appeared to reach for a weapon, the defendant continued firing after the victim was incapacitated.
Defense of Others	Use of force may be justified to protect others from imminent harm, provided it is reasonable and proportional.	Considered but not substantiated: the threat was directed at the defendant rather than others present.

Table 1
Common Defenses in Murder Cases and Application to the Present Case

Type of Defense	General Explanation	Application to This Case
Exercise of Duty	Killings by law enforcement or public officers in the line of duty may be justified.	Not applicable: defendant was a private citizen.
Accident or Misfortune	Unintentional killings occurring during lawful activity may reduce liability to manslaughter.	Not applicable: multiple deliberate shots were fired.
Insanity Defense	Based on the inability to appreciate the nature of the act or to understand its wrongfulness due to mental illness or cognitive impairment.	Most relevant: defense strategy emphasized trauma-related psychiatric conditions and possible 'temporary insanity' stemming from prior family conflict and emotional dysregulation.

Methods

Psychological Evaluation History and Test Battery (Current Functional Status)

The defendant was reportedly born at term via uncomplicated vaginal delivery, and he described his early developmental history as unremarkable. There is no documented evidence of prenatal drug or alcohol exposure in the available records, although prenatal history was based solely on his self-report and is therefore limited. His medical history included intermittent headaches, dizziness, visual blurring, fatigue, chest pain, palpitations, allergies (penicillin), and lower-back pain, with no known neurological disorders. He has experienced lifelong depression, anxiety with panic episodes, attentional problems, and periods of impulsivity, and has a history of self-injurious cutting following his father’s death. He had been treated with sertraline (Zoloft) 200 mg daily for approximately 2 years, which he reported taking consistently, including at the time of the alleged offense. He also reported regular alcohol use (approximately two shots nightly for sleep). Prior medical care included allergy immunotherapy during childhood and brief treatment for acid reflux. Family history is notable for significant psychiatric disorders (depression, anxiety, suicidality), learning problems, deafness in both parents, and substance abuse in multiple relatives, indicating a substantial hereditary vulnerability to mood and behavioral dysregulation.

A psychological battery was administered to assess cognitive, behavioral, personality, and emotional functioning using age-normed instruments with established reliability. Tests included:

- 16 Personality Factor–5 (16PF)
- Adult Self-Report, 18–59 (ASR/18–59)
- Adult Manifest Anxiety Scale–Adult (AMAS-A)
- Behavior Rating Inventory of Executive Function–Adult (BRIEF-A, self and informant)
- Green’s Word Memory Test (WMT)
- Integrated Visual and Auditory Continuous Performance Test (IVA/CPT)
- Miller Forensic Assessment of Symptoms Test (M-FAST)
- Minnesota Multiphasic Personality Inventory–2 (MMPI-2)
- Sensory Profile (Adolescent/Adult)
- Trauma Symptom Inventory (TSI)
- Wechsler Adult Intelligence Scale–IV (WAIS-IV)
- Wide Range Achievement Test–4 (WRAT-4; selected subtests)
- Neurometric qEEG

Administration/Scoring. All instruments were administered and scored per publisher manuals within a single evaluation period preceding EEG acquisition. Standard validity checks (e.g., MMPI-2 validity scales, WMT performance indicators) were used to determine interpretability. Instruments were selected for their routine forensic relevance (general intellectual ability, effort/response validity, executive function, attention, personality/psychopathology, trauma, anxiety/depression).

Rationale for Instrument Selection. Instruments were selected to capture distinct constructs relevant to forensic competence: the WAIS-IV and WRAT-4 assessed general intellectual ability and academic skills; the WMT and M-FAST evaluated effort and response validity; the BRIEF-A and IVA/CPT measured executive attention and inhibitory control; the 16PF, MMPI-2, AMAS-A, and TSI characterized personality, psychopathology, anxiety, and trauma exposure; and the Sensory Profile and ASR provided contextual information on sensory processing and adaptive behavior. Together, these measures addressed competence to stand trial, potential mitigating mental disorders, and possible malingering, aligning with forensic best practices.

Behavioral Analysis

Prior to the EEG data collection, a 10-min behavioral assessment tool provided by Target Training International (TTI) was administered (Gehrig & Bonnstetter, 2021). The tool subdivides a person's behaviors based on the strength of four constructs: (D) dominance, (I) influencing, (S) steadiness, and (C) compliance. This tool was administered to document the defendant's baseline behavioral style to aid stimulus selection and finally to align subsequent EEG measures with self-reported traits.

Neurometric qEEG Examination

Hardware/Montage. EEG was acquired using a Cadwell Easy II amplifier with a 19-channel 10–20 montage (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, O2). Data were collected eyes open with a 1–70 Hz band-pass and a 60 Hz notch filter. Frequency metrics were computed for delta (1–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (12–25 Hz), beta-2 (25–35 Hz) and gamma (38–42 Hz). Medication status at the time of testing was recorded (sertraline/Zoloft, 20 mg daily) as a procedural covariate.

Source Localization. Three-dimensional current density maps were generated with sLORETA. By activation, we refer to an observed increase in the measured sLORETA current source density (CSD) level, when compared with the baseline level at rest, which is measured from the EEG immediately preceding each stimulus. Given that the subject was on sertraline it is important to know that selective serotonin reuptake inhibitors such as sertraline (Zoloft) can influence electrophysiological measures of brain activity obtained through quantitative EEG and source-localization techniques such as sLORETA. These medications increase extracellular serotonin by inhibiting the serotonin transporter, thereby modulating cortical excitability and altering

the balance between excitatory pyramidal neurons and inhibitory interneurons within cortical networks (Knott et al., 1996; Leuchter et al., 2012). Gamma-band activity is particularly sensitive to these effects because gamma oscillations reflect synchronized activity of local cortical circuits and GABAergic interneurons. Pharmacology-EEG studies have shown that selective serotonin reuptake inhibitors (SSRI) treatment may produce modest increases in frontal gamma activity and improved synchronization of cortical oscillations in individuals with mood or anxiety disorders. These changes are thought to reflect greater stability of cortical network functioning and improved integration within prefrontal regulatory systems (Arns et al., 2015; Knott et al., 1996). Source-localization studies using standardized low-resolution electromagnetic tomography have similarly demonstrated treatment-related increases in activity within the dorsolateral prefrontal cortex, particularly in Brodmann areas 9 and 46, following successful antidepressant treatment (Leuchter et al., 2012; Pascual-Marqui, 2002). Such findings are generally interpreted as reflecting enhanced engagement of prefrontal executive networks that exert top-down regulatory control over limbic emotional systems. In addition to regional activation changes, antidepressant treatment has been associated with greater stability of EEG microstates and more coherent transitions among transient functional brain states. These effects suggest improved coordination across distributed neural networks and more stable large-scale cortical dynamics (Arns et al., 2015; Lehmann et al., 1998). Overall, the electrophysiological literature indicates that treatment with sertraline may be associated with modest increases in prefrontal cortical activity, stabilization of gamma-band oscillations, and more organized temporal dynamics of cortical network activity. However, there are no known effects to induce influence on frontal gamma asymmetry in either direction with the use of SSRI agents.

Paradigm to Assess Emotional Valence via Frontal Gamma Asymmetry

Gamma activity was measured by taking the average of all voxels within each region of interest (ROI). The sLORETA model was used for all computations, using the T matrix published by Pascual-Marqui (2002). The ROI for Brodmann area 9 (BA9) contained 225 voxels per side, for which the CSD was calculated for each voxel, and the average CSD used as the measured value. Gamma activity was recorded by processing the EEG at 256 samples/s with a digital filter (quadrature type, order 3) with corner frequencies set at 35 and 45 Hz. The quadrature filter provided instantaneous

estimates of gamma amplitude through complex demodulation, for the moment-to-moment changes in gamma amplitude. These were projected to sLORETA space using a local T matrix, at the rate of 6239 voxels

(x, y, and z) times 256 samples, for a processing rate 5 million sLORETA voxel calculations per second. Amplitude samples of the sLORETA voxel amplitudes were taken at intervals of 125 ms, providing 8 estimates per second of the entire sLORETA projection, reflecting the momentary changes in brain microstates. The rate of 8 images per second was limited primarily by the ability to generate and save images at that rate on a high-speed PC. While a faster rate of reconstruction may be of benefit, it was confirmed visually that the successive images using sLORETA demonstrated a smooth set of transitions, with no evidence of missing states. It was also confirmed by taking data at 12 images per second, that no new information was evident when using a faster rate.

Trigger Derivation

Researchers reviewed case materials (interviews, testimony) to derive 33 single words or short phrases representing key persons/events. The defendant completed a paper-and-pencil Likert rating for each item to anchor stimulus categories.

Stimulus Presentation and EEG Recording Protocol

Detailing all protocols used for the following frontal lobe gamma asymmetry process is beyond the scope of this article. The interested reader may consult (Bonnstetter, Bonnstetter, et al., 2015; Bonnstetter et al., 2016; Bonnstetter & Collura, 2020; Bonnstetter et al., 2012; Bonnstetter et al., 2022; Bonnstetter et al., 2018; Bonnstetter, Hebets, et al., 2015; Collura & Bonnstetter, 2020; Collura et al., 2016; Collura et al., 2014a; Collura et al., 2014b). These references will highlight the original application of gamma for ipsative validation using electroencephalography (GIVE) process to access asymmetric gamma wave bursts in the prefrontal cortex to validate the underlying preconscious decisions behind self-report responses at the very moment of decision-making.

The following analysis used the same process to expose the underlying mental state at the moment of decisions, thus revealing the thoughts and emotional load behind responses. This process reveals the likely evoked emotionally laden brain activity and documents both the intensity of human emotional responses and the directionality of the response.

Asymmetries in the frontal lobes with increased gamma in the right hemisphere have been identified with events or individuals experiencing an aversive reaction, while increased gamma activity in the left frontal region has been correlated to positive regard. Symmetric activity is typically noted in the frontal lobes with events that have been correlated to a neutral emotional reaction (Bonnstetter, Hebets, et al., 2015).

Documented Gamma Asymmetry Protocols

At the time of this case study, several international and national professional presentations had been made describing this process of identifying emotional valence, but only one peer-reviewed article had been published on the protocols (Bonnstetter et al., 2012). With this in mind, great care was given to document the process used in an effort to provide further insight regarding the defendant's emotional reactivity to key individuals and events related to the felony murder case. The first step for the researchers was to review hundreds of pages of testimony to derive a list of events and key people tied to the case so as to capture and rate the extent of his likely positive or negative feelings for each possible trigger. Prior to collecting EEG imaging and qEEG data, the client was asked to fill out a survey composed of 33 questions that would ultimately be used as stimuli for the EEG imaging.

Stimuli were programmed on a presentation workstation and displayed centrally on a monitor while EEG was recorded.

- Presentation software: E-Prime (timing control).
- On-screen duration: 1.5 s per item.
- Inter-stimulus interval: Randomized black screen 2–3 s.
- Runs: Two exposures to all 33 items, with a 5-min interval between runs (the second run served as a within-session replication).
- EEG acquisition for gamma asymmetry used BrainMaster BrainAvatar™ hardware and software in conjunction with the Cadwell system; raw EEG/EDF files were exported for postprocessing.

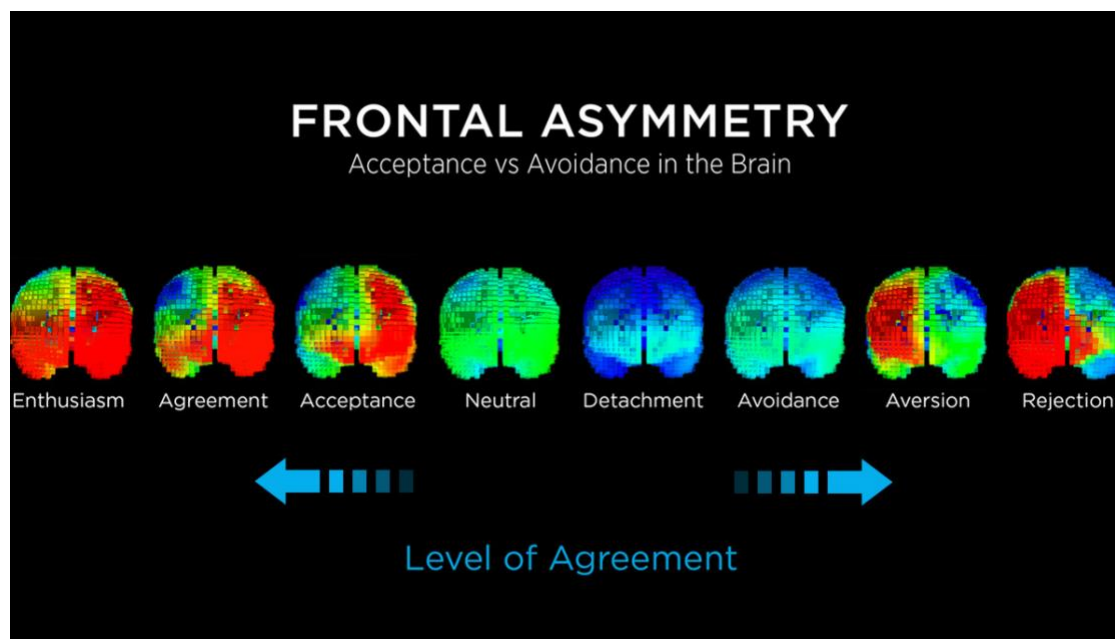
Processing and Analysis (Replication Details)

- Preprocessing: band-pass 0.5–40 Hz for artifact-sensitive analyses; ocular and muscle artifacts addressed using routine artifact rejection procedures (with independent component or equivalent artifact inspection and removal as needed).

- Spectral features: power estimates were calculated for standard bands and gamma (≈ 38 – 42 Hz) for asymmetry indices.
- Asymmetry metric: frontal gamma from F3/F4 (natural log transform), Asymmetry = $\ln(\text{right}) - \ln(\text{left})$ computed at the trial level and summarized by stimulus condition.
- Source imaging: sLORETA current density maps generated from stimulus-locked epochs.
- Reference/thresholding: age-normed databases; deviations considered notable at $|Z| \geq 2.0$ (documentation retained in the case archive).
- Replication check: first versus second run compared for direction consistency (see Findings).

Figure 1 presents an example of frontal lobe gamma asymmetry with positive, neutral, and negative responses. The orientation of the brain is facing forward such that the right hemisphere is on the left side of the image. The red indicates an increase in gamma activity, the blue indicates a decrease in activity, and the green indicates little or no activation. In addition to color depicting a range of response intensity, the gamma burst location is also key to interpretation. A left frontal lobe flare is an indication of acceptance or a positive response and a right-side flare demonstrates avoidance or a negative response to a stimulus.

Figure 1. Frontal Asymmetry.



Findings

Psychological Battery

Behavioral observations during testing indicated adequate attention, consistent effort, and cooperative engagement, supporting validity of the results. Performance suggested average intellectual functioning (WAIS-IV) with relative weakness in visual processing. Self-report measures reflected elevated anxiety and intermittent depressive symptoms relative to age-expected norms. Screening indicated borderline attentional

difficulties; informant ratings on parallel executive-function/attention items were not elevated, suggesting limited collateral awareness of the defendant's internal distress or functional complaints. The defendant self-reported executive-function challenges (planning, organization, initiation, flexibility) that were not corroborated by informant ratings. On a computerized CPT, overall indices were age-average, with mild impulsivity tendencies to auditory targets. Personality profiles were consistent with introversion, limited social trust, and heightened anxiety, with episodic depressed

mood. The totality of data supported that the defendant was competent to stand trial.

Behavioral Analysis

Figure 2 depicts the defendant’s adapted and natural behavioral styles. The “natural” style index represents a sense of self, and the “adapted” style index describes how a person might be adapting to their environment.

The primary “S” behavioral style suggests that the defendant is likely very loyal, enjoys stability and consistency and typically is very slow to anger. Such individuals value relationships, typically with a smaller group of people with whom they form strong bonds. Because of this tie to close relationships, family tends to be extremely important to them. The dominant “S” style, coupled with the high “C” style suggests a person follows rules and regulations. While the high “S” style tends to be refrained, when pushed beyond some critical emotional point, they tend to overreact.

It is also noteworthy to point out that Graph I and Graph II are basically identical, which indicates that

these descriptive characteristics are consistent likely to a high likelihood of stability over time and therefore most likely applicable to the time of the murder.

Figure 3 is a similar chart but with word descriptors highlighted with color bands to depict his “natural style.” A quick review of the descriptive words in each of the four columns lay the groundwork for a better understanding of the defendant’s natural behavioral style.

Limitations of the Behavioral Style Assessment.

The DISC-derived behavioral style report is a commercially available tool designed for workplace coaching rather than clinical diagnosis. Its adaptation here provided descriptive information about natural versus adapted interpersonal tendencies but has not been validated for forensic decision-making or for assessing emotional reactivity under high-stress conditions. Consequently, interpretations drawn from this measure should be seen as provisional and supplementary to the more rigorous neuropsychological and neurophysiological data.

Figure 2. Defendant’s Adapted and Natural Behavioral Styles.

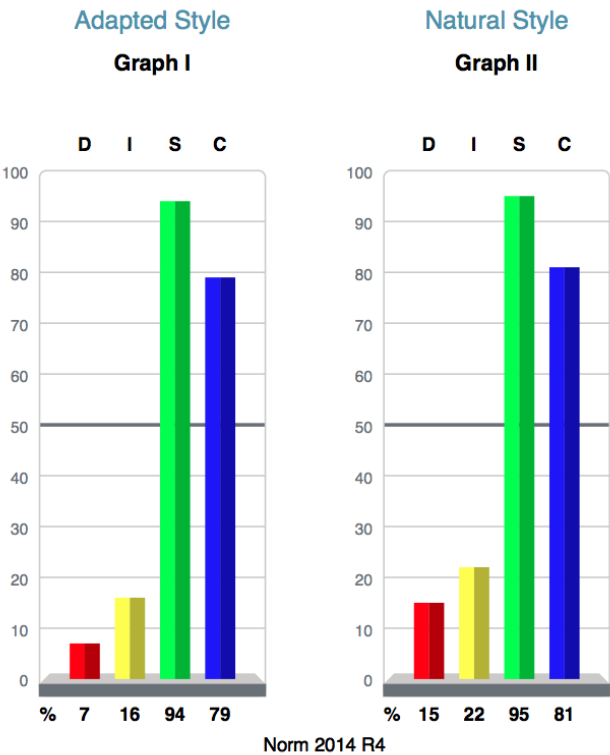


Figure 3. Word Descriptor Chart.

Demanding Egocentric Driving Ambitious Pioneering Strong-Willed Forceful Determined Aggressive Competitive Decisive Venturesome Inquisitive Responsible	Effusive Inspiring Magnetic Political Enthusiastic Demonstrative Persuasive Warm Convincing Polished Poised Optimistic Trusting Sociable	Phlegmatic Relaxed Resistant to Change Nondemonstrative Passive Patient Possessive Predictable Consistent Deliberate Steady Stable	Evasive Worrisome Careful Dependent Cautious Conventional Exacting Neat Systematic Diplomatic Accurate Tactful Open-Minded Balanced Judgment
Dominance	Influencing	Steadiness	Compliance
Conservative Calculating Cooperative Hesitant Low-Keyed Unsure Undemanding Cautious Mild Agreeable Modest Peaceful Unobtrusive	Reflective Factual Calculating Skeptical Logical Undemonstrative Suspicious Matter-of-Fact Incisive Pessimistic Moody Critical	Mobile Active Restless Alert Variety-Oriented Demonstrative Impatient Pressure-Oriented Eager Flexible Impulsive Impetuous Hypertense	Firm Independent Self-Willed Stubborn Obstinate Opinionated Unsystematic Self-Righteous Uninhibited Arbitrary Unbending Careless with Details

QEEG / sLORETA Findings

Eyes-open qEEG with sLORETA revealed a marked alpha current-density increase in BA9 (dorsolateral prefrontal cortex), approximately ~4 SD above age-normed means in resting state. Interpretively, this focal alpha elevation was treated as nontrivial given BA9’s role in cognitive–affective integration and regulatory control. Medication status (sertraline) at the time of testing was noted as a possible physiological confound; while normative comparisons were conducted against population data, no sertraline-matched subgroup analyses were available. Selective serotonin reuptake inhibitors have been reported to increase frontal alpha power in resting EEG, necessitating caution in attributing the observed elevation solely to psychopathology.

Gamma Asymmetry (Emotional Valence)

The gamma asymmetry approach employed here derives from the guided imagery valence evaluation (GIVE) process described by Bonnstetter et al. (2018) and further developed by Collura and Bonnstetter (2020). It assumes that lateralized gamma band activity between 38–42 Hz indexes preconscious affective reactions to stimuli. This

model posits that right-dominant gamma reflects aversive/avoidant valence, whereas left-dominant gamma reflects approach/positive valence, with symmetrical patterns indicating neutral responses. These assumptions extend traditional alpha asymmetry theories into higher-frequency bands; however, independent peer-reviewed replication is limited, and alternative frameworks (e.g., beta asymmetry or theta connectivity) exist for indexing emotional processing. Therefore, the findings reported here should be interpreted within a provisional conceptual model rather than as definitive evidence of latent emotion.

Frontal gamma asymmetry patterns were used to infer valence direction: right-frontal > left associated with aversive/avoidant, left-frontal > right with approach/positive, and symmetry with neutral responses. Across stimuli:

Directionality was stable across both runs; the second run showed reduced intensity consistent with habituation, but valence direction was unchanged, as shown in Figure 5.

Figure 4. Eyes-Open Brain Functional Analyses Showing 3D sLORETA Findings Indicating a Maximum Increased Alpha Current Density in Brodmann Area 9.

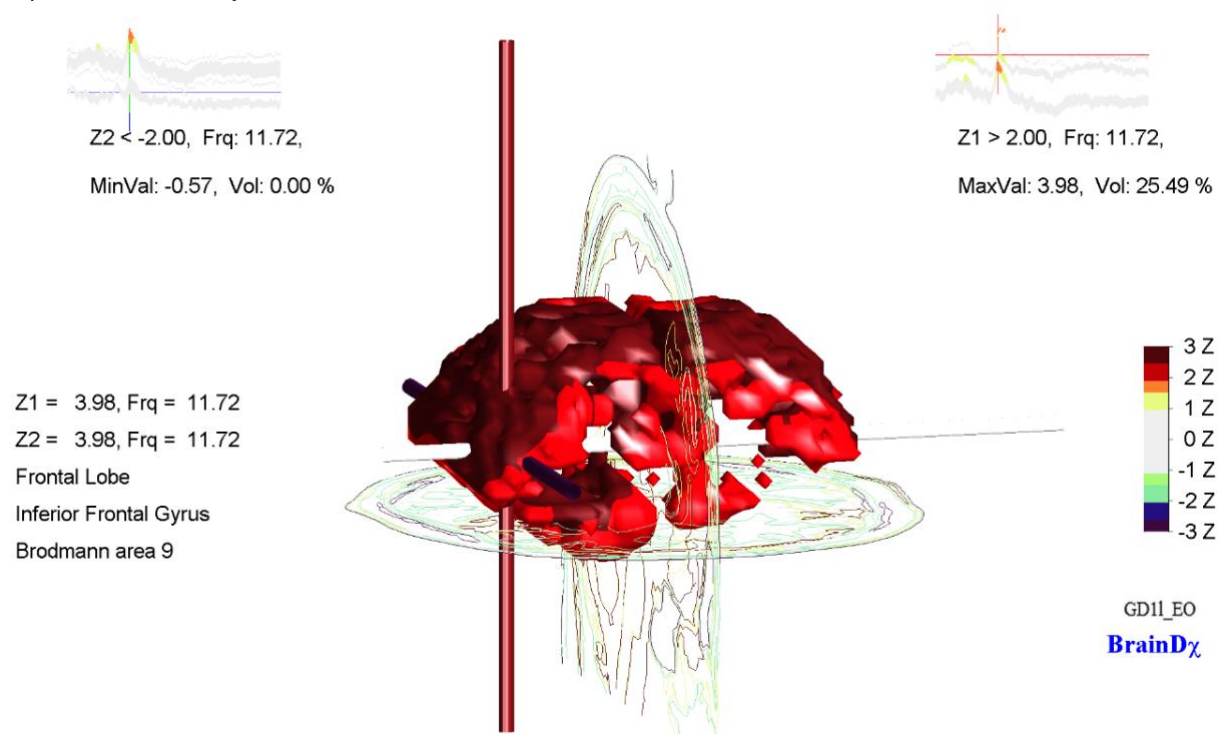


Figure 5. First Run Baseline Image Second Run Baseline Image.

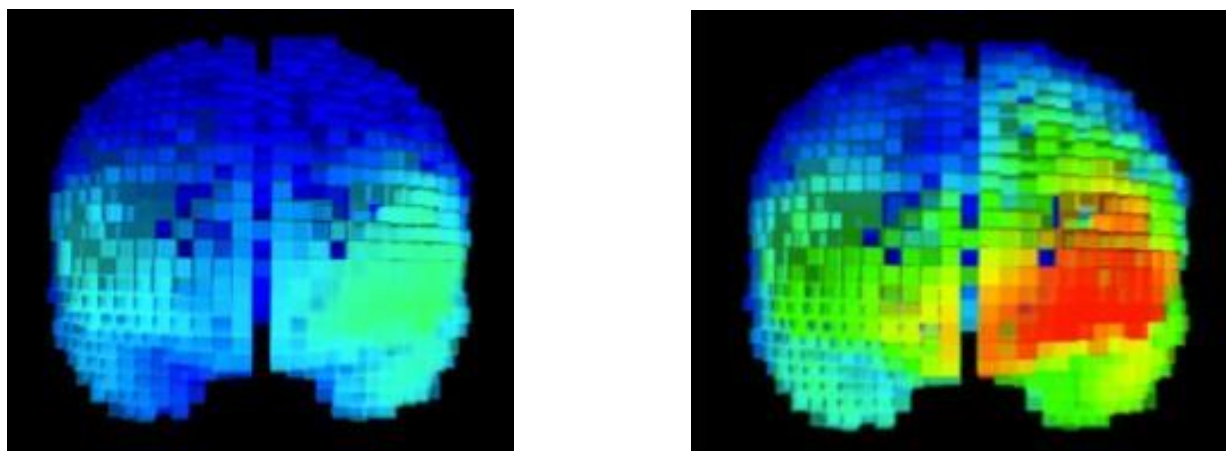


Figure 5 depicts this habituation, even in the client's baseline status; yet in both runs, the client exhibited a left-dominant asymmetry. The image of the frontal cortex is facing the observer, so the red is on the left side of the brain.

Baseline images in both runs showed left-dominant asymmetry. In the lab's broader experience set, less than 10% of more than 200 clients exhibit left-dominant baselines; when present, it has frequently

been observed among individuals reporting meditation practice or elevated positive affect at recording. In the present case, sertraline (Zoloft) may also have contributed to baseline left dominance.

Paper-and-pencil ratings showed few extreme responses (only three items at the most extreme end), broadly mirroring the moderated EEG reactivity.

To quantify the magnitude of asymmetry responses, gamma asymmetry indices were calculated for each stimulus as the normalized difference between left and right frontal gamma power. Across stimuli, effect sizes (Cohen’s *d*) relative to a normative sample of more than 200 adults averaged 0.9 for aversive triggers and 0.4 for positive triggers, indicating large and moderate departures, respectively. Paired comparisons between baseline and stimulus conditions showed statistically significant differences in asymmetry direction (Wilcoxon signed-rank tests, *p* < .05), although these analyses remain exploratory given the single-case design.

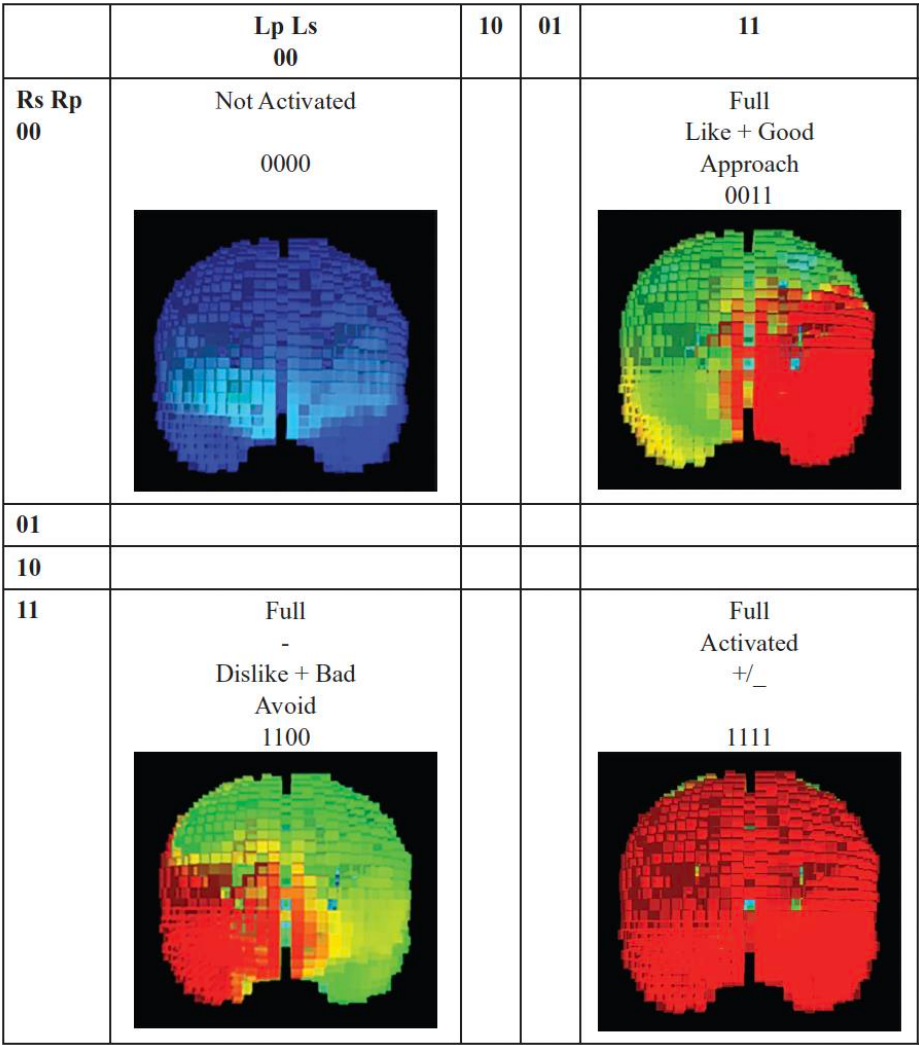
Updated Display Protocols (Post Hoc Visualization)

Since the gathering of the initial EDF file from the defendant in 2016, an additional gamma asymmetry

emotional model has been developed that takes into account the presence of moment-by-moment decision-making processing, referred to as microstates (Bonnstetter & Collura, 2020; Bonnstetter et al., 2022; Collura & Bonnstetter, 2020), and is employed on that original data set to expand on our understanding of the defendant’s responses.

Employing the protocols of gamma asymmetry described by Bonnstetter and colleagues (2018), Figure 6 depicts the four corners of the model as sLORETA (standardized low-resolution brain electromagnetic tomography) images and how the upper left-hand corner with no activation can move through a continuum down to avoidance, across to approach or diagonally to full activation.

Figure 6. Simplified Emotional Decision Model.



The top row labels represent left frontal lobe primary and left secondary brain gamma (38-42 Hz). The first column represents the right frontal lobe and again depicts the secondary and primary possible responses. Regarding the numbers: 0 = *no activation* and 1 = *activation*.

This simplified version of the model that provided an introduction to the 16-cell model by elaborating on the four extremes. The upper left corner shows low or no activation as represented by both right and left numeric zeros (0000). The upper right corner shows an initial primary emotion (Like) and a secondary judgment (Good) in the left hemisphere and no response in the right (0011). The lower left depicts an initial emotion of dislike and a judgment of bad as shown by the right hemisphere gamma response and the lack of left hemisphere reaction (1100). The lower right-hand corner depicts full activation (1111).

While the 4×4 decision model and microstate analysis provided an illustrative framework for parsing complex gamma patterns, it is important to note that microstate segmentation of gamma activity remains experimental in forensic contexts. Simpler metrics, such as average asymmetry indices across time windows, may offer comparable discriminative power with fewer assumptions. Future research should directly compare microstate-based and traditional asymmetry methods to determine whether the added complexity yields meaningful improvements in forensic prediction.

Frontal Gamma Asymmetry as a Forensic Tool

Table 2 displays the resulting s-LORETA image, the corresponding trigger word or phrase, the defendant's Likert score paper and pencil response and the final decision as defined by the 4x4 model in Figure 6. For brevity, only the most useful images are shown.

Figure 7. Resulting 16-Cell Decision-Making Model.

		Lp Ls			
		00	10	01	11
Rs Rp	00	NOT ACTIVATED 0000	PRIMARY + "Like" PLEASANT 0010	SECONDARY + "Good" SAFE 0001	FULL + "Like + Good" APPROACH 0011
	01	PRIMARY - "Dislike" UNPLEASANT 0100	PRIMARY +/- "Suspend Feeling" 0110	PRIMARY- SECONDARY+ "Dislike" + "Good" (Dieting) 0101	PRIMARY+/- SECONDARY+ + "Mixed Feeling" + "Good" FOLLOW HEAD 0111
	10	SECONDARY- "Not Good" UNSAFE 1000	PRIMARY+ SECONDARY- "Like" + "Not Good" (NAUGHTY) 1010	SECONDARY +/- "Suspend Judgment" 1001	PRIMARY+ SECONDARY+/- "Like" + "Mixed Judgment" FOLLOW HEART 1011
	11	FULL "Dislike" + "Bad" AVOID 1100	PRIMARY+/- SECONDARY- "Mixed Feeling" + "Bad" FOLLOW HEAD 1110	PRIMARY- SECONDARY+/- "Dislike" + "Mixed Judgment" FOLLOW HEART 1101	FULL ACTIVATED +/- 1111

Note. Emotional decision model. Rs, right secondary; Rp, right primary; Lp, left primary; Ls, left secondary.

Table 2
Resulting sLORETA Images

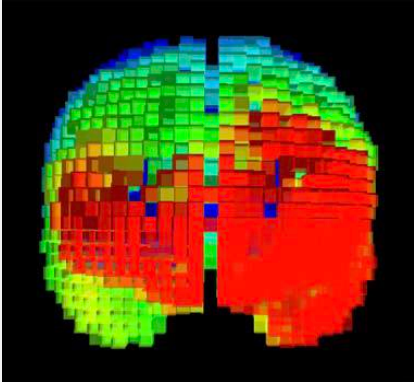
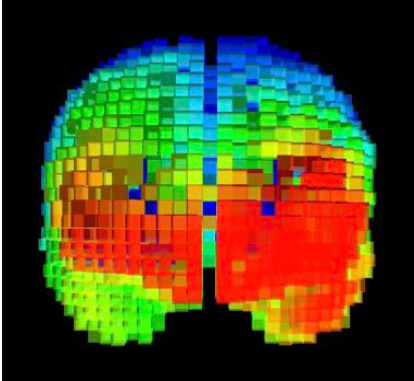
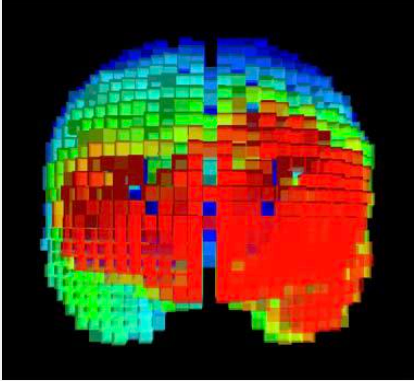
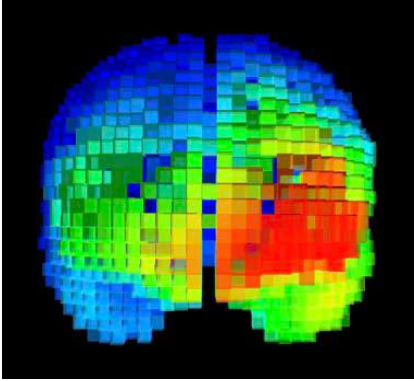
Image	Trigger	Likert Score	Resulting 4x4 Cell
	Mother's name	5	0111 Mixed Feeling + Good
	Uncle is coming at me	2	1110 Mixed feeling + Bad
	Threats to mother	2	0111 Mixed Feeling + Agree
	Panic attacks	1	0100 Dislike

Table 2
Resulting sLORETA Images

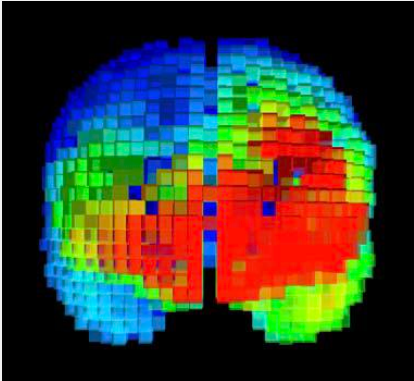
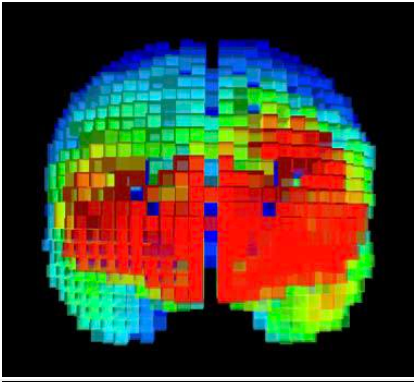
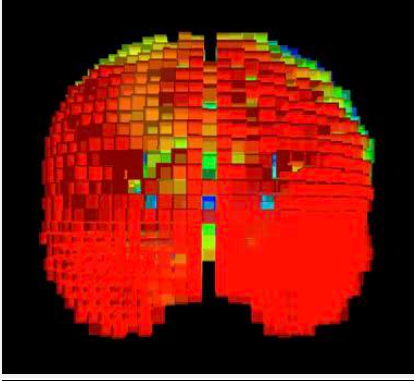
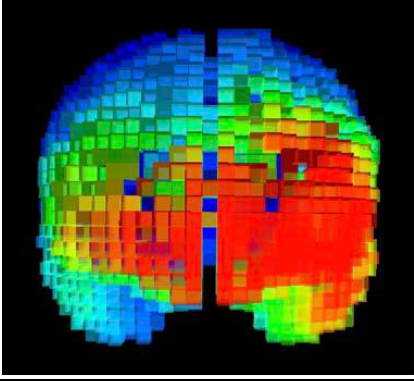
Image	Trigger	Likert Score	Resulting 4x4 Cell
	Self-guilt	3	0110 Mixed Feeling
	Caring for parents	6	0110 Mixed Feeling
	Name of victim	6	1111 Full Activation
	Gardening	4	0110 Mixed Feeling + Bad

Table 2
Resulting sLORETA Images

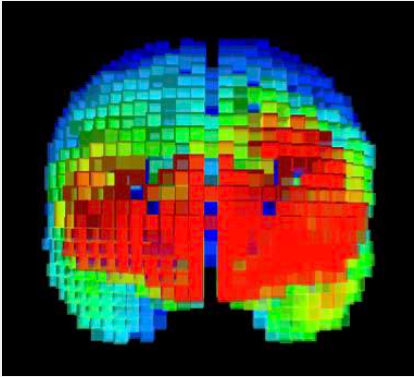
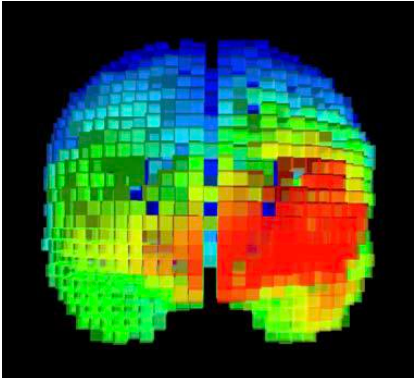
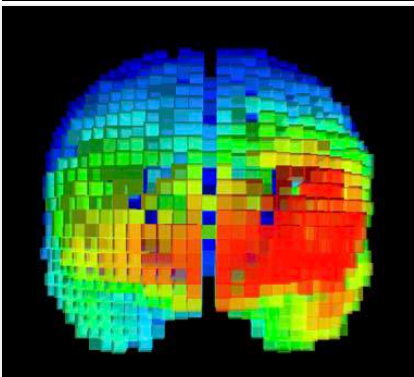
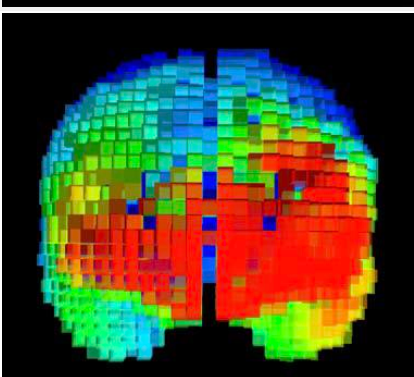
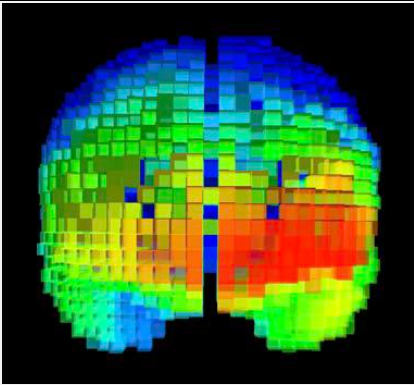
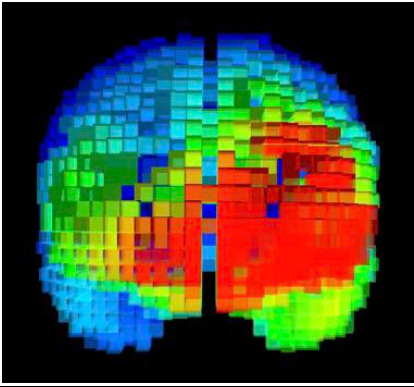
Image	Trigger	Likert Score	Resulting 4x4 Cell
	Caring for parents	4	0111 Mixed Feeling + Good
	Worthlessness	3	0111 Mixed Feeling + Agree
	Protecting mother	4	0111 Mixed Feeling + Good
	Uncle is coming for mother	1	0111 Mixed Feeling + Agree

Table 2
Resulting sLORETA Images

Image	Trigger	Likert Score	Resulting 4x4 Cell
	Protecting family	5	0110 Mixed Feeling + Agree
	Uncle is dead	5	0111 Mixed Feeling + Good

Discussion

The integration of frontal gamma asymmetry using quantitative EEG (qEEG) and current density source localization (sLORETA) provides a novel and compelling method for evaluating emotional processing in forensic contexts, particularly in determining the presence or absence of mens rea, or “guilty mind,” in murder cases. In this case study, a structured and repeatable protocol enabled the direct measurement of emotional responses to highly relevant stimuli, offering unique insight into the defendant’s internal affective states at the time of the offense. This approach holds significant implications for forensic neuropsychology and legal decision-making.

The results suggest that measures of frontal gamma asymmetry are not only sensitive to emotional valence but may serve as reliable indicators of unconscious or automatic affective responses tied to significant people, memories, or perceived threats. In particular, stimuli involving the victim elicited right frontal gamma asymmetry patterns indicative of an aversive response, while references to parental

figures elicited more mixed or positive left-sided activation. These neurophysiological signatures were consistent with the defendant’s reported history of protectiveness toward his mother, chronic emotional distress, and reactive traits shaped by environmental trauma and familial conflict. The convergence of these findings with self-report data and behavioral assessments enhances the ecological and construct validity of this forensic methodology (Bonnstetter et al., 2012; Collura et al., 2014a, 2014b).

Interestingly, discrepancies emerged between self-report and informant-based measures on instruments such as the BRIEF-A and ASR: the defendant rated his executive functioning and internalizing symptoms as average, whereas informants described significant difficulties. Such mismatches may reflect limited insight, intentional minimization, or contextual differences between testing environments and daily functioning. These discrepancies underscore the importance of integrating collateral information and highlight how neural metrics may capture implicit affective processes not fully acknowledged in self-report.

Moreover, the documentation of increased alpha activity in BA9 during resting-state EEG suggests a potential neurophysiological substrate for compromised emotional regulation and decision-making. BA9 has been implicated in integrating emotional signals with cognitive control, including reappraisal, inhibition of impulsive behavior, and moral reasoning (Etkin et al., 2011; Goldin et al., 2008; Ochsner & Gross, 2005). Heightened alpha in this region may reflect cortical disengagement or inhibitory gating. However, given evidence that selective serotonin reuptake inhibitors can elevate frontal alpha power, it is important to interpret this finding as correlational and multifactorial rather than as direct evidence of a dual-system breakdown.

The co-occurrence of elevated BA9 alpha power and atypical gamma asymmetry patterns raises the possibility of interactions between top-down and bottom-up processes, but causal mechanisms cannot be established from a single case. High alpha power may attenuate gamma bursts (Scheeringa et al., 2011), thereby influencing emotional processing; conversely, chronic stress, trauma history, and medication effects may also contribute to these patterns. Therefore, any model positing a dual system failure—impaired top-down inhibition via alpha-related disengagement and disorganized bottom-up affective processing via gamma dysregulation—remains speculative. Future research with larger samples and controlled pharmacological conditions is required to clarify these relationships.

This excessive alpha band current density in BA9, nearly four standard deviations increase from the age normed mean in a resting-state condition, is not trivial. Firstly, the alpha frequency band of the EEG, typically defined as oscillatory activity between 8–12 Hz, has long been recognized as more than just a passive indicator of cortical idling. Contemporary research demonstrates that alpha rhythms play an active and organizing role in the regulation of information processing across cortical networks. Rather than reflecting disengagement, alpha oscillations are now understood to support functional inhibition, selectively gating neural resources and modulating the flow of information across brain regions (Jensen & Mazaheri, 2010).

One of the most well-established findings is that increased alpha power in a specific region often correlates with the suppression of task-irrelevant processing, while reduced alpha power is associated with enhanced excitability and readiness in task-

relevant regions. For instance, during tasks requiring spatial attention, alpha power increases over parietal-occipital regions contralateral to the unattended visual field, acting as a “neural filter” to inhibit distractions (Foxe & Snyder, 2011). This illustrates how alpha activity is dynamically modulated to organize perception and attention based on behavioral demands. Furthermore, alpha-band synchrony has been implicated in top-down control mechanisms.

Studies using magnetoencephalography (MEG) and EEG show that long-range alpha coherence between prefrontal and posterior cortices is predictive of task performance and cognitive control, supporting the hypothesis that alpha rhythms facilitate large-scale coordination of brain networks (Palva & Palva, 2007). These findings suggest that alpha oscillations help stabilize cortical representations by coordinating when and where information flows during decision-making and working memory operations. Additionally, intracranial and EEG studies have shown that alpha oscillations often precede and predict successful stimulus detection, encoding, and memory retrieval. This temporal alignment implies that alpha rhythms may serve a timing or pacing function, organizing neural activity into cycles that allow for efficient processing of sensory and mnemonic information (Klimesch, 2012).

Importantly, BA9 situated in the dorsolateral prefrontal cortex, plays a central role in the integration of emotional information with cognitive processes. Far from being confined to abstract reasoning or working memory, BA9 is consistently engaged during tasks that require individuals to evaluate, regulate, or act upon emotional stimuli. Its function appears to be critical when emotions must be consciously modulated or weighed in relation to decision-making outcomes (Ochsner & Gross, 2005). Neuroimaging studies have repeatedly shown that BA9 becomes activated during cognitive reappraisal—the process through which individuals reinterpret emotionally charged stimuli to reduce their psychological impact. For example, functional MRI investigations have demonstrated that individuals who successfully downregulate negative affect tend to show heightened activity in right BA9 (Goldin et al., 2008). This suggests that the region is actively recruited to suppress or reshape the salience of negative emotional responses.

Moreover, volumetric studies have revealed that individuals with greater gray matter volume in this region exhibit stronger reappraisal abilities,

indicating a structural basis for its role in emotion regulation (Beauregard et al., 2001). Beyond emotion regulation, BA9 is involved in the processing of emotionally relevant information during decision-making tasks. When subjects are faced with risky or emotionally laden choices, BA9 appears to mediate the cognitive strategies that support weighing emotional consequences against long-term goals (Etkin et al., 2011). It is not merely a passive relay but an active processor that helps determine the balance between immediate emotional reactions and deliberate executive functions. In this sense, BA9 acts as a neural fulcrum between impulsivity and foresight (Ochsner & Gross, 2005). Additionally, studies show that BA9 participates in self-reflective emotional processes. When individuals are instructed to introspect about their own emotional states—asking themselves how they feel or why they reacted emotionally—BA9 shows increased activation (Bzdok et al., 2011). This role in self-focused emotional appraisal highlights the region's involvement not only in top-down regulation of emotion but also in the conscious interpretation of internal affective experiences.

Finally, mood induction studies have shown that the function of BA9 is mood sensitive. For example, during working memory tasks, BA9 activity can be enhanced or suppressed depending on whether the participant is in a negative or neutral emotional state, respectively. This illustrates how emotional context influences the recruitment of BA9 even during tasks traditionally considered “cold” cognition (Etkin et al., 2011).

Together, this body of evidence supports the conclusion that BA9 is a critical hub for integrating affective and cognitive processes. It supports the ability to downregulate negative emotion, evaluate emotional consequences, and reflect upon internal emotional states—all of which are essential for adaptive decision-making and emotional resilience. In the context of *mens rea*, these findings are significant. Legal determinations of criminal intent often hinge on subjective interpretations of emotional state and motive, which are notoriously difficult to substantiate without external corroboration. The application of EEG-based neurofunctional imaging offers an objective, repeatable, and noninvasive means of validating or challenging claims of temporary insanity, trauma-induced emotional disturbance, or dissociation at the time of an alleged crime. By revealing preconscious emotional reactions to specific stimuli, gamma asymmetry mapping complements traditional forensic interviews and psychometric tools, providing

a multidimensional picture of internal state (Bonnstetter et al., 2018; Collura et al., 2016). Furthermore, the refined 4x4 emotional decision model used in this study allows for nuanced categorization of responses—such as mixed feelings with positive or negative judgments—which could be highly informative in legal cases where dichotomous labels like “intentional” or “accidental” may oversimplify complex affective processes. The emotional data obtained via EEG microstates and gamma asymmetry not only substantiates the defendant's reported psychological state but also provides temporal markers of affective reactivity that align with known microstate transitions involved in decision-making (Bonnstetter et al., 2022).

Importantly, this case also highlights key methodological considerations for future applications. Pharmacological influences, such as the subject's use of Zoloft, may modulate baseline neural activity and should be accounted for when interpreting EEG patterns (Goldin et al., 2008). Likewise, habituation effects noted during repeated stimuli exposure suggest the need to limit exposure windows to preserve the emotional salience of trigger stimuli.

While preliminary and limited by its single-case design, this work highlights both the promise and the constraints of integrating neuroscience into forensic practice. Broader applications may extend to evaluating emotional regulation, motive, or credibility, but any suggestion of “emotional lie detection” should be framed cautiously as a potential future research direction rather than an established forensic tool. Ethical considerations and generalizability to diverse populations must also be addressed before such methods can be applied more widely. By demonstrating the feasibility of combining psychological and neurophysiological measures, this study contributes to ongoing interdisciplinary efforts to develop more objective approaches to understanding affect in legal contexts.

Conclusion

Limitations and Ethical Considerations

This evaluation reflects data from a single individual assessed in one setting. There was no behavioral follow-up or replication across sessions, and normative comparisons were based on laboratory samples rather than large epidemiological cohorts. The subject was taking sertraline, which may modulate alpha and gamma activity, and medication-specific norms were unavailable.

Consequently, generalizability is limited, and causal inferences cannot be made. Future research should include larger samples, medication-controlled comparisons, and longitudinal designs.

The application of neural measures in legal contexts also raises important ethical considerations. While EEG-derived metrics may reduce reliance on subjective testimony, there is a risk of overinterpreting ostensibly “objective” data without sufficient empirical validation. Courts must consider the admissibility standards for neuroscientific evidence and the potential for misuse or misinterpretation. Neuroscientific measures should complement rather than replace traditional legal assessments until robust replication and normative databases are established.

In conclusion, our findings suggest that EEG-derived frontal gamma asymmetry and alpha power distribution may hold promise as tools in forensic evaluations of mens rea. Nevertheless, these measures remain experimental and should not be construed as definitive evidence of emotional states or intent. Given the single-case design, medication confounds, and proprietary nature of the gamma protocol, broader forensic utility must await independent replication and multisite validation studies. Neuroscientific evidence should be presented transparently alongside conventional psychological assessments, and its admissibility should be weighed against evolving legal standards and ethical considerations. Until a robust evidence base is established, neurophysiological metrics should complement, not replace, traditional forensic methods.

Author Disclosures

Thomas Collura is the founder and an owner of BrainMaster Technologies, Inc. Ronald Bonnstetter is Executive Director of the Mind Science Center and Vice President of Research for Target Training International. This project was not supported by any grant. AI was not used in the creation of the content in this manuscript. Authors accept responsibility for the accuracy of reported data and findings.

References

- Arns, M., Etkin, A., Hegerl, U., Williams, L. M., DeBattista, C., Palmer, D. M., Fitzgerald, P. B., Harris, A., deBeuss, R., & Gordon, E. (2015). Frontal and rostral anterior cingulate (rACC) theta EEG in depression: Implications for treatment outcome? *European Neuropsychopharmacology*, 25(8), 1190–1200. <https://doi.org/10.1016/j.euroneuro.2015.03.007>
- Beauregard, M., Lévesque, J., & Bourgoin, P. (2001). Neural correlates of conscious self-regulation of emotion. *The Journal of Neuroscience*, 21(18), Article RC165. <https://doi.org/10.1523/JNEUROSCI.21-18-j0001.2001>
- Bonnstetter, B. J., Bonnstetter, R. J., Hebets, D., & Collura, T. F. (2015). *Behavioral and cognitive profile assessment system* (U.S. Patent No. 9,060,702). U.S. Patent and Trademark Office.
- Bonnstetter, B. J., Bonnstetter, R., Hebets, D., & Collura, T. F. (2016). *Validation process for ipsative assessments* (Canadian Patent No. 2,808,691). Canadian Intellectual Property Office.
- Bonnstetter, R., & Collura, T. (2020). Brain activation imaging in emotional decision making and mental health: A review—Part 1. *Clinical EEG and Neuroscience*, 52(2), 98–104. <https://doi.org/10.1177/1550059420916636>
- Bonnstetter, R. J., Collura, T., Hebets, D., & Bonnstetter, B. J. (2012). Uncovering the belief behind the action. *NeuroConnections*, Winter, 20–23.
- Bonnstetter, R. B., Collura, T., Zalaquett, C., & Wang, H. (2022). EEG microstates in relation to emotional decision-making. In D. R. Chartier, M. B. Dellinger, J. Evans, & H. K. Budzynski (Eds.), *Introduction to quantitative EEG and neurofeedback: Advanced applications* (3rd ed., pp. 219–234). Elsevier Academic Press.
- Bonnstetter, R. J., Gehrig, E., & Hebets, D. (2018). Response process validation protocol using neurophenomenological gamma asymmetry. *NeuroRegulation*, 5(3), 93–102. <https://doi.org/10.15540/nr.5.3.93>
- Bonnstetter, R. J., Hebets, D., & Wigton, N. L. (2015). Frontal gamma asymmetry in response to soft skills stimuli: A pilot study. *NeuroRegulation*, 2(2), 70–85. <https://doi.org/10.15540/nr.2.2.70>
- Bzdok, D., Langner, R., Caspers, S., Kurth, F., Habel, U., Zilles, K., & Eickhoff, S. B. (2011). ALE meta-analysis on facial judgments of trustworthiness and attractiveness. *Brain Structure and Function*, 215(3–4), 209–223. <https://doi.org/10.1007/s00429-010-0287-4>
- Cantor, D. (2016). Integration of frontal gamma asymmetry as a forensic tool for emotional valence in a murder case. Presentation at *Proceedings of the 18th World Congress of Psychophysiology (IOP2016) of the International Organization of Psychophysiology (IOP)*, 108, 65.
- Chow, R. T. S., Yu, R., Geddes, J. R., & Fazel, S. (2025). Personality disorders, violence and antisocial behaviour: Updated systematic review and meta-regression analysis. *The British Journal of Psychiatry*, 227(1), 471–491. <https://doi.org/10.1192/bjp.2024.226>
- Collura, T. F., & Bonnstetter, R. J. (2020). Brain activation imaging in emotional decision making and mental health: A review part 2. *Clinical EEG and Neuroscience*, 52(2), 105–113. <https://doi.org/10.1177/1550059420916642>
- Collura, T. F., Wigton, N. L., Zalaquett, C., Chatters, S., & Bonnstetter, R. J. (2016). The value of EEG-based electromagnetic tomographic analysis in human performance and mental health. *Biofeedback*, 44(2), 72–80.
- Collura, T. F., Zalaquett, C., & Bonnstetter, R. J. (2014a). Seeing inside the client's mind. *Counseling Today*, 57(6), 24–27.
- Collura, T. F., Zalaquett, C., Bonnstetter, R. J., & Chatters, S. (2014b). Toward an operational model of decision making, emotional regulation, and mental health impact. *Advances in Mind-Body Medicine*, 28(4), 18–33.
- Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition and Emotion*, 12(3), 307–330. <https://doi.org/10.1080/026999398379628>
- Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences*, 15(2), 85–93. <https://doi.org/10.1016/j.tics.2010.11.004>
- Ford, J. D., & Courtois, C. A. (2021). Complex PTSD and borderline personality disorder. *Borderline Personality*

- Disorder and Emotion Dysregulation*, 8(1), Article 16. <https://doi.org/10.1186/s40479-021-00155-9>
- Foxe, J. J., & Snyder, A. C. (2011). The role of alpha-band brain oscillations as a sensory suppression mechanism during selective attention. *Frontiers in Psychology*, 2, Article 154. <https://doi.org/10.3389/fpsyg.2011.00154>
- Gehrig, E., & Bonnstetter, R. (2021). *Styles Insights Technical Manual* (Version 1.0). Target Training International, Ltd.
- Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry*, 63(6), 577–586. <https://doi.org/10.1016/j.biopsych.2007.05.031>
- Jensen, O., & Mazaheri, A. (2010). Shaping functional architecture by oscillatory alpha activity: Gating by inhibition. *Frontiers in Human Neuroscience*, 4, Article 186. <https://doi.org/10.3389/fnhum.2010.00186>
- Klimesch, W. (2012). Alpha-band oscillations, attention, and controlled access to stored information. *Trends in Cognitive Sciences*, 16(12), 606–617. <https://doi.org/10.1016/j.tics.2012.10.007>
- Knott, V., Telner, J., Lapierre, Y., Browne, M., & Horn, E. (1996). Quantitative EEG in the prediction of antidepressant response to imipramine. *Journal of Affective Disorders*, 39(3), 175–184. [https://doi.org/10.1016/0165-0327\(96\)00003-1](https://doi.org/10.1016/0165-0327(96)00003-1)
- Lehmann, D., Ozaki, H., & Pal, I. (1987). EEG alpha map series: Brain micro-states by space-oriented adaptive segmentation. *Electroencephalography and Clinical Neurophysiology*, 67(3), 271–288. [https://doi.org/10.1016/0013-4694\(87\)90025-3](https://doi.org/10.1016/0013-4694(87)90025-3)
- Leuchter, A. F., Cook, I. A., Hunter, A. M., Cai, C., Horvath, S. (2012). Resting-state quantitative electroencephalography reveals increased neurophysiologic connectivity in depression. *PLoS ONE*, 7(2), Article e32508. <https://doi.org/10.1371/journal.pone.0032508>
- Nourse, V. F. (2002). Hearts and minds: Understanding the new culpability. *New Criminal Law Review*, 6(1), 361–402.
- Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences*, 9(5), 242–249. <https://doi.org/10.1016/j.tics.2005.03.010>
- Palva, S., & Palva, J. M. (2007). New vistas for alpha-frequency band oscillations. *Trends in Neurosciences*, 30(4), 150–158. <https://doi.org/10.1016/j.tins.2007.02.001>
- Pascual-Marqui, R. D. (2002). Standardized low-resolution brain electromagnetic tomography (sLORETA): Technical details. *Methods and Findings in Experimental and Clinical Pharmacology*, 24(Suppl. D), 5–12.
- Scheeringa, R., Mazaheri, A., Bojak, I., Norris, D. G., & Kleinschmidt, A. (2011). Modulation of visually evoked cortical fMRI responses by phase of ongoing occipital alpha oscillations. *The Journal of Neuroscience*, 31(10), 3813–3820. <https://doi.org/10.1523/JNEUROSCI.4697-10.2011>
- Teicher, M. H., & Samson, J. A. (2016). Annual research review: Enduring neurobiological effects of childhood abuse and neglect. *Journal of Child Psychology and Psychiatry*, 57(3), 241–266. <https://doi.org/10.1111/jcpp.12507>

Received: November 28, 2025

Accepted: March 14, 2026

Published: September 28, 2026