NeuroRegulation

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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

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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., cardiovagal 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

A Priori Protocol (Operational Summary) With Deviations/Notes

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 (*n*)", "Minutes per session", "Withdrawals (NF/Control)"; AE terms aligned to each trial's definitions.
Effect measures	SMD (Hedges' g) 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 Q, I^2, τ^2 and 95% prediction interval (PI) when $k \geq 3$ (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, *SD*s, 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

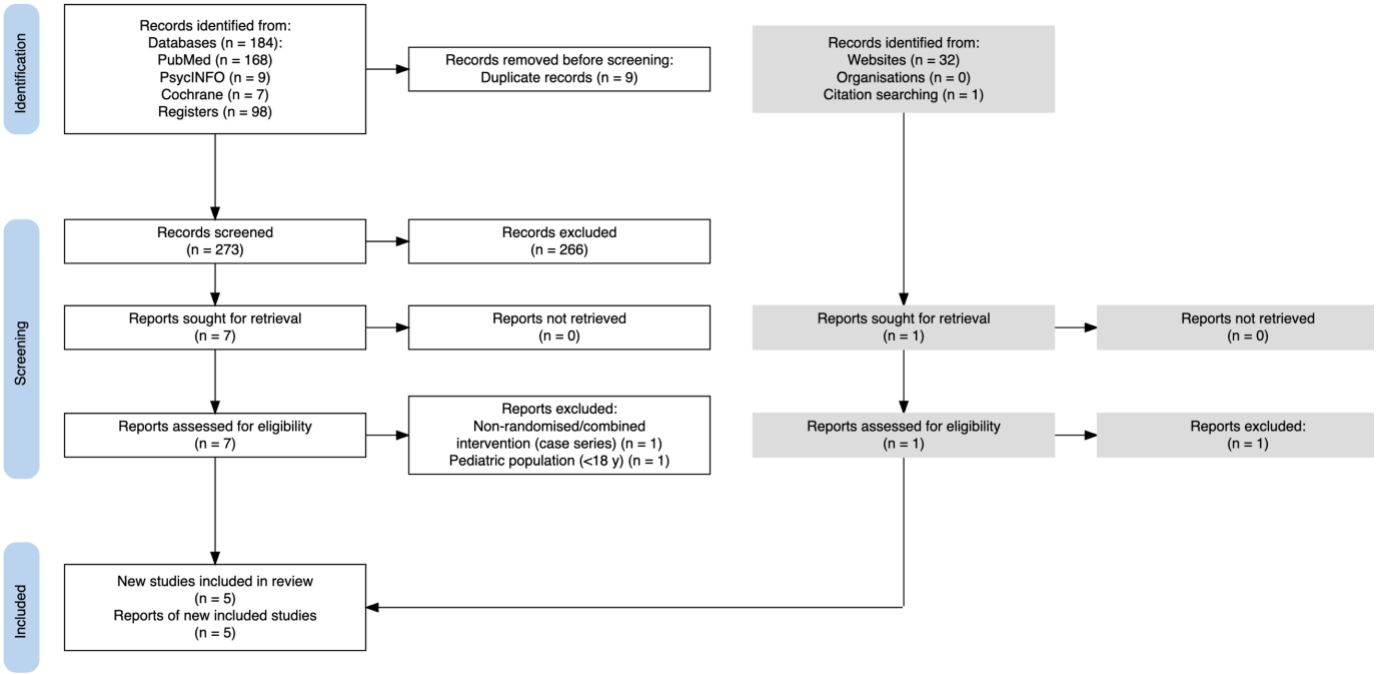
The review used public sources and did not require ethics approval. A priori planning, verbatim documentation of search strategies (PRISMA-S), and recognized tools (RoB 2, GRADE) ensure transparency and auditability (Page et al., 2021; Rethlefsen et al., 2021). All relevant methodological decisions—derivation 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.

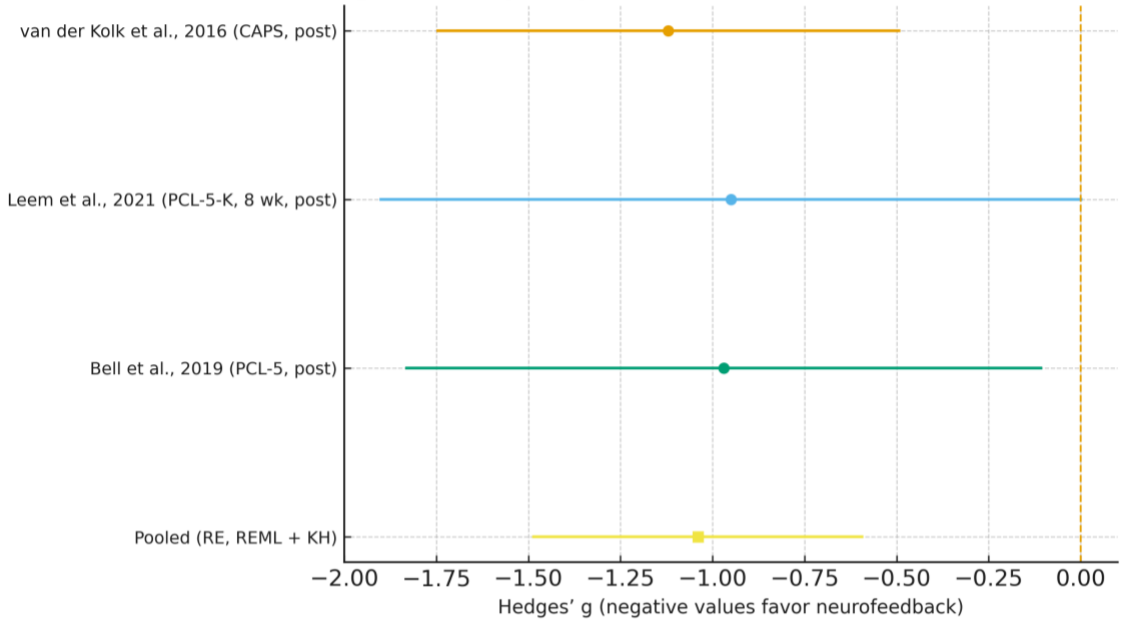
Table 5

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.

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