Parsons *Discover Neuroscience* (2026) 21:21
https://doi.org/10.1186/s13064-026-00252-x

Discover Neuroscience

PERSPECTIVE Open Access

Rethinking control conditions in clinical neurofeedback trials

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Abstract
Double-blind, placebo-controlled sham neurofeedback (sNFB) trials are often regarded as the methodological gold standard for evaluating neurofeedback efficacy. However, this paradigm rests on a false equivalence with pharmacological models of intervention. Like genuine neurofeedback (NFB) and unlike inert placebos, sNFB provides structured sensory feedback—including both contingent and non-contingent reinforcement—that engages core learning mechanisms such as operant conditioning. This feature means that sham conditions are not behaviorally or physiologically inert but partially active, undermining their role as true controls. Moreover, the double-blind design is incompatible with a second essential mechanism of neurofeedback: voluntary self-regulation. To bypass this methodological limitation, research practices distance themselves from clinical standards, and in doing so reduce the active effects belonging to NFB. Together, this misalignment leads to systematic underestimation of neurofeedback's specific effects and to misinterpretation of null findings. Drawing on parallels from psychotherapy and learning-based neuroscience, this paper argues for a reconceptualization of control design in neurofeedback research. Some suggestions for a revised framework are proposed to allow for the separation of specific from non-specific mechanisms without invoking an impossible inert placebo. Adopting this approach can realign the appraisal of neurofeedback efficacy with the principles appropriate to it as an interactive and adaptive intervention.

Keywords Neurofeedback, EEG biofeedback, Placebo, Active control, Double-blind

1 Introduction

Over the past two decades, research on neurofeedback has expanded rapidly, reflecting its growing role within applied neuroscience and clinical neurotechnology. Yet, methodological approaches have not always kept pace with this growth. Neurofeedback occupies a distinctive position between neuroscience, psychology, and behavioral learning, requiring an understanding of both neural dynamics and experiential self-regulation.

Despite accumulating empirical evidence demonstrating efficacy and specificity, neurofeedback continues to be framed by some as a sophisticated placebo rather than an active intervention [1–7]. This view has shaped experimental design and interpretation across the field, often leading to the adoption of sham neurofeedback (sNFB) as a

gold-standard placebo control under double-blind conditions. However, the assumption that sham feedback is inert and that the double-blind is achievable overlooks two fundamental features of neurofeedback: its basis in operant conditioning and its reliance on voluntary self-regulation.

This paper examines how the placebo-controlled double-blind framework, when applied to neurofeedback, introduces systematic conceptual and methodological errors. It argues that the sham model does not isolate specific effects but instead compares two partially active interventions, while the attempt to achieve a double-blind reduces the active components of genuine neurofeedback (NFB). Finally, it proposes some alternatives towards an evaluative framework better suited to interactive, learning-based brain–behavior paradigms.

2 Scope and definitions

Neurofeedback research spans heterogeneous aims and outcome domains, and the appropriateness of a control condition depends critically on the causal claim under test. The present perspective focuses primarily on clinical neurofeedback trials, defined here as intervention studies in which the primary dependent variables are clinical symptoms, functional outcomes, or wellbeing, and where the principal inference concerns therapeutic benefit. This scope is distinguished from mechanistic neurofeedback studies, in which the primary dependent variables are neural measures (e.g., modulation of a trained EEG feature, fMRI activation, or connectivity) and the central aims are target engagement and the testing of brain–behavior models.

Within this framework, sham neurofeedback (sNFB) refers to procedures that preserve the form of neurofeedback (credible instructions and feedback display in comparable sensory modalities) while removing real-time contingency between the participant's own neural activity and the feedback signal (e.g., prerecorded EEG, synthetic signals, or randomized feedback). Although sNFB is designed to remove the intended real-time neural contingency, in practice many implementations can retain partial or intermittent overlap between a participant's ongoing brain state and the delivered reward signal (e.g., through thresholding rules, reinforcement density, and task-linked state fluctuations), as discussed later in this paper. At the same time, sNFB preserves multiple active ingredients that are likely to influence learning, arousal, motivation, expectancy, and self-regulatory effort. Accordingly, in clinical trials sNFB is often better conceptualized as a partially active control rather than an inert placebo.

3 Boundary conditions for mechanistic studies

Although the emphasis of this article is on clinical outcome trials, similar interpretive issues can arise in mechanistic studies depending on the inference sought. In target-engagement designs, sNFB can be an informative comparator when, and only when, the loss of real-time contingency is empirically demonstrated (i.e., the feedback signal is effectively decoupled from the participant's concurrent neural dynamics). In such cases, sham conditions can help isolate protocol-specific neural mechanisms by matching instructional set, sensory feedback, and task engagement while selectively removing the closed-loop link that defines neurofeedback. A practical implication is that studies should quantify the degree of residual contingency by concurrently recording the participant's true EEG (or other neural signal) while false or prerecorded feedback is delivered

and then calculating how often the delivered reward coincides with target-state occurrences under the same thresholding and reinforcement rules, as outlined later in this paper.

However, sNFB is not necessarily inert at the level of cognition and neurophysiology: it can recruit attention and control processes and may induce indirect, network-level changes that are not captured by a single trained metric. Contemporary causal accounts therefore encourage bounded interpretation: protocol-specific neural change supports causal relevance, while behavioral change may be mediated by direct and indirect pathways within broader causal networks. Accordingly, the suitability of sham and blinding should be evaluated with explicit reference to the study aim, the dependent variables, and the intended causal inference.

4 How did we get here?

The widespread use of sNFB as a placebo control emerged from the longstanding dominance of the double-blind, placebo-controlled randomized clinical trial (RCT) in biomedical research. Within this framework, the failure of NFB to consistently outperform sNFB has often been interpreted as evidence that NFB lacks specific therapeutic effects. However, this paper does not address those outcome-based critiques directly; detailed responses to such arguments have been well-documented elsewhere (e.g. [8]). Instead, it examines the deeper methodological assumption underlying those interpretations: namely, that the sham paradigm is appropriate.

While the placebo model is considered highly suited to pharmacology, where an inert control condition can meaningfully isolate drug-specific effects, its application to neurofeedback introduces conceptual and mechanistic errors. First, sham feedback is not biologically inactive but delivers structured, sensory input that engages learning and self-regulatory processes central to neurofeedback itself. Next, the use of double-blind limits the active components of the genuine NFB intervention.

This paper therefore re-evaluates the methodological foundation of the sham-controlled paradigm and argues that it is fundamentally incompatible with the active, interactive mechanisms that define neurofeedback.

5 The randomized, double-blind, placebo-controlled model

The double-blind, placebo-controlled RCT remains the cornerstone of biomedical research, particularly in pharmacology, where it is used to isolate the specific effects of an intervention from the many non-specific influences that shape outcomes. By ensuring that the active treatment and placebo are indistinguishable except for the presence of the therapeutic agent, this design allows investigators to attribute between-group differences to the intervention itself, rather than to expectation, time, or context [9].

When applied to neurofeedback, this logic assumes that the only distinction between the experimental and control groups is the presence or absence of brain-contingent feedback. The neurofeedback (NFB) condition is presumed to provide the "active ingredient," while the sham neurofeedback (sNFB) condition serves as the "inert placebo". It is also assumed that a double-blinding can be achieved. These assumptions, however, are both conceptually and mechanistically problematic.

As will be demonstrated, sham feedback is not a passive control but a structured sensory input stream that can engage learning. The double-blind restricts the development

of voluntary self-regulation. These two processes are central to neurofeedback's efficacy [10].

Even within its native domain of pharmacology, the placebo-controlled paradigm is not without limitations. Seminal critiques have highlighted how double-blind placebo-controlled RCT designs can produce systematic biases and fail to capture complex interactions between expectation, context, and biological response [11, 12]. These concerns are magnified in interventions that depend on continuous feedback, active participation, and skill acquisition. Neurofeedback exemplifies this challenge: its mechanisms are interactive and adaptive, not passive, static or dose-dependent.

As illustrated in Fig. 1, the traditional pharmacological model isolates the specific effect of an active agent by subtracting inert placebo effects from total outcome variance. In contrast, neurofeedback's active mechanisms, operant conditioning and voluntary self-regulation, cannot be subtracted without distorting the intervention itself. The double-blind placebo-controlled model, when transferred uncritically from pharmacology to neurofeedback, thus creates a methodological paradox: an experimental design that eliminates the very processes it seeks to measure.

5.1 Specific and non-specific effects

In clinical research, *specific effects* refer to outcomes that arise directly from the active components of an intervention, whereas *non-specific effects* encompass all other influences that may shape symptom change. This includes but is not limited to: expectation, therapeutic context, and spontaneous recovery. The placebo effect represents only one subset of these broader non-specific influences.

In pharmacology, this distinction is relatively straightforward: the specific effect is tied to a well-defined molecular agent whose mechanism of action can be isolated experimentally. The placebo, in contrast, is inert. It is identical in form but devoid of pharmacological activity. This allows researchers to quantify drug-specific variance with reasonable confidence [9].

In psychological and behavioral interventions, however, the boundary between specific and non-specific effects becomes blurred. Factors once dismissed as ancillary – such as therapist engagement, attention, or perceived agency—are now recognized as central mechanisms of change. As Enck and Zipfel [13] observe, "the current standards for drug trials, e.g., true placebo interventions [and] double-blinding, cannot be applied to most psychotherapy techniques," and further, some non-specific effects in pharmacology "have very specific effects in psychotherapy." While some consider neurofeedback a

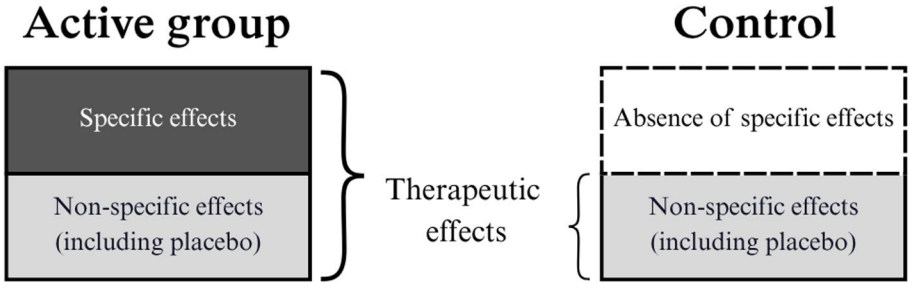

Fig. 1 Conceptual breakdown of therapeutic effects in a double-blind placebo-controlled RCT. Note: The placebo effect is just one component of non-specific effects. Bar sizes of specific and non-specific effects are equal for illustration purposes only

form of technology-assisted psychotherapy [14], this paper does not aim to settle that debate. What matters is that the same limitations in separating specific and non-specific effects in psychotherapy also apply to neurofeedback, regardless of how it is classified.

Yet within neurofeedback research, non-specific effects are frequently conflated with placebo response alone, leading to oversimplified and false interpretations of null findings. Expectation, attention, observation, and therapist-participant interaction are all legitimate non-specific contributors that operate in both active and sham conditions [15, 16]. Recognizing their role does not diminish neurofeedback's validity but clarifies why traditional placebo designs cannot cleanly separate specific and contextual effects.

Table 1 summarizes common non-specific factors across clinical research domains. As illustrated, these influences are neither negligible nor undesirable—they represent integral elements of human learning and therapeutic engagement. The challenge, therefore, is not to eliminate them, but to design studies that distinguish specific mechanisms without erasing the ecological validity of the intervention.

For neurofeedback, this means adopting experimental models that account for the full spectrum of active processes rather than treating non-specific effects as methodological noise. Such a shift reorients efficacy research from an outdated pharmacological paradigm toward one suited to dynamic, participant-driven brain–behavior interventions.

5.2 Neurofeedback

Applying a placebo-controlled model to neurofeedback may appear reasonable: the intervention is technologically mediated, involves quantifiable inputs, and can be standardized across participants. Yet this analogy with pharmacology collapses under closer examination. Neurofeedback is not a passive administration of a fixed dose but an adaptive learning process driven by continuous feedback and active self-regulation. Proper neurofeedback methodology involves linking a functional target to a specific brain activity (frequency, amplitude, location) and the modulation of these parameters by a clinician based on experiential self-regulation.

Neurofeedback is typically defined as an operant conditioning procedure in which individuals learn to modulate specific patterns of brain activity—most often recorded

Table 1 Common non-specific effects in clinical research

Non-specific effect	Brief description
Placebo effect	Symptom improvement due to belief in the treatment, independent of its specific action.
Nocebo effect	Worsening of symptoms due to negative expectations about the treatment.
Hawthorne effect	Behavioral or symptom changes resulting from the awareness of being observed.
Observer bias	Researcher expectations influence how outcomes are measured or interpreted.
Expectation bias	Participant expectations shape perceived outcomes or symptom reporting.
Attention effect	Increased attention from clinicians or researchers contributes to improvement.
Social desirability bias	Participants respond in ways they believe are socially acceptable or expected.
Demand characteristics	Participants modify their behavior based on perceived study goals or researcher cues.
Therapeutic alliance	Symptom improvement resulting from the strength of the patient-provider relationship.
Natural recovery	Symptoms improve over time due to the condition's natural course, independent of treatment.
Regression to the Mean	Extreme symptoms tend to revert toward average levels over time, independent of treatment.

A partial list of non-specific effects in clinical research

with EEG—through real-time sensory feedback. When target activity is detected, reinforcing stimuli (e.g., visual or auditory cues) are delivered; when it deviates, feedback is withheld. Over repeated trials, participants learn to associate internal states with external cues and to sustain the desired neural pattern. Sessions are typically guided by a trained clinician who adjusts thresholds, modalities, and strategies based on observed and subjective responses—an iterative process central to both research and clinical practice [17].

While operant conditioning represents the foundation of neurofeedback, it is only one component of its active mechanism. A second, equally essential element is *voluntary self-regulation*. Early research demonstrated that participants are not merely conditioned by reinforcement schedules but can intentionally reproduce specific brain states once they learn to recognize them. Sterman's work in the 1960s showed that animals trained to increase sensorimotor rhythm (SMR) activity developed resistance to chemically induced seizures, illustrating direct neurophysiological adaptation through reinforcement. Beyond basic conditioning, Sterman's work hinted that animals were learning to actively generate desired brain states [18]. Around the same time, Kamiya demonstrated that human participants could voluntarily modulate alpha activity even in the absence of real-time feedback, underscoring the cognitive and intentional dimension of the process [19].

Together, these foundational studies established neurofeedback as a hybrid mechanism that combines operant conditioning and conscious self-regulation. The relative contribution of each likely depends on protocol, target population, and training strategy, but both are significant contributors to outcome. This dual mechanism stands in direct conflict with the assumptions of traditional double-blind placebo-controlled RCT designs, which presuppose that active (NFB) and sham (sNFB) conditions differ only by the presence of an inert element. In neurofeedback, by contrast to pharmacology, engagement, feedback, and volition are inseparable components of the treatment itself—making an "inert" sham condition conceptually impossible. In that regard, neurofeedback more closely resembles psychotherapy.

5.3 Active neurofeedback (NFB): the experimental group

In pharmacological research, the experimental group receives the active compound thought to produce therapeutic effects. In neurofeedback, by contrast, the "active" intervention is not a passive administration but a dynamic learning process. Participants receive real-time sensory feedback derived from their own brain activity, which guides them in modifying specific neural patterns through reinforcement and intentional control.

A typical session involves continuous interaction between participant and system: EEG signals are recorded, transformed into visual or auditory cues, and presented in near-real time. When the targeted brain activity occurs, reinforcing stimuli are delivered (positive reinforcement); when it deviates, feedback is withheld (reinforcement withdrawal; negative punishment). Across repeated trials, guided by a clinician, participants learn to recognize internal cues associated with successful modulation and to intentionally reproduce those states.

The essential feature of the active condition, therefore, is contingency: the direct, moment-to-moment relationship between the participant's neural activity and the

feedback received. This contingency simultaneously engages two active mechanisms: operant conditioning and voluntary self-regulation. It is this dual contingency that defines neurofeedback as an *active* intervention and that cannot be meaningfully replicated by sham feedback designs.

5.3.1 A note on thresholds

A defining feature of the neurofeedback paradigm is the *threshold*: the criterion at which feedback is triggered (reinforcement) or withheld (punishment). In a protocol designed to reward beta activity, for instance, reinforcement may occur when beta amplitude exceeds a specified value (e.g., 5 μV), and punishment occurs (via reinforcement withdrawal) when it falls below that level.

To improve standardization across participants and groups, many studies adopt automatic thresholds, in which reinforcement is calibrated to occur for a fixed proportion of time – commonly 80% – independent of actual performance. Although this approach enhances experimental consistency, it introduces a critical methodological artifact: it decouples reinforcement from behavioral or neurophysiological effort. Participants who actively succeed in self-regulation and those who remain passive ultimately receive identical reinforcement ratios, despite markedly different levels of control.

This procedural simplification effectively weakens the defining mechanism of neurofeedback, the *contingency* between brain activity and feedback, reducing the learning process to a pseudo-random reinforcement schedule. Such detachment undermines both the operant and volitional components of training and calls into question the ecological validity of results obtained under these conditions. While auto-thresholding satisfies the statistical symmetry sought in controlled trials, it simultaneously obscures the very causal link that neurofeedback is designed to measure, illustrating how methodological convenience can inadvertently erode mechanistic fidelity.

5.4 Sham neurofeedback (sNFB): the active control group

Understanding the dynamics of the sNFB condition is essential, as it is assumed to represent an inert placebo; the equivalent of an empty capsule in pharmacological research. In sNFB, participants are exposed to what appears to be real-time contingent feedback, but the EEG signal driving the procedure is not derived from real-time brain activity. Instead, it may consist of prerecorded EEG data, a synthetic signal, or randomized activity. The sensory modalities used for feedback (typically visual, auditory, or tactile) are identical to those used in the active condition.

Critically, participants are unaware that the feedback is not driven by their own brain activity. Believing the signal reflects their own neural activity, they engage in genuine attempts at self-regulation, deploying attention and effort in line with training instructions. The result is a paradox: although the sham condition is intended to serve as an inert control, it remains behaviorally and psychologically active. Participants still recruit the very cognitive and motivational processes that neurofeedback aims to harness.

This point is supported empirically by Ninaus et al., who used an fMRI sham-neurofeedback paradigm in which participants believed they were receiving genuine neurofeedback while the visual feedback was in fact based on a recording rather than their own brain activity. Despite the absence of true contingency, participants remained credibly engaged and, relative to passive viewing, showed increased activation in a broad

cognitive control and salience-related network, including bilateral anterior insula, anterior cingulate cortex, supplementary motor area, and dorsomedial/lateral prefrontal regions. In other words, the *belief* that one is training, combined with the structured feedback context, is sufficient to recruit neurocognitive systems that are plausibly relevant to neurofeedback learning and to non-specific clinical change—underscoring that sNFB is better understood as a partially active control rather than an inert placebo [20].

Consequently, sNFB cannot be considered a true inert placebo. It delivers structured sensory stimulation within a credible training context, prompting engagement and expectation, conditions known to influence learning and brain activity. By mimicking the form of neurofeedback while severing real contingency, the sham design preserves many active ingredients of the intervention, making it a *partially active control* rather than an inert comparator.

5.5 Non-specific elements shared between NFB and sNFB groups

At a procedural level, participants in both the NFB and sNFB conditions undergo nearly identical experiences. Each receives identical amounts of feedback, identical psychoeducation, and equivalent provider interaction within an indistinguishable technical setup. These design features are intended to control for non-specific factors such as attention, expectation, and therapeutic alliance. Yet it is precisely within these apparent similarities that a critical methodological problem emerges.

Under common auto-threshold protocols (e.g.: [21]), both groups receive positive reinforcement for 80% of each session and experience negative punishment for the remaining 20%. In the active NFB condition, this feedback is fully contingent on the participant's own neural activity. In the control sNFB condition, feedback follows the same 80/20 schedule but is driven by an unrelated, supposedly non-contingent signal.

The assumption in both paradigms is that every aspect of the intervention is equivalent, except for the active ingredient: contingent reinforcement. Therefore, in both the NFB group and the sNFB group, the participant is in the target state 80% of the time and is generating the target brain activity 80% of the time. The NFB group receives active training (80% contingent reinforcement), and the sNFB group a supposedly inert substitute.

Simply put, the math doesn't work. The sNFB schedule yields a mixed feedback profile due to chance overlap between the sNFB subject being in the correct state (80%) and producing the correct brain activity (80%) to get contingent feedback. To illustrate, consider the breakdown of feedback using auto-thresholds for the sNFB group based on simple averages: this yields 64% (80% of 80%) contingent reinforcement (correct reinforcement), 16% (80%-64%) non-contingent reinforcement (incorrect reinforcement), 4% (20% of 20%) contingent punishment (correct punishment), and 16% (20%-4%) non-contingent punishment (incorrect punishment). As summarized in Table 2, an estimated 68% of feedback events in the sNFB condition remain functionally contingent, engaging operant mechanisms similar to those in true neurofeedback. The remaining 32% consists of non-contingent feedback that introduces inconsistency but not inertness. Note that when auto-thresholding is not used, these calculations will vary, for example, based on the final amount of reinforcement and punishment provided.

The assumption that the sNFB feedback is wholly non-contingent is thus false. The sNFB condition constitutes a *partially contingent active intervention*, not an inert

Table 2 Comparison of feedback types in NFB and sNFB trials

Feedback type	Definition	NFB (%)	sNFB (%)	Feedback state
Contingent reinforcement	Feedback ON when in target state	80%	64%	Correct reward
Non-contingent reinforcement	Feedback ON when in non-target state	0%	16%	Incorrect reward
Contingent punishment	Feedback OFF when in non-target state	20%	4%	Correct withdrawal
Non-contingent punishment	Feedback OFF when in target state	0%	16%	Incorrect withdrawal
Operant (Contingent) feedback	Sum of contingent reinforcement + punishment	100%	68%	Specific/active feedback
Non-operant (Random) feedback	Sum of non-contingent reinforcement + punishment	0%	32%	Non-specific or inconsistent

Percentages assume a fixed 80/20 feedback schedule using auto-thresholds. Values for sNFB are estimated based on a uniform distribution of feedback unrelated to the subject's brain state

placebo in the pharmacological sense. Because participants remain cognitively engaged and the system continues to deliver structured, temporally meaningful reinforcement, the sham condition reproduces much of the operative mechanism it aims to control. The result is a conceptual and methodological paradox: the very design intended to isolate specific neurofeedback effects systematically preserves them.

One could even argue that while the traditional NFB paradigm utilises a continuous reinforcement schedule that facilitates learning, the sNFB paradigm accidentally provokes a discontinuous reinforcement schedule which has long been known to slow learning, but protects against extinction [22]. A planned transition from continuous reinforcement during acquisition to intermittent reinforcement during later training could be used to consolidate learning and increase its persistence. This highlights that what is accidental in sNFB may, in part, resemble a strategy that can be beneficial when applied deliberately.

5.6 Differential non-specific elements between NFB and sNFB groups

Beyond their shared structural and procedural similarities, the neurofeedback (NFB) and sham neurofeedback (sNFB) conditions also differ in the *type* and *distribution* of feedback events, with important consequences for behavioral and physiological engagement. In the sNFB condition, approximately 16% of feedback events constitute *non-contingent reinforcement*: feedback delivered while the participant is in a non-target state. Although often treated as random noise, non-contingent reinforcement can itself produce measurable behavioral effects. Studies in applied behavior analysis for developmental disorders, including ADHD, have shown that non-contingent reinforcement following random reinforcement schedules can modify behavior and maintain engagement even in the absence of explicit contingency [23]. Thus, this component of the sham condition may exert genuine though non-specific therapeutic influence.

A further 16% of feedback events represent *non-contingent punishment*, where participants are in the target state but receive no feedback. From a reinforcement learning perspective, such inconsistency introduces partial unpredictability. Paradoxically, this variability can sustain responding and motivation by preventing rapid extinction—a phenomenon well established in both animal and human learning research [24].

Together, these two forms of non-contingent feedback account for roughly one-third of the total stimulation received in the sham condition. Far from being inert, they engage the same motivational and attentional systems that contribute to neurofeedback's

therapeutic action, though in a slightly altered manner. Consequently, the sNFB condition functions not as a passive placebo but as a subtly active behavioral training environment—further eroding the distinction between control and treatment.

5.7 Box 1 Illustrative examples linking study aim to control choice

The implications of sham neurofeedback depend on the causal claim, the dependent variables under investigation, and the extent to which "sham" is truly decoupled from participants' concurrent neural dynamics. The brief examples below illustrate how control conditions can be tailored to different aims while avoiding overgeneralized interpretations.

Example 1: Clinical efficacy trial where sham is a partially active control

Aim: Evaluate whether neurofeedback improves patient-relevant outcomes.

Design: A randomized trial in ADHD using an SMR-based protocol with outcomes focused on symptom ratings and functional impairment.

Interpretation issue: In sham neurofeedback, participants typically receive the same training context and feedback display and believe they are attempting self-regulation. Even if intended contingency is reduced, participants still engage attention, effort, and expectancy processes that can influence symptoms and functioning. Depending on implementation, additional overlap between ongoing brain state and reward delivery may occur through thresholding rules and state-linked fluctuations, further limiting the assumption that sham is inert.

Design implication: A null or small difference between active neurofeedback and sham does not uniquely imply lack of efficacy, because both arms may contain therapeutically relevant ingredients and may differ in ways that are not captured by simple active versus sham labels (e.g., differences in perceived control, frustration, or strategy exploration). When the primary question is clinical benefit, strengthen interpretability by measuring expectancy, perceived agency over the feedback, and engagement repeatedly across sessions, and by explicitly quantifying the degree of contingency retained in the sham condition.

Example 2: Mechanistic target engagement study where sham can isolate the closed-loop component

Aim: Test whether a protocol produces modulation of a specified neural feature and whether that modulation transfers to behavior.

Design: Healthy participants train theta-range synchrony during a working-memory task, with the primary neural outcome defined a priori as task-state modulation of the targeted theta feature (rather than resting baseline alone), plus transfer to working-memory performance.

Interpretation issue: In such designs, sham can be informative when and only when the feedback signal is empirically shown to be decoupled from participants' concurrent neural dynamics. Without this verification, residual or random contingency can confound the inference that only the active arm had target engagement.

Design implication: Concurrently record the true neural signal while delivering false feedback and quantify residual contingency, then interpret group differences in the trained metric and transfer measures in light of these estimates. Where feasible, include a yoke or replay design (one participant's true feedback drives another's sham feedback) to preserve realistic reward dynamics while removing self-contingency.

Example 3: Hybrid clinical and mechanistic trial requiring layered inference
Aim: Determine whether neurofeedback improves symptoms and engages the intended neural target, and whether target engagement mediates clinical change.
Design: A PTSD trial combining symptom outcomes with neuroimaging-based measures of network function, alongside active neurofeedback and sham neurofeedback arms, including pre–post fMRI validation of target-network modulation (for an example, see [25]).
Interpretation issue: Sham may be adequate for testing whether the trained neural target changes (target engagement), yet still be a partially active control for symptoms through expectancy, effort, therapist contact, and structured feedback. Moreover, neurofeedback effects are often state-dependent and not necessarily one-to-one with the trained metric: for example, protocols that inhibit alpha during training or task engagement can be associated with post-training alpha increases during subsequent resting-state recordings, consistent with compensatory or rebound dynamics and broader network reconfiguration. Thus, symptom improvement in sham does not invalidate target engagement findings, nor does target engagement guarantee symptom change.
Design implication: Pre-specify primary and secondary outcomes, treat target engagement and symptom change as separable questions, and define in advance the measurement context for neural outcomes (e.g., during training, during an active task, and at rest). When protocols are individualized or clinically adaptive, predefine allowable adjustment rules and report them transparently so that "treatment as delivered" can be compared across arms. Consider analyses that test mediation and state-dependence, alongside designs that strengthen causal inference (e.g., dismantling approaches that isolate specific training elements).
Cross-cutting reporting and manipulation checks
To avoid overinterpretation and to make control conditions comparable across studies, trials should report (i) the exact thresholding and reinforcement rules, including whether thresholds are fixed, auto-adjusted, or manually titrated; (ii) an estimate of feedback contingency in each arm (including residual contingency in sham) based on concurrent recording; (iii) participant expectations, perceived agency over feedback, and guesses about group assignment; and (iv) engagement and strategy use, ideally captured repeatedly across sessions. These elements clarify what the control condition actually controlled for and help distinguish null results due to inadequate target engagement from null results due to genuinely equivalent active ingredients.
Implications for blinding in neurofeedback trials
In neurofeedback, the central mechanism is closed-loop learning embedded in an interactive, adaptive clinical process, in ways that closely resemble other complex behavioral health interventions such as psychotherapy. For many clinical protocols, strict double-blinding is therefore not only difficult but conceptually mismatched to the intervention: maintaining provider blinding can require ignoring clinically meaningful information about whether feedback is coherent with the participant's state, or refraining from the iterative calibration that constitutes competent delivery. In this sense, double-blinding can distort "treatment as delivered" by constraining responsiveness (threshold adjustment, coaching, parameter refinement) and by converting a dynamic learning intervention into a rigid procedure.
Accordingly, trials should specify which level of blinding is appropriate for the causal question (e.g., participant-blind, assessor-blind, analyst-blind) and treat "double-blind" as an empirical design choice with trade-offs rather than a universal gold standard. As in psychotherapy research, where double-blinding of both therapist and participant is generally inappropriate, rigor is typically achieved through alternative safeguards rather than by attempting to render the intervention relationally inert. When full double-blinding is inappropriate, rigor can be preserved through alternative safeguards, including blinded outcome assessors with prespecified analysis plans, standardized instructions and interaction scripts, and role separation between intervention staff and assessors. In addition, trials should report allowable clinical adaptations and fidelity to them and include manipulation checks assessing expectancy, perceived agency, and guesses of group assignment. These approaches reduce bias while respecting the epistemic structure of an adaptive, learning-based treatment.

6 The "Pill" isn't empty

The central problem with so-called placebo-controlled neurofeedback trials is that they do not employ a true placebo and therefore cannot isolate purely non-specific effects. Instead, they juxtapose two partially active interventions, each engaging attentional focus, reinforcement-based learning, and intentional self-regulation. In effect, such designs compare neurofeedback against a diluted form of itself rather than against an inert control.

In pharmacological research, the logic of the placebo rests on chemical inertness: an empty pill that is indistinguishable from the active drug but devoid of pharmacological activity. This design allows researchers to attribute between-group differences to the specific molecular mechanism of the intervention. When the drug and placebo yield similar

outcomes, the result implies that observed benefits arise from factors related to expectation (placebo) or natural remission (other non-specific) rather than the compound itself.

In neurofeedback, however, to apply this logic is fundamentally flawed. Sham feedback is not inert; it remains behaviorally and psychologically active, delivering structured sensory stimulation and maintaining participant engagement. Thus, when an NFB condition fails to outperform sNFB, the result does not indicate the absence of specific mechanisms but rather the absence of statistical difference in outcome between two active paradigms. Such comparisons obscure, rather than clarify, the true efficacy and mechanisms of neurofeedback.

6.1 Is placebo the right term?

The designation of sham neurofeedback (sNFB) as a placebo is conceptually inaccurate. In classical biomedical research, a *placebo* is defined as an intervention that produces beneficial outcomes through expectancy or contextual factors rather than through any intrinsic physiological activity [25]. Two criteria must be met for an intervention to qualify as a true placebo:

1. The participant expects a therapeutic benefit, and.
2. The intervention itself is inert; that is, incapable of eliciting a specific physiological effect.

The sNFB paradigm satisfies the first criterion. Participants typically anticipate improvement, and the structure of the intervention (multiple sessions, professional supervision, and intensive engagement) reinforces this expectation. However, sNFB fails the second criterion. It is not inert. As discussed earlier, the sham condition delivers structured sensory feedback that includes both contingent and non-contingent reinforcement, and engages operant learning processes. Participants remain cognitively and behaviorally active throughout, often investing effort comparable to that of the genuine NFB condition.

In pharmacological research, the placebo is passive and satisfies the second requirement: an inert pill administered on a fixed schedule, requiring no skill, interaction, or learning. Neurofeedback, by contrast, is inherently active, relying on dynamic feedback loops between brain and behavior. The sham condition preserves a significant portion of these same loops, albeit with altered contingencies.

Thus, labeling sNFB as a "placebo" conflates an *active cognitive-learning task* with an *inert comparator*, a categorical error that obscures both mechanism and meaning in the interpretation of trial results.

6.2 A broader perspective on placebo

Placebo effects are not unique to neurofeedback; they are ubiquitous across therapeutic modalities, including pharmacological, psychotherapeutic, and complementary interventions. Expectation-driven physiological and psychological responses are intrinsic to human healing processes and reflect adaptive features of neurobiology rather than methodological artifacts.

As Hammond [26] emphasizes, placebo mechanisms can be harnessed ethically and constructively in the context of neurofeedback. The presence of a placebo effect does not invalidate an intervention but underscores the need to distinguish specific mechanisms

from contextual contributions. The challenge for researchers, therefore, is not to eliminate placebo and other non-specific effects, which is neither possible nor desirable, but to integrate them appropriately into study design and interpretation.

In this sense, neurofeedback should be evaluated much as psychotherapy is: for its *total therapeutic impact*, encompassing both its specific learning-based mechanisms and its non-specific contextual influences. Recognizing the ubiquity of placebo effects reframes them not as confounds but as components of a complex biopsychosocial system. This broader understanding reinforces the central argument: sham feedback cannot serve as a valid placebo.

6.3 Can neurofeedback still demonstrate specificity?

A fair evaluation of neurofeedback requires determining whether its effects extend beyond expectancy and contextual influences. One way to disentangle these variables is through animal research, which eliminates much the cognitive expectation necessary for a placebo response. In controlled research designs, non-human subjects do not anticipate therapeutic outcomes. As such, they provide a direct test of physiological specificity: a logic long used to validate pharmacological interventions.

Early neurofeedback studies in animals provide clear evidence of specific, learning-based mechanisms. In a series of landmark experiments, Sterman and colleagues demonstrated that cats trained to enhance sensorimotor rhythm (SMR) activity exhibited markedly reduced susceptibility to chemically induced seizures [27]. Similar effects were later replicated in rhesus monkeys, strengthening the generalizability of the phenomenon [28]. Complementary work by Bauer and Jones [29] showed that gamma-band training in cats altered visual processing, indicating that operant modulation of neural oscillations can produce measurable changes in sensory and cognitive systems.

These findings offer evidence that neurofeedback's mechanisms operate independently of human belief or suggestion. The capacity to induce targeted neurophysiological adaptations in non-human subjects demonstrates that its active ingredients are at least partially embedded in the brain's intrinsic learning architecture, not in the expectations of the participant.

6.4 Protocol-specific effects support causal specificity

A further challenge to the placebo explanation arises from the protocol-specific nature of neurofeedback effects. If expectancy alone accounted for observed outcomes, different neurofeedback protocols should yield similar or randomly distributed results. Instead, convergent evidence from neuroimaging and electrophysiological studies indicates that distinct protocols produce targeted and replicable changes in neural function and structure.

For example, Levesque and colleagues [30] reported selective activation of attention networks following neurofeedback in children with ADHD. Ros et al. [31] observed modulation of the salience network after alpha rhythm training, while Nicholson et al. [32] demonstrated normalization of default mode network activity in individuals with post-traumatic stress disorder (PTSD). Additional structural and functional imaging studies have revealed protocol-dependent alterations in cortical thickness, connectivity, and network coherence across populations and training modalities [33–35].

These findings are incompatible with a purely expectancy-driven explanation. Instead, they indicate that neurofeedback engages distinct neural systems through targeted causal pathways that differ according to the feedback frequency, region, and behavioral context. The reproducibility of these protocol-specific effects across modalities and populations provides strong empirical support for the specificity, and by extension, the validity of neurofeedback as a therapeutic intervention.

Importantly, protocol-specific neural change supports causal relevance without requiring the stronger claim that the trained signal is the single, causally primary driver of the behavioral effect. Kvamme and colleagues argue that neurofeedback can demonstrate that a trained neural feature is non-spuriously related to behavior, yet the causal interpretation remains vulnerable to the possibility that training concurrently alters upstream or parallel brain dynamics that are themselves causally efficacious. On this view, the trained feature may be best conceptualized as one node within a broader causal network, such that protocol-specific effects reflect network-level reconfiguration rather than an isolated one-to-one mapping between a single frequency metric and a single cognitive outcome [36]. It would be reasonable to assume that in addition to these direct and indirect causal elements, that the non-specific active factors at play in the sNFB group (see [20]) also influence genuine neurofeedback outcome.

The authors further propose that stronger causal claims can be triangulated by converging evidence across methods and by designs that reduce strategic confounds (e.g., implicit or multivariate neurofeedback approaches), reinforcing the argument that neurofeedback can reveal specificity while encouraging appropriately bounded causal language [36].

7 What about the double-blind?

Another assumption of the double-blind, placebo-controlled RCT design is that the blinding of both participants and clinical researchers is achievable, without interfering with the active mechanisms of the NFB intervention.

In clinical practice, neurofeedback inherently involves iterative hypothesis testing. Protocols are continuously adjusted based on real-time neural data, behavioral observations, and client feedback. For instance, in a common ADHD training paradigm aimed at reducing the theta/beta ratio, the practitioner evaluates whether increasing beta and decreasing theta activity corresponds to observable improvements in attention and behavioral inhibition. If these changes fail to occur, the working hypothesis, in this case the application of a theta/beta protocol, is revised. This process repeats throughout treatment as the clinician adapts all aspects of training – including but not limited to active electrode sites, targeted metrics and reinforcement parameters, sensory feedback – to the individual's evolving neural and behavioral responses.

Such ongoing calibration renders the traditional double-blind model, in which neither participant nor provider knows the treatment condition, impossible to implement without compromising integrity. A skilled clinician can readily detect when feedback is non-contingent or incoherent with the client's state. To preserve blinding under these circumstances would require the provider to ignore evident mismatches between brain activity and feedback, effectively sustaining an invalid or counterproductive protocol.

Beyond methodological impracticality, this introduces ethical concerns. Maintaining blindness in the face of obvious inefficacy conflicts with professional responsibility and

may risk psychological or motivational harm, a point noted in the clinical literature [37, 38]. Once again, this feature of neurofeedback stands in stark contrast to pharmacological research, where a pill's inertness and uniform administration make blinding both feasible and ethical.

The inability to meaningfully blind neurofeedback interventions highlights their interactive, adaptive, and relational character. Like psychotherapy, neurofeedback relies on responsiveness, feedback, and alliance between practitioner and participant. These qualities are incompatible with strict double-blind procedures. These same methodological challenges have been recognized in broader mental health research. As a recent Cochrane review noted, "blinding of participants and personnel is a persistent issue in randomized trials with psychosocial interventions" [39]. The difficulty arises not from poor study design but from the nature of the intervention itself, human engagement cannot be rendered inert.

Thus, applying pharmacological standards of blinding to neurofeedback is not an expression of rigor but a category error: it violates the epistemic structure of a dynamic learning-based treatment. Recognizing this distinction allows for the development of ethically sound and methodologically coherent alternatives that respect the intervention's inherent adaptivity.

8 Discussion

There is broad consensus that neurofeedback research requires rigorous experimental control. However, the standard placebo-controlled model, designed for passive pharmacological interventions, is conceptually mismatched to an active and interactive technique such as neurofeedback.

8.1 The "gold standard"

Despite its conceptual limitations, the sham-controlled model remains the dominant framework for evaluating neurofeedback, even as other brain–computer interface (BCI) applications, based on similar principles of feedback, self-regulation, and neuroplasticity, are readily accepted within mainstream neuroscience and rehabilitation research. This asymmetry reflects not superior methodological rigor but a misapplied standard of evidence.

The insistence on double-blind, placebo-controlled RCT designs in neurofeedback stems from an uncritical importation of pharmacological logic into a domain governed by entirely different mechanisms. When feedback, engagement, and learning are the active agents of change, attempting to create an "inert" equivalent and to "blind" participants and clinical researchers to these processes dilutes the very phenomena under study. The result is not scientific stringency but methodological distortion: designs that obscure efficacy rather than reveal it.

In this context, the double-blind, placebo-controlled paradigm, often regarded as the gold standard, functions more accurately as *fool's gold*. It carries the appearance of methodological purity while concealing critical conceptual flaws. Recognizing this distinction is not a dismissal of rigor but an appeal to scientific coherence. Abandoning an ill-fitting standard will not weaken neurofeedback research; it will liberate it to develop experimental models that reflect its true nature.

8.2 Is neurofeedback the only domain with these problems?

The limitations of placebo-controlled designs in neurofeedback mirror challenges long recognized in psychotherapy research. A recent Cochrane review of 96 randomized trials across 15 mental health disorders found that the type of control intervention dramatically alters the apparent efficacy of psychological treatments. Psychological placebos, particularly those involving human interaction, produced effect sizes nearly half a standard deviation greater than inert controls. The authors concluded that in mental health research, the choice of control condition can have "as large an influence on effect estimates as the experimental intervention itself." This finding underscores the inherent problem of applying pharmacological trial logic, built around inert placebos and double blinding, to interactive, participatory interventions such as psychotherapy or neurofeedback [39].

Importantly, acknowledging the presence of non-specific effects in these domains does not diminish their legitimacy. Comparable proportions of variance in treatment outcome are attributed to non-specific mechanisms in cognitive behavioral therapy (CBT) and pharmacotherapy, yet neither field faces existential doubt on that basis. To apply stricter interpretive standards to neurofeedback while accepting these realities elsewhere represents an inconsistency in scientific reasoning, not a deficiency in the intervention itself.

If neurofeedback research continues to be amalgamated to the pharmacological model, it is essential to understand that the sNFB condition more closely approximates an *active placebo*: a control condition that mimics procedural and sensory aspects of treatment while still exerting partial physiological or behavioral effects. In pharmacology, active placebos are rarely used (approximately 0.5% of trials in 2013) precisely because they confound interpretation by retaining components of the active mechanism [40]. This confound applies to sNFB, which replicates neurofeedback's structure and sensory experience while delivering partially contingent, and partially non-contingent but still mechanistically meaningful, feedback.

The current crisis facing neurofeedback, therefore, is not empirical but conceptual. It arises from evaluating a dynamic, adaptive, brain–behavior therapy through the methodological lens of a static, pharmacological intervention. Until that misalignment is corrected, the evidence base for neurofeedback will remain distorted by the very standards intended to strengthen it.

Are more RCTs the way forward? To directly quote a recent umbrella review: "After more than half a century of research, thousands of RCTs, and millions of invested funds, the effect sizes of psychotherapies and pharmacotherapies for mental disorders are limited, suggesting a ceiling effect for treatment research as presently conducted" [41]. It would stand to reason that this statement should apply to neurofeedback as well.

Rather than making the same methodological mistakes, a refinement of clinical research methodology is needed as well as a greater appreciation of the contextual complexity for all interventions targeting mental health.

8.3 An existential crisis: are the critics of neurofeedback too harsh?

The methodological inconsistencies outlined throughout this paper have contributed to a pervasive mischaracterization of neurofeedback as little more than a "neuroplacebo" [1]. When analysed through this scope, the findings of neurofeedbamany critical meta-analyses; take for example the recent Westwood et al. claim of "outcomes provided

no evidence of meaningful benefits of neurofeedback as a treatment for ADHD" [ck research are leagues away 42]. In some cases, this pervasive negative framing has prompted calls for the field's abandonment [43].

Not all neurofeedback reviews are as harsh. Another recent meta-analysis examining neurofeedback for ADHD by Lee et al. [44] yields more nuanced insight: neurofeedback versus no treatment demonstrates a large effect size (Hedge's g = 1.25), while compared with other active paradigms the effect size is understandably smaller (a few examples: NFB vs. physical exercise, g = 0.31; NFB vs. EMG biofeedback, g = 0.44; NFB vs. methylphenidate, g = − 0.25). These conclusions are similar to findings showing that both neurofeedback (NFB) and sham neurofeedback (sNFB) conditions yield significant symptom reductions, often with effect sizes approaching or exceeding 1.0; values comparable to first-line pharmacological treatments such as methylphenidate [45].

While scrutiny is valuable insofar as it helps design more rigorous research, critiques of neurofeedback must be interpreted within the broader context of treatment science.

A comprehensive umbrella review of pharmacological and traditional psychological interventions by Leichsenring and colleagues [41] analyzed 102 meta-analyses encompassing 3,782 randomized controlled trials and over 650,000 patients across multiple psychiatric disorders, including depression, anxiety, PTSD, OCD, ADHD, and eating disorders. Their conclusions were unequivocal: "Across disorders and treatments, the majority of effect sizes for target symptoms were small".

These are the most established treatment modalities in psychiatry and psychology for the most prevalent mental health disorders. Yet the recognition of small, variable effects of pharmacotherapy and psychotherapy has not prompted nearly as much criticism as has neurofeedback.

In contrast to these widely accepted domains, neurofeedback is often held to a disproportionately stringent standard. Methodological imperfections are sometimes interpreted not as normal scientific challenges but as evidence against the intervention itself. This could be construed as an interpretive bias magnified by reliance on inappropriate control designs such as sham feedback.

This apparent paradox reflects a deeper cultural issue in research methodology. The widespread adoption of sham feedback as the default control condition has embedded a conceptual error within the evidentiary framework itself. Even the influential Consensus on the Reporting and Experimental Design of Neurofeedback (CRED-nf) guidelines explicitly endorse sham feedback as a preferred control strategy, stating: "This item is essential. Use a control group (between subjects) or control condition (within subjects). This could include a placebo-control (e.g., sham-neurofeedback, neurofeedback from a largely unrelated brain signal, or inversing the neurofeedback reward contingency)" [46].

Yet, as this paper has demonstrated, sNFB designs are methodologically incoherent for interventions that depend on interactive, contingent learning. The persistence of this paradigm may reflect the entrenched influence of the pharmacological model, which privileges blinding and inert comparators even when these principles are ill-suited to behavioral and relational interventions. Only the final recommendation of inversing reward contingency negates the sNFB problem as described herein, and this control method is rarely employed for ethical reasons.

9 What research model applies to neurofeedback?

A more fitting analogy lies in psychotherapy research. Like psychotherapy, neurofeedback is participatory, adaptive, and context-sensitive; its efficacy depends on engagement, relational factors, and learning. In psychotherapy, placebo-controlled designs are recognized as inadequate for isolating specific effects, yet this has not led to the dismissal of established therapies. Meta-analytic data from cognitive behavioral therapy (CBT) indicate that roughly 15% of treatment effects are attributable to specific techniques, while placebo and expectancy account for about 15%, common relational factors for 30%, and extra-therapeutic influences for approximately 40% [47]. The therapeutic alliance alone may contribute therapeutic effects nearly three times as strong as specific treatment differences [48].

A similar distribution may also apply to neurofeedback, where outcomes likely reflect the interaction of specific neural learning mechanisms with broader contextual and relational processes. This approach must be considered in best-practice recommendations, such as the CRED-nf framework [46], which advocates for methodological rigor through transparency, replication, and notably: appropriately matched active comparators. Sham paradigms and other control conditions that deliver partially contingent as well as active non-contingent feedback must be removed from this list.

Reframing control design in this way aligns neurofeedback research with the logic of complex behavioral interventions rather than the misplaced conventions of pharmacological trials.

9.1 A familiar pattern—and a path forward

Despite the challenges posed by methodological misalignment and inconsistent interpretation, there is reason for cautious optimism. The current skepticism toward neurofeedback parallels earlier debates in the history of psychotherapy. In the 1980s, psychotherapy itself was frequently dismissed as a placebo, its efficacy attributed to expectation rather than mechanism [49, 50]. By the 1990s, scholars began questioning whether the concept of a placebo was even applicable to complex, relational treatments [51]. In the following decade, Kirsch and colleagues argued that psychotherapy and placebo mechanisms are inseparable, yet that this interdependence does not diminish psychotherapy's legitimacy [52, 53]. More recent perspectives have reframed psychotherapy as an *open-label placebo*, a consciously participatory process that still yields meaningful and reproducible outcomes [54].

A similar trajectory may now be emerging for neurofeedback. Empirical and conceptual advances continue to substantiate its value across clinical, educational, and performance contexts. A recent bibliometric analysis identified more than 3,600 peer-reviewed publications encompassing EEG-, MEG-, fMRI-, and fNIRS-based feedback modalities. The authors comment that the analysis "significantly enhances our understanding of the dynamic field of neurofeedback, indicating its potential in treating ADHD and improving performance. It offers non-invasive, ethical alternatives to conventional psychopharmacology and aligns with the trend toward personalized medicine" [55].

These developments suggest that neurofeedback is undergoing a familiar scientific evolution: from skepticism and conceptual uncertainty toward integrative legitimacy. By recognizing its shared lineage with other learning-based interventions, and by adopting

methodological frameworks appropriate to its interactive nature, the field can move beyond defending its existence to refining its application.

10 Conclusion: where does neurofeedback go from here?

The future of neurofeedback research depends on abandoning the misplaced ambition to replicate pharmacological standards of specificity. Instead, the field should adopt a framework more closely aligned with psychotherapy and other interactive, learning-based interventions, one that acknowledges individual variability, relational dynamics, and the interplay of specific and non-specific mechanisms.

Moving forward, research should prioritize three key directions. First, the identification of reliable neural biomarkers, as outlined in the NIMH Research Domain Criteria (RDoC) framework, can help anchor neurofeedback protocols in empirically defined brain–behavior relationships. Second, the development of model-based and personalized training paradigms will allow interventions to adapt dynamically to individual neural profiles, improving both efficacy and generalizability. Third, outcome measurement should be expanded beyond behavioral metrics to include multimodal indicators—combining psychometric data with structural and functional neuroimaging, electrophysiology, and other neurobiological assays [56].

Together, these advances will allow neurofeedback to evolve beyond the rigid double blind placebo-controlled RCT paradigm that has constrained its scientific progress. A revised evidentiary model—one that integrates mechanistic precision with contextual awareness—can position neurofeedback as a robust, evidence-based approach to self-regulation and brain–behavior change. By embracing the complexity inherent in human learning rather than attempting to subtract it, the field can finally move from defending its existence to defining its contribution.

Author contributions
B.P. conceived the study, conducted the literature review, developed the theoretical framework, performed all analyses, prepared the figures and tables, and wrote the full manuscript.

Funding
Not applicable. The author received no specific grant from any funding agency, commercial or not-for-profit sectors for the preparation of this work.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The author discloses multiple professional affiliations and roles that may be perceived as potential conflicts of interest. He is a member of several national and international biofeedback and neurofeedback associations and is the founder and director of a private company specializing in neurofeedback education and professional mentorship. He has also been practicing in the field of neurofeedback for nearly two decades. These roles inform the author's perspective but have not influenced the data, analysis, or conclusions presented in this manuscript.

Received: 8 November 2025 / Accepted: 2 March 2026
Published online: 15 March 2026

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