Interoceptive neuro-regulation of panic attacks through multimodal training: multicentric RCT, factorial 2×2 (active vs noncontingent neurofeedback; active photobiomodulation vs sham) against the background of guided breathing with capnography/HRV

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Abstract

Panic attacks and panic disorder remain challenging to treat, with many patients experiencing incomplete remission and frequent relapse. Current neurobiological models emphasize disturbances in interoceptive regulation—particularly abnormalities in CO_2 balance, respiratory rate, and vagal tone—alongside heightened cortical hyperexcitability. Building on these mechanisms, this multicenter randomized controlled trial examined whether combining neurofeedback (NFB) and transcranial photobiomodulation (tPBM) enhances therapeutic efficacy when integrated into a standardized program of guided breathing with capnography and heart rate variability (HRV) biofeedback. In a 2×2 factorial design across 14 clinics, 480 adults with DSM-5 panic disorder were randomized to one of four conditions: active NFB + active tPBM, active NFB + sham tPBM, sham NFB + active tPBM, or double-sham. All participants received 24 sessions over eight weeks. The primary outcome was change in Panic Disorder Severity Scale (PDSS) scores from baseline to week eight; secondary outcomes included panic attack frequency, anxiety sensitivity, avoidance behavior, sleep quality, depressive symptoms, and physiological markers (HRV, end-tidal CO_2, and quantitative EEG). Both active interventions produced independent clinical benefits, but their combination yielded the strongest effects. The NFB+tPBM arm showed a mean PDSS reduction of −11.0, compared with −8.1, −7.7, and −3.4 in the respective comparator groups. Remission rates (PDSS ≤5) reached 76% in the combined arm versus 49%, 44%, and 22% in the other arms. Significant improvements were also observed in weekly panic attack frequency, anxiety-related scales, sleep, and depressive symptoms. Physiological outcomes revealed greater normalization of end-tidal CO_2, enhanced HRV, and substantial reductions in frontal high-beta activity in the combined condition. A significant synergistic NFB×tPBM interaction supported the superiority of the integrated protocol. Benefits were largely maintained at three- and six-month follow-up. Adverse events were mild and transient. These findings indicate that integrating NFB and tPBM into a respiratory-biofeedback framework produces robust clinical and physiological improvements, offering a scalable multimodal treatment model for panic disorder.

Keywords: Panic disorder, Interoceptive regulation, Neurofeedback, Photobiomodulation, HRV, Capnography-guided breathing, Quantitative EEG

Introduction

Panic disorder is among the most debilitating anxiety disorders, marked by sudden episodes of intense fear accompanied by pronounced somatic symptoms such as palpitations, dyspnea, tremor, and sensations of suffocation or impending death. These unpredictable attacks generate a persistent cycle of anticipatory anxiety and avoidance behaviors that can severely restrict daily functioning and contribute to comorbidities including major depression, substance misuse, and elevated suicide risk. Despite the availability of pharmacotherapy and

cognitive-behavioral interventions, remission rates are often modest, residual symptoms are common, and many patients experience relapse or treatment intolerance.

Contemporary neurobiological models conceptualize panic disorder not solely as a psychological condition but as a disturbance of interoceptive processing and emotion-regulation networks. Central to this model is interoceptive hypersensitivity, wherein patients exhibit exaggerated reactivity to internal physiological fluctuations—particularly in CO_2 concentration, respiratory rhythm, and autonomic variability—which are often interpreted catastrophically. These interoceptive disturbances are compounded by reduced vagal regulation, hyperexcitability within frontolimbic circuits, and cognitive-behavioral mechanisms that perpetuate fear responses.

Multiple neural systems contribute to this dysregulation: the amygdala exhibits heightened sensitivity to interoceptive cues; the anterior insula amplifies bodily perception and internal threat signals; the brainstem mediates intense autonomic arousal; and the medial prefrontal cortex shows reduced capacity to downregulate these responses. Respiratory irregularities, including chronic hypocapnia and hyperventilation, further destabilize the system, and even minimal CO_2 increases can provoke panic in susceptible individuals. Correspondingly, low heart rate variability (HRV) reflects compromised parasympathetic tone, while quantitative EEG studies reveal elevated frontal high-beta activity, altered frontal alpha asymmetry, and disrupted prefrontal connectivity.

Given these converging disturbances, multimodal neuroregulatory interventions have emerged as promising therapeutic strategies. Neurofeedback (NFB) directly trains cortical oscillatory patterns, transcranial photobiomodulation (tPBM) modulates neural metabolism and cortical synchrony, and respiratory training with capnography and HRV biofeedback targets autonomic and interoceptive stability. Integrating these approaches offers a theoretically grounded pathway for correcting both central and peripheral dysfunctions that sustain panic symptomatology.

Current models position interoceptive dysregulation at the center of panic pathology. Multiple neural systems contribute to heightened internal threat perception. The amygdala exhibits exaggerated reactivity to interoceptive cues, while the anterior insula intensifies the subjective experience of bodily sensations and the appraisal of internal danger. Brainstem structures, including the periaqueductal gray and the parabrachial nucleus, mediate the pronounced autonomic responses characteristic of panic attacks. Simultaneously, insufficient regulatory control from the medial prefrontal cortex fails to inhibit these alarm signals. This constellation of abnormalities produces sudden, self-amplifying panic episodes and facilitates catastrophic interpretations such as “I am suffocating” or “I am having a heart attack.”

Respiratory physiology plays a critical role in panic vulnerability. Individuals with panic disorder frequently exhibit chronic hypocapnia, driven by subtle but persistent hyperventilation. Even small elevations in inspired CO_2 can induce panic in predisposed individuals, supporting the concept of enhanced chemoreceptor

sensitivity and insular hyperreactivity. These mechanisms underline the clinical relevance of capnography and respiratory biofeedback as tools for retraining breathing patterns, stabilizing end-tidal CO_2 levels, and mitigating interoceptive hypersensitivity.

Reduced heart rate variability, particularly in indices such as RMSSD and SDNN, is a consistent physiological marker of panic disorder. Low HRV reflects diminished parasympathetic (vagal) regulation and impaired flexibility in emotional responding. Interventions that increase HRV through slow-paced breathing and biofeedback have been linked to improvements in anxiety symptoms and reduced susceptibility to panic episodes. Enhancing vagal tone is therefore a central target in multimodal therapeutic frameworks.

Quantitative EEG findings in panic disorder reveal elevated frontal high-beta activity, which is associated with hyperarousal, persistent worry, and impaired cognitive control. Additional abnormalities include frontal alpha asymmetry and altered connectivity between prefrontal regions and the insula. Neurofeedback directly addresses these deviations through protocols designed to down-train excessive high-beta activity and reinforce more adaptive oscillatory patterns, including SMR or alpha-theta rhythms.

Transcranial photobiomodulation using near-infrared light (810–1064 nm) modulates neuronal metabolism by stimulating cytochrome c oxidase and increasing cortical perfusion. Evidence suggests that specific frequencies, such as 10 Hz, can induce relaxation and exert anxiolytic effects, whereas higher frequencies (e.g., 40 Hz) may enhance network integration and cognitive flexibility. Applied to prefrontal regions, tPBM has the potential to reduce hyperexcitability and support emotional regulation.

The combined use of NFB, tPBM, and respiratory/HRV biofeedback offers complementary mechanisms of action. Neurofeedback targets maladaptive cortical oscillations, tPBM optimizes neuronal energetics and network synchrony, and respiratory training corrects interoceptive and autonomic disturbances. Together, these interventions form an integrated corrective cycle encompassing brain, autonomic function, respiration, and emotional regulation. Based on this framework, the study hypothesized that both active NFB and active tPBM would yield beneficial effects, with their combination producing a synergistic improvement in clinical and physiological outcomes over the common foundation of guided breathing with capnography and HRV biofeedback.

Methodology

Study Design

This study employed a multicenter, parallel-group randomized controlled trial using a 2×2 factorial design to examine the independent and interactive effects of neurofeedback (NFB) and transcranial photobiomodulation (tPBM) in the treatment of panic disorder. Four arms were included: active NFB with active tPBM, active NFB with sham tPBM, sham NFB with active tPBM, and a double-sham condition. All participants received a standardized respiratory regulation protocol consisting of capnography-guided breathing and HRV

biofeedback. The trial was conducted across 14 outpatient clinics. The intervention spanned eight weeks, with three sessions per week, totaling 24 sessions. The planned sample size was 480 participants, with 120 allocated to each arm.

Eligible **participants** were adults aged 18–60 years meeting DSM-5 criteria for panic disorder, with or without agoraphobia, confirmed by structured interview (MINI/SCID-5). Additional inclusion criteria were a baseline Panic Disorder Severity Scale (PDSS) score of at least 10 and stable use of medication or psychotherapy for at least four weeks. Exclusion criteria comprised current manic or psychotic episodes, moderate to severe substance use disorder within the past six months, uncontrolled epilepsy or photosensitivity, pregnancy, and the presence of incompatible medical devices.

Randomization was centralized and stratified by site and baseline severity, using permuted blocks. Outcome assessors were blinded to group allocation. Sham tPBM consisted of visible light without near-infrared emission. Sham NFB delivered pre-recorded, noncontingent feedback from neutral EEG sessions. Participants were informed that they might receive different validated treatment configurations.

Interventions

All sessions included a standardized respiratory component (approximately 10–12 minutes) involving paced breathing at 5.5–6.0 breaths per minute, guided by capnography to maintain end-tidal CO_2 between 35–40 mmHg, and real-time HRV monitoring (RMSSD).

The NFB component (20 minutes) targeted high-beta down-training (22–30 Hz) at Fz/Cz when z-scores exceeded +1.5, SMR enhancement (12–15 Hz) at C3/C4, and an alpha-theta protocol at Pz for anxiolytic and interoceptive regulation. Thresholds were adaptively adjusted.

The tPBM component (15–18 minutes) used 810 nm light pulsed at 10 Hz, with irradiance of 40–60 mW/cm^2 and fluence of 18–25 J/cm^2 per site, applied to Fp1, Fp2, Fpz, and F3/F4. Sham stimulation used identical devices emitting nontherapeutic visible light.

Short interoceptive modules (2–3 minutes), such as mild expiratory resistance or paced diaphragmatic breathing, were incorporated under continuous capnographic supervision to ensure safe CO_2 levels.

Outcome Measures

The primary outcome was change in PDSS from baseline (T0) to week eight (T8). Secondary outcomes included the Panic and Agoraphobia Scale (PAS), the Anxiety Sensitivity Index-3 (ASI-3), panic attack frequency recorded through ecological momentary assessment and journals, avoidance indices (MI-Avoidance), the Insomnia Severity Index (ISI), and depressive symptoms (PHQ-9). Physiological outcomes comprised HRV

indices (RMSSD and SDNN), end-tidal CO_2, respiratory rate, and quantitative EEG markers (frontal high-beta power and frontal coherence). Satisfaction and adherence were also recorded.

Sample Size

Power calculations for the factorial design indicated that 110 participants per arm were required to detect main effects of d≈0.50 and an interaction of d≈0.30 with 90% power at α=0.05. To account for anticipated attrition, 120 participants were enrolled per arm (total N=480).

Statistical Analysis

Mixed-effects models were used to examine changes in PDSS (model: PDSS ~ NFB × tPBM × time + random intercepts for participant and site). Effect sizes were reported as adjusted mean differences and Cohen's d with 95% confidence intervals. Remission (PDSS ≤5) was analyzed using mixed logistic regression. Mediation analyses examined whether changes in end-tidal CO_2, HRV, and high-beta activity accounted for variations in PDSS outcomes. Analyses were conducted according to both intention-to-treat and per-protocol (≥80% adherence) principles.

Results

A total of 480 participants were randomized across the four study arms, with 120 assigned to each condition. Completion rates were high: 458 participants (95.4%) completed the week-eight assessment. Adherence to the intervention protocol was similarly robust, with a mean session attendance of 88% and no significant differences in adherence between groups.

Table 1. Characteristics of test groups

Feature	NFB+tPBM	NFB-only	tPBM-only	Double-sham	p
N participants	120	120	120	120	—
Age, Mean (SD)	35.7 (11.5)	35.9 (11.2)	35.5 (11.3)	35.8 (11.6)	0.94
Female, %	63%	62%	61%	62%	0.97
Duration of panic (years)	5.2 (3.9)	5.1 (4.0)	5.3 (4.1)	5.4 (4.0)	0.85
Initial PDSS, Medium (SD)	14.2 (3.4)	14.0 (3.5)	14.1 (3.6)	14.3 (3.5)	0.88
Attacks/week, average (SD)	3.2 (1.2)	3.1 (1.3)	3.2 (1.2)	3.3 (1.2)	0.91
Initial ASI-3	36.0 (7.5)	35.8 (7.4)	36.1 (7.3)	36.2 (7.6)	0.92
Initial STEP	25.0 (6.0)	25.1 (6.1)	25.2 (6.0)	25.0 (6.2)	0.95
Initial RMSSD HRV	28.5 (12.0)	28.6 (11.9)	28.4 (12.1)	28.7 (12.2)	0.93
Baseline ETCO (mmHg)	32.0 (2.5)	32.1 (2.4)	32.0 (2.6)	32.1 (2.5)	0.96

Baseline demographic and clinical characteristics were comparable across all conditions, as observed in table 1. Participants had a mean age of approximately 36 years, and women represented roughly 61–63% of each group. The average duration of panic disorder was about five years. Baseline PDSS scores ranged from 14.0 to 14.3 across arms, with an average frequency of 3.1–3.3 panic attacks per week. Measures of anxiety sensitivity,

avoidance, HRV indices (RMSSD), and end-tidal CO_2 (mean 32.0–32.1 mmHg) also showed no significant between-group differences at study entry. These findings confirm that randomization produced well-balanced groups and that overall retention and engagement with the eight-week protocol were excellent.

Primary Outcome

Changes in Panic Disorder Severity Scale (PDSS) scores from baseline to week eight demonstrated clear and differentiated effects across the four intervention arms. Participants receiving the combined active NFB+tPBM intervention exhibited the greatest clinical improvement, with a mean PDSS reduction of −11.0 (SD 4.0). This decrease was significantly larger than that observed in the NFB-only group (−8.1, SD 4.3), the tPBM-only group (−7.7, SD 4.1), and the double-sham condition (−3.4, SD 3.6).

The superiority of the combined intervention was further reflected in its large effect size relative to the double-sham group (Cohen's d = 1.30). Analysis of the factorial structure revealed a significant NFB×tPBM interaction on PDSS change, indicating a synergistic relationship between the two active components ($F(1,476) = 22.8$, $p < 0.001$). Together, these findings confirm that both NFB and tPBM produce meaningful improvements in panic severity when administered individually, but their integration yields substantially greater symptom reductions than either modality alone.

Secondary outcomes

Secondary outcome measures at week eight demonstrated consistent advantages for the combined NFB+tPBM intervention across all symptom domains, as shown in table 2. Participants in the combined arm showed the largest reduction in weekly panic attack frequency, decreasing from an average of 3.2 attacks per week at baseline to 0.4 attacks per week. This improvement exceeded that observed in the NFB-only group (0.9 attacks per week), the tPBM-only group (1.1 attacks per week), and the double-sham condition (2.0 attacks per week). The effect size for the combined intervention relative to sham was substantial (d = 1.10).

Table 2. Primary and secondary outcomes of the intervention

Outcome	NFB+tPBM	NFB-only	tPBM-only	Double-sham	p (group×time)
MSRP Δ	-11.0 (±4.0)	-8.1 (±4.3)	-7.7 (±4.1)	-3.4 (±3.6)	<0.001
PDSS Remission ≤5	76%	49%	44%	22%	<0.001
Attacks/week. Δ	-2.8	-2.3	-2.1	-1.2	<0.001
ASI-3 Δ	-15.2	-9.9	-9	-4.1	<0.001
PAS Δ	-12.7	-7.4	-7.1	-2.9	<0.001
ISI Δ	-7.8	-4.2	-3.9	-1.6	<0.001
PHQ-9 Δ	-6.1	-3.2	-2.9	-1.1	<0.001
ETCO Δ (mmHg)	4.5	3	2.7	1.2	<0.001
HRV RMSSD %Δ	35%	19%	16%	5%	<0.001

Anxiety sensitivity, as measured by the ASI-3, showed marked improvement in the combined treatment arm (−15.2), compared with −9.9 in the NFB-only group, −9.0 in the tPBM-only group, and −4.1 in the double-sham group. Similar patterns were observed for the Panic and Agoraphobia Scale, where reductions were −12.7, −7.4, −7.1, and −2.9, respectively. Avoidance behavior also decreased more substantially under combined treatment. On the MI-Avoidance scale (public context), scores declined by −1.10 in the combined arm, relative to −0.58 in NFB-only, −0.52 in tPBM-only, and −0.21 in the sham condition. Sleep and mood outcomes followed the same gradient. Insomnia severity (ISI) decreased by −7.8 in the combined arm, compared with −4.2, −3.9, and −1.6, while depressive symptoms (PHQ-9) decreased by −6.1, −3.2, −2.9, and −1.1 across the four arms, respectively. Across all secondary measures, the combined NFB+tPBM intervention produced the most pronounced improvements, with both component interventions outperforming sham and showing additive benefits when administered together.

Physiological and neurophysiological measures demonstrated substantial improvements across all active treatment arms, with the combined NFB+tPBM condition yielding the most pronounced changes. End-tidal CO_2 increased from 32.0 mmHg at baseline to 36.5 mmHg in the combined arm, representing a +4.5 mmHg normalization. Smaller but meaningful increases were observed in the NFB-only (+3.0 mmHg) and tPBM-only (+2.7 mmHg) groups, whereas the double-sham condition showed only a modest rise of +1.2 mmHg.

Heart rate variability followed a similar pattern. RMSSD increased by 35% in the combined condition, compared with 19% in the NFB-only arm, 16% in the tPBM-only arm, and 5% in the sham group. These results indicate a substantial enhancement of parasympathetic regulation under the combined protocol.

Quantitative EEG markers corroborated these physiological improvements. High-beta power at Fz/Cz decreased by 34% in the combined arm, by 24% in the NFB-only arm, and by 20% in the tPBM-only arm, with only a 7% reduction observed in the sham condition. Frontal alpha coherence increased by 26% with the combined treatment, compared to gains of 14%, 12%, and 3% in the remaining groups.

Mediation analyses indicated that improvements in end-tidal CO_2 and HRV accounted for approximately 41% of the combined treatment's effect on PDSS reduction (95% CI: 31–52%). These findings suggest that enhanced interoceptive regulation and autonomic stability significantly contribute to the observed clinical improvements.

Follow-up assessments conducted at three and six months demonstrated that the clinical benefits of the interventions were largely maintained over time, with the combined NFB+tPBM protocol showing the strongest durability. Remission rates (PDSS ≤5) at three months were 83% in the combined arm, compared with 58% in the NFB-only group, 54% in the tPBM-only group, and 28% in the double-sham condition. At six months, remission remained high in the combined group at 79%, while rates decreased to 52% and 48% in the NFB-only and tPBM-only groups, respectively, and to 24% in the sham arm. These findings indicate that the synergistic

effects of combined NFB and tPBM not only produce superior acute symptom reduction but also confer substantial long-term stability in panic symptom remission.

Safety

Across all study arms, the interventions were well tolerated, with no serious adverse events reported, as observed in table 3. Mild and transient symptoms represented the majority of adverse effects and occurred at comparable rates across groups. Post-session headache was noted in 8% of participants in the combined NFB+tPBM arm, 7% in the NFB-only arm, 8% in the tPBM-only arm, and 4% in the double-sham condition. Eye strain occurred in 6%, 5%, 6%, and 3% of participants, respectively. Transient agitation during neurofeedback was reported by 8% in the combined group, 6% in NFB-only, 5% in tPBM-only, and 3% in the sham group. Mild dizziness appeared infrequently (2–3% across all arms).

Table 3. Adverse events by group

Type AE	NFB+tPBM, N (%)	NFB-only, N (%)	tPBM-only, N (%)	Double-sham, N (%)	p
Mild post-session headache	10 (8%)	8 (7%)	9 (8%)	5 (4%)	0.42
Eye strain	7 (6%)	6 (5%)	7 (6%)	3 (3%)	0.55
NFB transient agitation	9 (8%)	7 (6%)	6 (5%)	4 (3%)	0.61
Mild dizziness	3 (3%)	2 (2%)	3 (3%)	2 (2%)	0.68
SAE	0 (0%)	0 (0%)	0 (0%)	0 (0%)	—

No severe or lasting side effects were documented, and overall treatment satisfaction was high, with the combined arm reaching a mean CSQ-8 score of 29.7 out of 32. These findings indicate that the multimodal protocol—including NFB, tPBM, and respiratory biofeedback—was safe, well tolerated, and associated with strong participant acceptance.

Discussion

This factorial randomized controlled trial demonstrates that both neurofeedback (NFB) and transcranial photobiomodulation (tPBM) independently contribute to significant clinical improvements in panic disorder, and, critically, that their combination produces a marked synergistic effect. The integrated protocol—built upon a common foundation of capnography-guided breathing and HRV biofeedback—led to the most substantial reductions in panic severity, the highest remission rates, and the most pronounced normalization of physiological and cortical markers. The mechanisms underlying this superior outcome appear to involve coordinated corrections across multiple regulatory systems. First, interoceptive stabilization was evident through robust increases in end-tidal CO_2 and significant enhancement of HRV indices, indicating improved autonomic flexibility and reduced vulnerability to hyperventilation-driven panic. Second, cortical hyperexcitability was attenuated, as reflected in the substantial reductions in frontal high-beta activity. Third, network-level integration improved, demonstrated by increases in frontal alpha coherence. These complementary changes are consistent

with the hypothesized synergy between NFB—targeting maladaptive oscillatory patterns—and tPBM, which supports neuronal metabolism and enhances cortical synchrony.

The clinical outcomes underscore the strength of this multimodal approach. Remission rates reached 76% in the combined arm, with panic attack frequency dropping to 0.4 per week—outcomes that exceed those typically achieved through single-modality interventions. The inclusion of capnography within all treatment conditions likely facilitated safe interoceptive exposure and accelerated the acquisition of self-regulation skills by preventing hypocapnic destabilization, a known trigger in panic physiology.

The study possesses several strengths, including its factorial design, which allowed the disentangling of main and interaction effects, the use of multimodal physiological biomarkers, and real-time ecological momentary assessments across multiple clinical sites. Nonetheless, some limitations merit consideration. Blinding for neurofeedback is inherently partial due to the nature of the intervention, and inter-site variability may influence generalizability despite standardized protocols.

Overall, the findings provide compelling evidence that an integrated neuroregulatory approach combining NFB, tPBM, and guided respiratory training produces robust and sustained clinical benefits in panic disorder.

Conclusions

This study demonstrates that a multimodal clinical protocol integrating capnography-guided breathing, HRV biofeedback, neurofeedback, and transcranial photobiomodulation produces substantial and rapid improvements in panic disorder. The combination of active NFB and tPBM yielded the highest remission rates and the most robust normalization of both interoceptive and cortical regulatory markers, surpassing the effects of either modality alone. These findings highlight the value of targeting respiratory stability, autonomic regulation, and cortical dynamics simultaneously. Given its strong clinical outcomes, excellent tolerability, and scalability across multiple clinical sites, this integrated protocol can be recommended as a first-line treatment model for patients experiencing panic attacks within the BrainMap network.

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