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Targeting Anxiety with Light: Mechanistic and Clinical Insights into Photobiomodulation Therapy: A Mini Narrative Review

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Abstract: Anxiety disorders are common and disabling, with current pharmacological and psychotherapeutic options often limited by suboptimal efficacy or adverse effects. Transcranial photobiomodulation (tPBM), a light-based neuromodulation technique using near-infrared wavelengths (typically 810–1064 nm), has emerged as a potential alternative through its effects on mitochondrial bioenergetics, neuroinflammation, and neuroplasticity. The objective of this mini review is to review mechanistic rationale and summarize recent preclinical and clinical findings on tPBM for anxiety disorders. A narrative search of PubMed, Web of Science, and Google Scholar was performed, focusing on experimental animal studies and clinical investigations published in the last decade that examined tPBM or related low-level light therapies for anxiety outcomes. Preclinical studies consistently report that repeated NIR exposure (e.g., 810 nm, 4–8 J/cm²) reduces anxiety-like behaviors in rodents, accompanied by increased serotonin levels, decreased nitric oxide, and modulation of microglial polarization. Early clinical studies—including small open-label trials (n = 10–15) and one randomized controlled trial (n = 70)—suggest that tPBM (810–830 nm, applied for 4–20 minutes, single or repeated sessions) can reduce Hamilton Anxiety scores and improve sleep and mood, with minimal adverse effects. However, study designs are heterogeneous, with small sample sizes and short follow-up durations. tPBM shows promise as a safe, non-invasive intervention for anxiety, supported by converging mechanistic and preliminary clinical evidence. Nonetheless, current findings remain exploratory, and large, rigorously designed randomized controlled trials are essential to establish efficacy, optimize dose parameters, and assess long-term outcomes.

Keywords: Photobiomodulation (PBM); Anxiety disorders; Transcranial low-level light therapy; Low-intensity laser therapy; Low-power laser therapy; Neuromodulation; Neuroinflammation

1. Introduction

Anxiety disorders are among the most prevalent mental health conditions, affecting hundreds of millions globally and contributing to significant disability [1]. Despite available therapies, a substantial proportion of patients experience limited remission or troubling side effects from pharmacological treatments such as Selective Serotonin Reuptake Inhibitors (SSRIs) or benzodiazepines. For instance, SSRIs can cause insomnia, sexual dysfunction, or gastrointestinal upset, while benzodiazepines are associated with sedation, dependency, and withdrawal symptoms [2]. These limitations have driven interest in non-invasive, device-based neuromodulation options. Transcranial photobiomodulation (tPBM), sometimes referred to as low-level light therapy (LLLT), is distinct from other forms of light therapy, such as bright light therapy for seasonal affective disorder, which act primarily through circadian rhythm regulation and retinal photoreceptors. By contrast, tPBM uses near-infrared (NIR)

light in the range of 800–1100 nm, a spectral window that achieves optimal penetration into brain tissue due to low absorption by hemoglobin and water. tPBM typically employs parameters such as wavelength (810–1064 nm), power density (10–250 mW/cm²), energy density (2–60 J/cm²), and treatment durations ranging from 4 to 20 minutes per session. Its primary mechanism involves mitochondrial photoacceptors, especially cytochrome c oxidase, leading to enhanced ATP production, modulation of reactive oxygen species, nitric oxide signaling, and downstream neurotrophic and anti-inflammatory effects. Devices used for tPBM include both lasers, which offer coherent, collimated light, and light-emitting diodes (LEDs), which provide less expensive, scalable, and more portable delivery systems. The latter are particularly relevant for clinical translation, as they enable home-based or outpatient applications without the need for specialized facilities [3–5]. This technique is safe, well tolerated and easy to administer. [3,6] Preliminary studies suggest tPBM can improve symptoms of depression and anxiety, possibly through modulation of cerebral blood flow, neuroinflammation, and neuroplasticity [3,4,6].

The goal of this paper is to synthesize mechanistic, preclinical, and clinical evidence regarding PBM for anxiety, with a focus on recent findings.

2. Methods

This work is a narrative review rather than a systematic or scoping review; therefore, no formal inclusion or exclusion criteria, quality assessments, or PRISMA framework were applied. The purpose was to provide a concise synthesis of mechanistic, preclinical, and clinical evidence on transcranial photobiomodulation (tPBM) in the context of anxiety.

Relevant studies were identified through non-systematic searches of PubMed, Web of Science, and Google Scholar up to April 2025. Search terms included combinations such as *"photobiomodulation AND anxiety," "low-level light therapy AND generalized anxiety disorder," "tPBM AND neuromodulation,"* and *"near-infrared light AND anxiety."* Additional articles were retrieved from reference lists of key papers. Emphasis was placed on recent experimental studies, early clinical trials, and mechanistic reports directly related to anxiety outcomes.

Given the narrative design, the review prioritizes breadth of coverage and mechanistic integration over exhaustive retrieval or critical appraisal of all available literature.

3. Results

Mechanistic Insights

PBM employs near-infrared (NIR) light in the spectral range (800–1100) nm to penetrate into brain tissue, especially when applied over the forehead where the skull is thinner and hairless [1,2]. The primary molecular photoacceptor is cytochrome c oxidase (CCO), the terminal enzyme in the mitochondrial respiratory chain. Upon photon absorption, CCO activation enhances mitochondrial membrane potential, increases ATP synthesis, and triggers a transient rise in reactive oxygen species (ROS), initiating downstream redox signalling [1,5]. These events modulate intracellular calcium, cyclic AMP (adenosine monophosphate), nitric oxide (NO), and reactive oxygen species (ROS), leading to activation of transcription factors such as Nuclear Factor kappa B (NF-κB) and Cyclic AMP Response Element-Binding Protein (CREB) [1,4,5]. This cascade can promote neurotrophic factor release, such as brain-derived neurotrophic factor (BDNF), and shift cells toward an anti-inflammatory state [1,4]. In the nervous system, PBM has been shown to reduce expression of inducible nitric

oxide synthase (iNOS), pro-inflammatory cytokines (IL-1β, TNF-α), and enhance markers associated with tissue repair and neuronal survival [1,5,6].

tPBM also increases regional cerebral blood flow and oxygenation through NO-mediated vasodilation [2,7]. These hemodynamic improvements can enhance brain metabolism and restore function in regions disrupted in anxiety, including the dorsolateral prefrontal cortex (dlPFC), ventromedial prefrontal cortex (vmPFC), amygdala, and hippocampus [2,6].

Recent modelling studies have demonstrated that transcranial and intranasal NIR light application can effectively reach key anxiety-related cortical areas. Simulations using 810 nm light suggest that illumination at F3/F4 (10–20 EEG system) provides efficient targeting of the dlPFC, while intranasal or midline frontal placements can affect deeper regions such as vmPFC and orbitofrontal cortex [6].

Mechanistically, anxiety is linked to dysregulation in emotion-processing circuits, excessive amygdala reactivity, reduced top-down prefrontal control, and heightened neuroinflammatory states [6,8]. tPBM may rebalance these pathways by attenuating microglial activation, promoting M2 (anti-inflammatory) macrophage phenotypes, and increasing BDNF expression, key mechanisms observed in animal models of stress and anxiety [1,5,6,8]. Figure A1 illustrates mechanisms and cortical targets of PBM in anxiety.

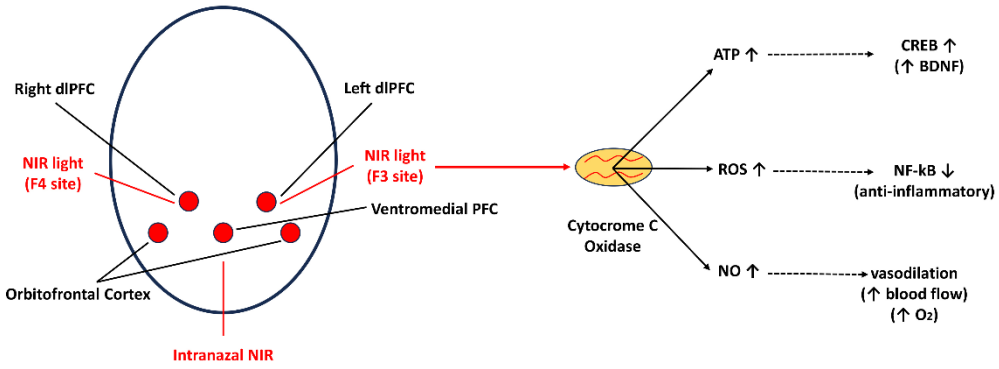

Figure A1. Transcranial photobiomodulation mechanisms and cortical targets in anxiety

Preclinical Evidence

A growing body of animal studies supports the anxiolytic potential of tPBM. These models consistently show that NIR light can reduce behavioral and molecular markers of anxiety and depression. One key mechanism is modulation of serotonergic and inflammatory signalling in brain regions such as the prefrontal cortex and hippocampus [5,9].

In a chronic stress mouse model, daily tPBM with an 810 nm laser (8 J/cm²) significantly reduced anxiety-like behavior and restored serotonin levels. The treatment also decreased nitric oxide (NO), a reactive mediator associated with neurotoxicity and inflammation, supporting the hypothesis that PBM helps normalize stress-induced neurotransmitter imbalance [5].

Another line of evidence shows PBM can shift activated microglia and macrophages from pro-inflammatory M1 phenotypes toward anti-inflammatory M2 types. In vitro, PBM downregulated iNOS (inducible nitric oxide synthase) and increased secretion of neurotrophic factors (BDNF, CNTF, LIF) in lipopolysaccharide-primed

macrophages via the cAMP–PKA–CREB pathway. In vivo, similar polarization changes have been linked to improved axonal regeneration and reduced glial scarring after injury [9].

PBM also promotes neuroplasticity. Repeated NIR stimulation upregulates BDNF and enhances hippocampal neurogenesis in rodents exposed to chronic stress, which may underlie improvements in behavior and cognition [4,5,9]. These mechanisms are relevant given the role of hippocampal atrophy and reduced BDNF in anxiety pathophysiology.

These studies underscore the ability of PBM to impact multiple anxiety-relevant mechanisms - serotonin, NO, microglial state, and neurotrophin expression - through a unifying mitochondrial-mediated pathway [10].

Clinical Evidence

Initial clinical studies suggest that tPBM is a safe and effective intervention for reducing anxiety symptoms. Pilot studies administered a single 4-minute session of 810 nm NIR light to the forehead in ten patients with major depressive disorder and comorbid anxiety. Two weeks post-treatment, both Hamilton Anxiety and Depression scores had significantly improved, and Single-Photon Emission Computed Tomography (SPECT) demonstrated increased perfusion in the prefrontal cortex during stimulation [6].

Subsequent trials using longer PBM protocols further reinforced these findings. Maiello et al. [11] conducted an open-label study in 15 adults with generalized anxiety disorder (GAD), applying 830 nm LED light via a headband daily for 8 weeks. The treatment was well tolerated, and 80% of patients completed the trial. There was a mean 50% reduction in HAM-A scores (17.3 to 8.5; $p < 0.001$), with 75% classified as responders [11]. The first randomized controlled trial assessing PBM for anxiety-related symptoms was published by Helali et al. [1] in 2025. In 70 patients undergoing methadone maintenance treatment, those randomized to receive a 4-minute session of 810 nm LED PBM at the forehead (60 J/cm², 250 mW/cm²) reported significantly greater reductions in HAM-A and depression scores compared to a sham group, with benefits sustained at 1- and 3-month follow-up [1]. Another randomized pilot study conducted by Cassano et al. [3] used 823 nm LED stimulation applied bilaterally to the forehead twice weekly for 8 weeks in patients with major depressive disorder (MDD). Though not designed specifically for anxiety, post hoc analyses indicated improvements in comorbid anxiety symptoms, and no serious adverse events were reported [3].

Overall, these studies show that PBM can safely induce meaningful reductions in anxiety, both as a primary condition and as a comorbidity of depression or substance use disorders, as shown in Table 1. The interventions were consistently well tolerated, with only minor transient side effects (e.g., scalp warmth, vivid dreams, or headache) reported [1,4,6,11].

Importantly, only a limited number of studies have directly assessed anxiety outcomes as primary endpoints. Some trials, such as Cassano et al. [3], were designed for depression and only reported improvements in comorbid anxiety symptoms as secondary or post hoc findings. These should be interpreted as indirect support rather than definitive anxiolytic evidence.

Table 1. Selected studies of tPBM for anxiety

Study	Model / Population	Type of Evidence	PBM Parameters	Key Outcomes
Salehpour et al. (2023)	Mice with chronic restraint stress	Animal model	810 nm laser, 8 J/cm² daily for 4 weeks	↓ anxiety-like behavior ↑ serotonin, ↓ NO in PFC
Zhang et al. (2020)	M1-polarized macrophages (in vitro)	In vitro (mechanistic)	810 nm laser, 4 J	↓ iNOS, ↑ BDNF, CNTF, LIF via PKA-CREB pathway
Wang et al. (2019)	Healthy adults	Humans – healthy volunteers	1064 nm laser, right PFC, 11 min	↑ alpha & beta EEG power (resting state modulation)
Helali et al. (2025)	Adults with anxiety + opioid dependence	Human -RCT	810 nm LED, 4 min at 250 mW/cm²	↓ HAM-A and depression scores at 1 and 3 months
Maiello et al. (2019)	GAD patients (open-label pilot, n = 15)	Human – open-label	830 nm LED headband, 20 min/day, 8 weeks	↓ HAM-A (~50%), improved sleep and CGI-S
Schiffer et al. (2009)	Depressed adults with anxiety	Human – pilot study	810 nm LED, 4 min forehead session	↓ anxiety and depression at 2 weeks, ↑ frontal perfusion

4. Discussion

Overall, light therapy utilizes focused light to treat a wide array of medical conditions, from eye surgery to pain management and, in combination with photosensitizing agents, even in cancer treatment (with especially promising results on prostate cancer) [12]. It works by delivering precise beams of light that can cut, coagulate, or vaporize tissue, or stimulate healing processes.

tPBM represents a novel, biologically plausible treatment modality for anxiety disorders. Mechanistically, it acts via mitochondrial activation in neurons and glia, increasing ATP production, modulating redox-sensitive transcription factors (e.g., NF-κB, CREB), and promoting anti-inflammatory and neurotrophic signalling cascades [1,3,6]. These effects target core neurobiological disruptions in anxiety, including dysregulation of limbic-prefrontal circuits and chronic neuroinflammation [13,14].

Unlike pharmacologic anxiolytics - which often have undesirable cognitive or sedative effects - PBM is non-systemic, non-invasive, and does not impair psychomotor performance or carry abuse potential [2]. The tolerability profile in clinical studies is favourable, and early open-label and randomized trials have demonstrated statistically and clinically significant anxiety reductions in both GAD and substance-use populations [10,15].

Additionally, devices such as LED headbands or intranasal applicators could enable broader access to neuromodulation in resource-limited or therapy-resistant settings [5,11].

Therefore, light-based neuromodulation through tPBM presents a promising, mechanistically grounded approach for the treatment of anxiety disorders. Supported by converging preclinical and clinical data, tPBM appears to reduce anxiety symptoms by enhancing mitochondrial bioenergetics, modulating neuroinflammation, and promoting neuroplasticity in brain regions critically involved in emotional regulation. Clinical studies to date - including open-label trials and a randomized controlled trial - have consistently reported significant reductions in anxiety symptoms, excellent tolerability, and sustained benefits over time. These findings suggest that tPBM could fill a therapeutic gap for individuals who do not tolerate or benefit from conventional pharmacological or psychotherapeutic options.

Overall, the current literature comprises a mix of mechanistic studies, animal models, and small clinical trials. Only a handful of trials have evaluated anxiety as a primary outcome, while many others report anxiety improvements incidentally in the context of depression or substance use. This distinction underscores the preliminary nature of the evidence base. To date, the majority of studies provide indirect or preliminary evidence, with only a few directly targeting anxiety as the primary endpoint.

Nevertheless, large-scale, adequately powered randomized trials are required to confirm efficacy, define optimal parameters (e.g., wavelength, dose, and frequency), and determine long-term safety and cost-effectiveness. Future investigations should also explore the interaction of PBM with existing treatments and examine biomarkers that may predict individual response [16].

Placing tPBM in the broader context of neuromodulation is important. **Repetitive transcranial magnetic stimulation (rTMS)** has demonstrated efficacy for depression and is increasingly explored in anxiety disorders, but it requires specialized equipment, trained operators, and is associated with substantial costs. **Transcranial direct current stimulation (tDCS)** is more affordable and portable, but its clinical effects on anxiety have been inconsistent and often modest. In contrast, **tPBM combines biological plausibility with relatively low cost, portability, and favorable tolerability**, making it potentially more accessible for widespread use, including home-based applications with LED devices. However, compared to rTMS, the clinical evidence base for tPBM in anxiety is far less developed, with only a handful of small trials. Thus, while tPBM may eventually complement or even compete with these established techniques, more rigorous head-to-head studies are needed to define its relative efficacy and cost-effectiveness.

Beyond psychiatry, light- and laser-based therapies have demonstrated important benefits across diverse medical fields, underscoring their translational potential. In **orthopedics and dentistry**, low-level laser therapy has been shown to enhance **osseointegration** [17] of dental implants and accelerate bone healing by stimulating osteoblast activity and angiogenesis. In **surgical practice**, lasers are widely used for precise tissue cutting, coagulation, and photocoagulation, minimizing bleeding and reducing recovery time [18,19]. In the management of **burns and wound healing**, PBM has been reported to reduce inflammation, promote re-epithelialization, and improve scar outcomes [20,21]. Applications in **dentistry** extend from pain control in orthodontic procedures to adjunctive treatment of periodontitis. Furthermore, in **craniofacial medicine**, PBM has been applied to support nerve regeneration, reduce postoperative swelling, and enhance tissue repair [22]. These well-documented effects across multiple specialties reinforce the safety profile and biological plausibility of PBM, supporting its exploration as a neuromodulatory intervention in anxiety and other neuropsychiatric disorders.

Despite encouraging findings, several limitations constrain the interpretation of current evidence. Most clinical studies involve **small sample sizes**, limiting statistical power and generalizability. There is substantial **heterogeneity in protocols**—including wavelength (810–1064 nm), power density, session duration, and number of treatments—which complicates comparison across studies and hinders identification of optimal parameters. Methodological concerns are also present: some early trials lacked **blinding or sham controls**, raising the possibility of expectancy and **placebo effects**. Moreover, in several studies, anxiety was not the primary endpoint but rather a secondary or exploratory measure, further limiting conclusions about direct anxiolytic efficacy. These issues underscore the need for larger, rigorously designed randomized controlled trials with standardized protocols and long-term follow-up.

Although tPBM has generally been well tolerated, several potential risks warrant consideration. At the mechanistic level, **excessive energy delivery may lead to overproduction of reactive oxygen species (ROS)**, which could shift signaling from adaptive to damaging pathways. Similarly, inappropriate dosing might produce **paradoxical effects**, as biphasic dose–response relationships have been reported in photobiology. **Eye safety** is another concern, since direct exposure to NIR light, particularly from laser devices, could cause retinal injury; proper shielding and safety protocols are therefore essential. While no serious adverse events have been reported in the small trials conducted to date, these theoretical risks highlight the importance of careful parameter selection, rigorous safety monitoring, and standardized reporting in future studies.

5. Conclusions

In conclusion, tPBM offers a novel, safe, and patient-friendly strategy that could meaningfully expand the clinical toolkit for managing anxiety. With growing evidence and technological accessibility, it has the potential to be translated into routine psychiatric practice in the near future.

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Appendix

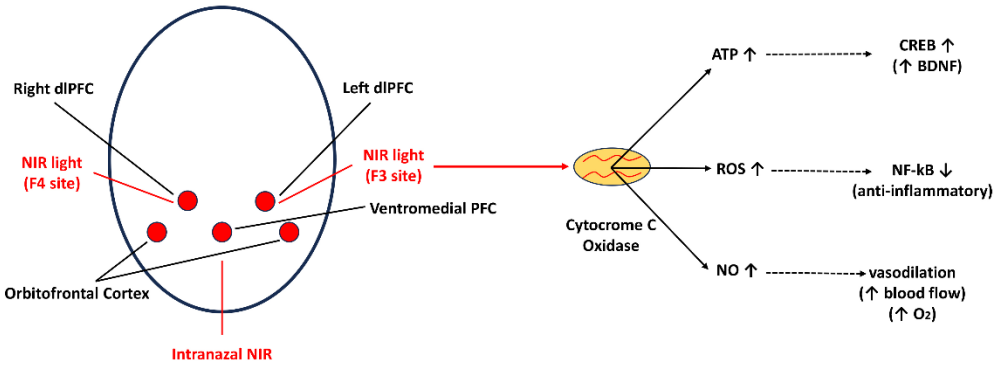

Figure A1. Transcranial photobiomodulation mechanisms and cortical targets in anxiety

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